Assessment of ECG Criteria for the Diagnosis of Right Ventricular Infarction

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Abstract

Background: This study was performed to assess the accuracy of standard electrocardiographic criteria in the diagnosis of Right ventricular infarct (RVI) in patients with inferior myocardial infarction (IMI)

Results: This was a retrospective analysis of patients admitted with an IMI. Proximal occlusion of the Right coronary artery (RCA) before the origin of the RV branch on angiography was considered to be diagnostic of RVI. A total of 129 patients (mean age 55.8±13.1 years, 105 or 81.3% male) with inferior STEMI were included in the analysis. 49 patients (mean age 56.8+12.6 years, 79.5% male) had RCA occlusion proximal to the RV branch. ST elevation in V4R and ST elevation in lead III more than lead II were the most sensitive (72.9% and 80.4% respectively), while ST elevation in V1 either alone or along with ST depression in V2 was the most specific (88.7% and 97.1% respectively). Combining all the criteria improved sensitivity to 85% but reduced specificity to 21.2%

Conclusion: No single ECG criteria was able to identify all cases of RVI in patients with IMI. Combining the different criteria will help to pick up more cases at the cost of increasing the false positives.

Keywords: Right Ventricular Infarction; Inferior Myocardial Infarction; Electrocardiogram,

Introduction

Coronary artery disease is a major cause of morbidity and mortality worldwide accounting for an excess of 17.5 million deaths annually.[1] They present either as an acute coronary syndrome (ACS) (which includes ST-segment elevation myocardial infarction (STEMI), non-ST segment elevation myocardial infarction (NSTEMI) or unstable angina), or as chronic stable angina (chronic coronary syndromes). The STEMI are classified based on the left ventricular wall that is affected. Inferior Myocardial Infarction (IMI) account for around 40-50% of all STEMI.[2] The Inferior wall of the heart receives its blood supply through the right coronary artery (RCA) in around 85% of the population and from the circumflex artery in the remainder.[3] It is further estimated that around a third of all acute IMI are associated with Right ventricular (RV) infarction.[4] RV infarction occurs when the RCA is occluded proximal to the origin of the RV branch which usually is given off by the RCA along its mid-course. Distal RCA occlusion that does not involve the RV branch results in an isolated IMI. However, not all cases of right ventricular artery occlusion can lead to RV infarction due to the presence of collaterals, multiple small RV branches or supply from the circumflex.[5] Therefore in this paper, we use the term RV involvement (RVI) rather than RV infarction.

Clinical sequelae of RVI vary widely and range from an asymptomatic condition to severe hypotension and cardiogenic shock.[6] Studies have indicated that RVI remains associated with significant morbidity and mortality, even in the mechanical reperfusion era with around 50% of these patients having poorer outcomes, which are usually related to profound hemodynamic and electrical complications.[6,7]

The presence of RVI appears to be underdiagnosed in patients with IMI.[7] It is, however, important to recognise the presence of RVI in these patients as the initial management is different from that of pure left ventricular infarctions.[7] The clinical manifestations can include hypotension and clear lung fields with elevated Jugular venous pressure. Recognition of RVI begins with identification of the ECG manifestations of inferior wall infarction (ST segment elevation in leads II, III and aVF) and the clinical signs of RVI. The signs of RVI on the standard 12 lead ECG in patients with IMI include ST segment elevation of >1mm in lead V1 either alone or along with, ST segment depression in lead V2, and higher ST segment elevation in Lead III than in Lead II.[8] However, confirmation of RVI can be made by obtaining right-sided ECG,(placing the chest leads on the right side of the chest corresponding to their original position). Presence of ST-segment elevation of >1 mm in lead V4R is considered significant and closely correlates with other non-invasive evidence of RV dysfunction with studies quoting a sensitivity of greater than 90%.[9,10]

The various ECG signs of RVI have varying sensitivities reported and are based on studies published mainly in the 1980s. The gold standard test to identify RVI in these studies were either myocardial perfusion imaging or post-mortem studies.[9,11-13] Recent studies using coronary angiography are lacking. In this study, we did not examine the presence of RVI per se, but rather occlusion of the RV branch, the aim of this study was therefore to assess the diagnostic accuracy of the different ECG criteria in identifying proximal RCA occlusion and the involvement its RV branch on angiography.

Methods

This was a retrospective case control study of patients who were admitted to our institution with a diagnosis of IMI. All patients who had presented with an IMI and who were taken up for a primary percutaneous coronary intervention between January 2014 and May 2016 were eligible to be included in the study. We excluded those with true posterior MI or anterior or lateral MI. The case notes of the eligible patients were reviewed, and the necessary information obtained. Ethical approval was obtained from the medical research committee (MREC) of the Sultan Qaboos university prior to commencing the study (Approval number 1345 and 1351 dated 30th August 2016).

A total of 253 patients had undergone primary PCI for a STEMI during this study period. Of these, 139 were IMI and the rest were either anterior wall (112) or isolated lateral (2) wall STEMI. Of those with documented IMI, 10 patients had incomplete data (missing ECG tracings) and so were excluded from the analysis.

The sample size was calculated with an estimated type 1 error of 5%, prevalence of RVI of around 33% of all IMI, 90% accuracy and a 10% confidence interval. Using these values, the sample size was calculated at 102. We had recruited more than the required number.

Patients were considered to have an IMI if they had at least 1mm ST segment elevation in two of the three inferior leads (leads II, III aVf). The ECG were analysed for presence of other anomalies such as conduction abnormalities or arrhythmias. The case notes were also reviewed for clinical presentation. The ECG criteria of RV involvement that we studied were ST elevation of >1mm in V4R, ST elevation of >1mm in lead V1 either on its own or along with ST depression in lead V2 and ST elevation in lead III higher than lead II. These are established criteria for identifying RVI in the presence of IMI.

Statistical analysis was performed using SPSS version 21. Data was presented as number (%) or mean $\pm$ standard deviation (SD) or median (Interquartile range- IQR). Data analysis was done by chi-square test or student t-test as appropriate (non parametric and normally distributed data respectively). Sensitivity, specificity, and positive and negative predictive value of the ECG changes were analysed. Sensitivity was calculated as a ratio of ECG and angio positive over total angiographic positive (true positive). Specificity was the ratio of ECG and angio negative over all angiographic negative (true negative). The positive predictive value was calculated as a ratio of ECG and Angiography

positive over all test positive. The negative predictive value was calculated as a ratio of ECG and angiography negative over all ECG negative. A p value of less than 0.05 was considered as significant.

Results

A total of 129 patients (mean age 55.8±13.1 years, 105 or 81.3% male) with inferior STEMI were enrolled our study (10 patients with IMI had missing ECG). All had presented with chest pain and ECG suggestive of inferior STEMI. Of these patients 54 had diabetes, 76 were hypertensive, 68 had dyslipidaemia and 56 were smokers. The mean systolic blood pressure of these patients was 131.28±29.3 mmHg with a mean heart rate of 77.3±18.6 bpm. Eight patients had syncope at the time of presentation.

All these patients were taken up for urgent primary PCI. Based on the angiographic findings, 16 patients had an occluded circumflex artery, and 113 patients had an occluded RCA of which 49 were occluded proximally before the origin of the RV branch indicating RV involvement and thus resulting in the incidence of possible RVI of 37.9% of all IMI. Table 1 shows the demographic features of the patients with and without RVI. There was no difference in age, gender, risk factors or hemodynamic features. There were also no differences in the ECG findings of conduction abnormalities or arrhythmias. However, patients with RVI were more likely to have breathlessness (42.8% vs 26.2%, p=0.05) and syncope (14.2% vs 1.2%, p=0.003). Although there was no difference in survival at one year, there was a trend towards higher mortality (16.4% vs 7.8%, p=0.19) in patients with RVI as compared to those without.

Table 1: Demographic features of patients with or without Right ventricular involvement

	No evidence of RV involvement (n=80)	**RV involvement (n=49)**	**P value**
Age (years)	55.1±13.6	56.8±12.6	0.49*
Sex	66 (82.5%)	39(79.5%)	0.68
Male	14 (17.5%)	10 (20.5%)	
Female			
Diabetes	34 (42.5%)	20 (40.8%)	0.92
Hypertension	47 (58.7%)	29(59.1%)	0.94
Dyslipidemia	41 (51.2%)	27(55.1%)	0.81
Smoker	33(41.2%)	23(28.7%)	0.76
Systolic blood pressure(mmHg)	129.3±26.5	134.4±32.9	0.31*
Heart rate(bpm)	76.61+16.5	78.4+2.1	0.59*
ECG rhythm change	10 (12.5%)	9(18.3%)	0.41
AV block	3(3.7%)	0	
RBBB	4(5%)	2(4.1%)	
Sinus arrhythmia	1(1.2%)	1 (2.0%)	
Atrial fibrillation	2 (2.5%)	3(6.1%)	
Junctional rhythm	0	1 (2.0%)	
LBBB			
Associated Symptoms	21 (26.2%)	21(42.8%)	0.05
Breathlessness	1 (1.2%)	7(14.2%)	0.003
Syncope			
Alive at one year	73 (91.2%)	41 (83.6%)	0.19

Bpm- beats per minute; AV- Atrio-ventricular; RBBB- Right bundle branch block; LBBB- Left bundle branch block
*Analysis by chi-square test except for * which was by students t-test*

There were ECG differences between the two groups [Table 2]. Those with RVI were more likely to have ST elevation in V4R (55.1% vs 38.7%, p=0.02), ST elevation in V1 (28.5% vs 10% p=0.004) and ST elevation in V1 along with ST depression in V2 (16.3% vs 3.7% p=0.004). There was no difference between the two groups in terms of higher ST elevation in lead III than lead II.

Table 3 gives the difference in the diagnostic accuracy of the different ECG criteria in diagnosing proximal RCA occlusion or RVI. ST elevation in V4R and ST elevation in III more than II were the most sensitive (72.9% and 80.4% respectively). However, the former was the least specific with a specificity of 15.4% (Indeed Table 2 showed that it was present in most cases of IMI). ST elevation in V1 by itself and ST elevation in V1 along with ST depression in V2 had very high specificity of 88.7% and 97.1% respectively. Combining all the criteria increased sensitivity to 85% while reducing the specificity (21.2%).

Table 2: ECG changes in patients with or without RV involvement

	No RV involvement (n=80)	**RV involved (n=49)**	**P value**
ST elevation V4R	31 (38.7%)	27 (55.1%)	0.02
ST elevation V1	8 (10%)	14 (28.5%)	0.004
ST elevation III > II	60 (75%)	37 (75.5%)	0.22
ST elevation V1, ST depression V2	3 (3.75%)	8 (16.3%)	0.004
Any of the above	63(78.7%)	42 (85.7%)	0.02

Analysis by Chi-square test

Table 3: Diagnostic accuracy of the different ECG criteria

	Sensitivity	**Specificity**	**Negative predictive value**	**Positive predictive value**
ST elevation V4R	72.9%	50.7%	46.5%	76.1%
ST elevation V1	30.4%	88.7%	66.3%	63.6%
ST elevation III > II	80.4%	15.4%	55%	38%
ST elevation V1, ST depression V2	17.3%	97.1%	63.8%	72.7%
Any of the above	85.7%	21.2%	70.8%	40%

Discussion

The incidence of proximal RCA occlusion and thereby possible RVI in patients with IMI was 37.9% in our study, which is comparable to those previously reported.[4] Those with RVI were more likely to have breathlessness and syncope on presentation. There were no other differences in terms of demography or presentation between the two groups. Unfortunately, there was no documentation of other clinical signs (Jugular venous pressure or pedal oedema or lung signs) in many patients and hence we did not include that in the analysis.

Our findings suggest that the presence of a higher ST elevation in lead III was the most sensitive of the different criteria. This could be because lead III is more "rightward facing" than lead II and hence more sensitive to the injury current produced by the right ventricle.[10,11] ST segment elevation in the right sided leads (V4R) was surprisingly absent in almost half of patients with RV involvement. ST elevation in V4R has been documented previously to be a transient event which can disappear in half of patients beyond 12 hours.[11,14,15]

While ST elevation in V4R and ST elevation in lead III greater than II were the most sensitive, the other criteria i.e. ST elevation in V1 and ST elevation in V1 with ST depression in V2 were the most specific. Combining all the four studied criteria further increased the sensitivity to around 85%, but at the cost of specificity. Even with all the criteria combined, there were still a few patients that had proximal RCA occlusion but did not have the corresponding ECG changes. Our findings are fairly similar to those obtained by Zimetbaum et al, who also found ST elevation in lead III greater than II along with ST elevation in V1 to be a strong predictor of proximal RCA occlusion.[16] Besides the studies mentioned, there are very few studies looking at the ECG findings in RVI and even those are more than 30 years old. There are almost no studies during the angioplasty era besides case reports during accidental occlusion of the RV branch during angioplasty as described below.

One of the reasons for this low sensitivity and specificity of ECG findings to predict RVI or RV infarction is due to the pathophysiology involved. The RV wall is thin and therefore changes may not be as pronounced as it is with

left ventricular infarction. During the IMI with RVI, the current of injury from the larger inferior LV wall mask those originating from the infarcted RV.[17] During an Inferior or posterior MI, the corresponding parts of the interventricular septum may also get infarcted giving changes in the RV leads.[8,10,17]

Animal experiments have demonstrated that occluding the RV branch would give rise to pure ST segment elevation in the right sided precordial leads (V4R-V6R).[8,18] However, finding an isolated human RV infarct is extremely rare and are limited to case reports.[19,20] Isolated RV infarction has also been described during PCI where the RV branch of the RCA gets occluded.[21] In both these scenarios, RV infarction has been associated with ST elevation in the precordial leads (V1-V4) along with the right sided leads (V4R-V6R). The absence of development of Q waves and progressive reduction in the size of the ST elevation in the anterior precordial leads has been suggested as a sign that would help differentiate an anterior MI from the rare isolated RV infarct.[18,20] Additionally, as mentioned earlier, proximal occlusion of the RCA and thereby occlusion of the RV branch, does not always lead to RV infarction as in a small proportion of patients, the RV is supplied by multiple small branches, collaterals and even branches from the circumflex.[5]

Even in the era of primary angioplasty, early recognition of RVI is still important. It should be part of the evaluation of all patients with IMI and its presence should be considered in all these patients especially those who are hypotensive as it will affect decisions on fluid management.[7,22] A quick bedside echocardiogram could help in the evaluation of RV involvement prior to shifting the patient to the catheter lab for an urgent angiogram if RVI is suspected and the ECG is not clear.[23,24] However, in practice, many of these patients are rushed to the catheter lab for primary angioplasty as soon as they arrive.

The retrospective nature of this study is a limitation as retrospective studies have inherent limitations. It depends on the quality of case note documentation. For example, many patients did not have detailed clinical examination findings. It was not possible to verify the information that was documented and we have to accept what was documented. We however were able to obtain some of the missing data by contacting the patients and verifying some of the information such as demographics and risk factors.

The sample size was low in our study but comprised all patients who presented for one year and was sufficient for analysis. It was however, greater than the sample size calculated and therefore the results are valid. The clinical presentation was not clearly documented for a sizeable number of patients. It is likely that as these patients would have been rushed in for a primary angioplasty upon arrival, the initial documentation was poor. Similarly, none of the patients had echocardiograms prior to their angioplasty, in order to reduce the door to balloon time. Pre-procedure echocardiography might have helped in making a diagnosis of RVI. In any case, this study was mainly to assess the effectiveness of the ECG criteria in diagnosing RV branch occlusion (proximal RCA occlusion) and thereby RVI. We do not have follow up on these patients to assess the prognosis of having RVI. Many of these patients were transferred from regional hospitals and the follow up was with them. It was difficult to obtain the necessary information. Again, this was out of the scope of the study, but previous studies, mainly from the thrombolysis era been reported to be associated with a poor prognosis.[6,14,15] It would be worthwhile studying the effects of RV involvement in the setting of primary angioplasty, to assess whether RVI still has the same poor prognosis as during the thrombolysis era. This is feasible and would help to further our understanding of RV physiology and its impact on long term prognosis. The strength of this study is in the fact that we used angiographic evidence for RV branch involvement to study the diagnostic efficacy of the different ECG criteria.

Conclusions

No single ECG criteria have shown high sensitivity combined with high specificity levels for appropriate electrocardiographic diagnosis of RVI in the setting of inferior STEMI. However, the ECG can guide the decision-making in patients with inferior STEMI, though careful attention to the clinical picture of the patients (signs and symptoms) remains the most important tool to diagnose RVI and subsequently to help decision-making about treatment strategy.

List of abbreviations

ACS- Acute coronary syndrome

STEMI- ST segment elevation myocardial infarction

NSTEMI- non ST segment elevation myocardial infarction

IMI- inferior myocardial infarction

RCA- Right coronary artery

RV- right ventricle

RVI- Right ventricular involvement

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