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PREDICTIVE ROLE OF ANGIOGRAPHIC AND ECHOCARDIOGRAPHIC PARAMETERS IN ACUTE RIGHT VENTRICULAR INFRACTION DIAGNOSED USING ELECTROCARDIOGRAPHY

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Abstract

Right coronary artery (RCA) emerged as an infarct-related artery (IRA) in patients with right ventricular myocardial infarction (RVMI), which is followed by some degree of RV dysfunction. The aim of our study was to identify significant predictors of IRA among angiographic and/or echocardiographic data of RV dimension and/or function in patients with acute RVMI.

Out of 122 hospitalized patients with acute inferior myocardial infarction, on the basis of electrocardiographic (ECG) criteria, RVMI was diagnosed in 58/47.5% of patients. Coronary angiography was applied in 52/89,6% immediately after admission and conventional 2D echocardiography was performed in order to assess right heart dimensions and function according to the professional association recommendations.

RCA emerged as IRA in 49/84,5 % of patients with ECG-diagnosed RVMI. Patients with RCA stenosis/occlusion had 4.9 times higher risk for RVMI presence (OR=4.941; 95%CI: 1.727-14.136; p=0.003) than those without and had significantly worse echocardiographic assessed RV global and/or regional systolic function. Logistic stepwise regression analysis confirmed the significant role of enlarged RV dimension (OR=1.1; 95%CI: 1.023-1.182; p=0.010), RCA stenosis/occlusion presence (OR=4.8; 95%CI: 1.649-14.199; p=0.004) and/or LAD stenosis/occlusion absence (OR=0.18; 95%CI: 0.067-0.476; p=0.001) in the prediction of RVMI. The optimal sensitivity of the model was 90% and the specificity was 75%.

RCA and conversely lack of LAD stenosis/occlusion presence along with some of the echocardiographic parameters showing RV dysfunction increased the odds for RVMI. Applying immediate and complete reperfusion of RCA is of great importance for the recovery of RV function.

Keywords: right ventricular myocardial infarction, right coronary artery, right ventricular dysfunction.

Introduction

The incidence of right ventricular myocardial infarction (RVMI) in patients with inferior wall myocardial infarction (MI) is described in the literature in a wide range of percentages (25-50%), mainly depending on the diagnostic criteria [1-4].

Suspicion of RVMI presence usually is established according to the determined ECG features with variable sensitivity and specificity that includes ST-segment elevations in inferior and precordial lead V1 and its relation, ST-segment depression in lead V2 along with ST-segment elevation ≥0.5 mm in right-sided precordial leads (V3R and V4R) [5-10].

However, conventional transthoracic echocardiography and angiography are methods widely recognized in a process of RVMI confirmation. The presence of RV dilation, depressed RV systolic function, RV regional wall motion abnormalities (RV WMA), and/or elevated pulmonary pressures are pathological changes frequently associated with RVMI presence [11-16].

Such echocardiographic changes are a strong foundation for emerging complications and hemodynamic impairment that leads to RV dysfunction responsible for significantly increased morbidity and mortality in patients with RVMI [6, 17-19]. In addition, angiographic studies revealed that the right coronary artery (RCA) was the most often the infarct-related artery (IRA), especially, its proximal part [20-23].

However, recent studies suggest that in patients with the acute coronary syndrome, successful reperfusion achieved by fibrinolysis and/or primary coronary angiography (PCA) can provide significant improvement in RV function and thus improve survival in those with RV dysfunction [9,24]. Early recognition of RVMI is essential for the reduction of mortality and the complications caused by it.

Our study aimed to identify a significant predictor of IRA between angiographic and/or echocardiographic data that could strongly suggest the presence of RVMI to overcome possible RV dysfunction and worse prognosis with timely intervention.

Methods

Study population

This was a prospective, case-control study examining 122 consecutive patients admitted to the intensive cardiac care unit (ICCU) at the University clinic of cardiology, Skopje with chest pain in the last 2-12 hours and ECG signs of acute MI of the inferior, inferior/posterior and/or lateral wall of the left ventricle (LVMI). The presence of RVMI was determined according to the established ECG criteria for its presence. [5-10] 12-channel ECG with at least one right precordial lead (V4R) was made in every patient with LVMI including right-sided precordial lead (V4R) at admission and in the consecutive 6, 12, and 24 h after. Patients were divided into two groups: those with at least two fulfilled ECG criteria for RVMI and the control group without any of them.

Echocardiography

Immediately after admission to ICCU, just before angiography, conventional 2D echocardiography was performed on commercially available equipment (Vivid 5 and/or 7; GE, USA). All patient's data were recorded in order to perform a more profound analysis later on by an echocardiographer blinded for angiographic data. Standard assessments of LV systolic and diastolic function were performed according to the professional association recommendations [16]. In addition right atrial (RA) and RV dimensions along with RV global and regional systolic function were assessed using the same equipment according to the professional association recommendations [15,16].

Thus, RV systolic function has been evaluated using several parameters: fractional area change (FAC%), tissue Doppler-derived tricuspid lateral annular systolic velocity (TDI), tricuspid annular plane systolic excursion (TAPSE) and pulmonary artery systolic pressure assessed using tricuspid regurgitation and diameter and collapsibility of inferior vena cava (IVC) [15,16].

Coronary angiography

Coronary angiography was applied in 52/89,7% immediately after admission to ICCU. Coronary angiograms were analyzed to determine the localization, severity, and complexity of stenosis, and the Syntax score was calculated.[24] A successful revascularization procedure (PCI) was considered when a TIMI epicardial flow grade 3 was restored.

Statistical analysis

Categorical parameters were summarized as percentages and continuous parameters as a mean ± standard deviation. The difference in clinical, echocardiographic, and angiographic parameters between the two groups was tested using the Mann-Whitney U test for continuous variables and Pearson's Chi-square test for categorical variables. Assessment of correlation was done using Spearman's correlation analysis. Multiple logistic regression analysis was performed to determine independent predictors of angiographic and/or echocardiographic data in the prediction of RVMI. All data analysis was performed using SPSS version 25.0 (IBM SPSS, Inc., Chicago, Illinois, USA), and $p \leq 0.05$ was considered as statistically significant.

Results

As presented in Table 1, baseline characteristics were rather similar in both groups of patients with and without electrocardiographically proven RVMI. At admission patients with RVMI had lower systolic and diastolic blood pressure, significantly lower heart rate as well as slightly worse Killip class (Table 1). Out of 58 patients with RVMI, 57 (98.3%) had ECG signs of inferior wall MI which was significantly more frequent than in patients without RVMI (p=0.0001) who on the contrary showed more frequently infero-posterior localization of MI (p=0.0001) (Table 1).

Table 1. Demographic, clinical and ECG characteristics in patients with and without RVMI.

Parameter	With RVMI n= 58	Without RVMI n=64	*p*
Age (years)	58.95±10.35	56.16±11.91	0.138
Gender: men/women (%)	75.9/24.1	76.6/23.4	0.548
BMI (kg/ m^2)	27.65±4.71	27.28±4.09	0.621
Smoking (n/%)	31/53.4	42/65.6	0.118
Dyslipidemia (n/%)	18/31.0	21/32.8	0.494
Hypertension (n/%)	36/62.1	39/60.9	0.523
Diabetes mellitus (n/%)	17/29.3	21/32.8	0.494
SP (mmHg)	135.09±25.12	140.86±25.46	0.208
DP (mmHg)	81.98±15.47	84.38±12.99	0.374
HR (Imp/min)	72.67± 19.00	81,13±18.47	0.026
Killip	1.17± 0.42	1.16±0.36	0.822
Inferior wall ST-elevation MI (n/%)	57/98.3	20/31.2	0.0001
Infero-posterior wall ST-elevation MI (n/%)	1/1.7%	42/65.6	0.0001

ECG=electrocardiography; RVMI=right ventricular myocardial infarction; BMI=body mass index; SP=systolic pressure; DP=diastolic pressure; HR=heart rate; MI=myocardial infarction.

Coronary angiography with or without percutaneous intervention was applied in 52 patients with RVMI (89,6%) and in 59 patients (92,1%) without RVMI. Significant coronary stenosis (≥50-99%) or complete occlusion of the right coronary artery (RCA) was observed significantly more frequently in patients with RVMI according to the ECG criteria in comparison to those without (p=0.0001) with the opposite frequency regarding significant coronary stenosis or complete occlusion of the left anterior descending artery (LAD) (p=0.0001) (Table 2). However, significant stenosis of the left circumflex artery (LCx) was almost identically distributed in those with and without RVMI (p=0.275) and the frequency of stenosis of other coronary arteries was without significance between the two groups (Table 2).

Table 2. Angiographic characteristics in patients with and without RVMI.

Parameter	With RVMI n= 52	Without RVMI n=59	*p*
RCA (n/%)	49/89.1	29/51.8	0.0001
RCA Proximal Mid Distal	 28/57.1 8/16.3 13/26.5	 12/41.4 13/44.8 4/13.8	0.021
LAD (n/%)	22/40	46/81.1	0.0001
LCx (n/%)	23/39.7	21/32.8	0.275
OM (n/%)	5/8.6	9/14.1	0.257
D1 (n/%)	8/13.8	15/23.4	0.129
PCI (n/%)	50/86.2	59/92.2	0.219

RVMI=right ventricular myocardial infarction; RCA=right coronary artery; LAD=left aterior descendens;LCx=circumflex coronary artery; OM=obtusa marginalis; D= first diagonal branch; PCI=percutaneous coronary intervention.

In the patients with RVMI, the analysis of the localization of the lesions in RCA showed a significantly higher frequency of the proximal segments in comparison to the mid and/or distal segment and/or regarding the distribution in patients without RVMI (p=0.021).

Such results were confirmed using multivariate logistic regression analysis which showed that patients with RCA stenosis and/or occlusion presence were almost five times more likely to have RVMI (OR=4.941; 95%CI: 1.727-14.136; p=0.003) while the absence of LAD disease was associated with lower odds for RVMI presence (OR=0.209; 95%CI: 0.84-0.524; p=0.001)(Table 3).

Table 3. The results of the multivariate logistic regression analysis with RVMI as the dependent variable and stenosis and/or occlusion of RCA or LAD as independent variables.

	B	**Wald**	**Sig.**	**Exp (B)**	**95% CI for Exp(B)**	
					Lower	Upper
RCA	1,598	8.872	0,003	4,941	1,727	14,136
LAD	-1.563	11.164	0,001	0,209	0,84	0,524

RCA=right coronary artery; LAD=left aterior descendens.

In order to assess and confirm the presence of RVMI, the echocardiographic parameters of right heart dimensions and function were analyzed and compared in patients with ECG signs of RVMI and without it (Table 4). Patients with RVMI in comparison to those without had a larger right atrial area (p=0.083) and mid RV dimension (p=0.026), significantly lower FAC% (p=0.01), and TAPSE (p=0.006) as a sign of reduced RV global and regional function respectively, almost identical reduced RV TDI and higher, but still normal systolic PAP (p=0.047) (Table 4).

Table 4. Echocardiographic measurements of right heart dimensions and function in patients with and without RVMI.

Parameter	**With RVMI n= 58**	**Without RVMI n=64**	***p***
RA area (cm^2)	16.94 ± 4.08	15.42 ± 3.27	0.083
RV mid (mm)	33.40 ± 6.44	30.36 ± 7.09	0.026
FAC (%)	47.44 ± 11.05	53.76 ± 8.99	0.001
TAPSE (mm)	16.66 ± 2.98	18.14 ± 2.43	0.006
RV s'TDI (cm/s)	13.05 ± 3.06	13.78 ± 3.39	0.213
PAPs (mmHg)	23.85 ± 12.19	19.93 ± 13.07	0.047

RVMI=right ventricular myocardial infarction; RA=right atria; RV=right ventricle; FAC= fractional area change; TAPSE=tricuspid annular plane systolic excursion; PAPs= pulmonary artery systolic pressure.

In order to identify variables independetly associated with RVMI, angiographic along with echocardiographic variables of RV dimensions and/or function (RA area, RV dimension, FAC, TAPSE, RVs'TDI, PAPs, represented in table 4), logistic stepwise regression analysis adjusted for age and gender was performed and three independent variables were identified (Table 5): enlarged RV mid-dimension (OR=1.1; 95%CI: 1.023-1.182; p=0.010), RCA disease (OR=4.8; 95%CI: 1.649-14.199; p=0.004) and/or LAD disease absence (OR=0.18; 95%CI: 0.067-0.476; p=0.001).

Thus, those who had RCA stenosis and/or occlusion in comparison to those who hadn't were 4.8 times more likely to have RVMI which was the highest odds among other significant variables that

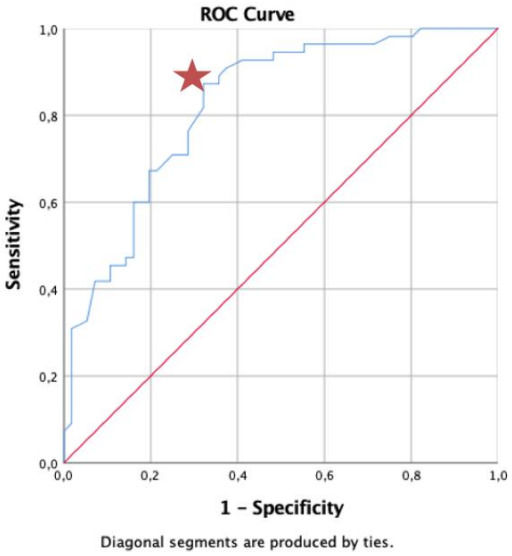
enter the model. Although LAD desease absence possessed the lowest odds of having RVMI, its contribution to the model was very important (Table 5).

Table 5. The results of the multivariate logistic regression analysis with RVMI as the dependent variable and RV dimensions and function along with stenosis and/or occlusion of coronary arteries (RCA or LAD) as independent variables.

	B	**Wald**	**Sig.**	**Exp (B)**	**95% CI for Exp(B)**	
					Lower	Upper
RCA	1.577	8.244	0,004	4,839	1,649	14,199
LAD	-1.722	11.862	0,001	0,179	0,067	0,476
RV mid (mm)	0.095	6.608	0.01	1.100	1.023	1.182

RCA=right coronary artery; LAD=left aterior descendens; RV mid= right ventricular mid dimension.

The area under the curve (ROC) was 0.823 (Fig.1) which means that in 82% of all possible pairs of subjects with and without RVMI, this model will assign a higher probability to the subjects with RVMI. The optimal sensitivity was 90% and the specificity was 75%.



Area Under the Curve

Test Result Variable(s): Predicted probability

Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
,823	,039	,000	,746	,900

The test result variable(s): Predicted probability has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased.

a. Under the nonparametric assumption

b. Null hypothesis: true area = 0.5

Figure 1. ROC curve of the probability of the model and area under it.

Discussion

The results of our study showed that RCA emerged as IRA in 89.1% of patients with RVMI, especially its proximal part. Patients with RCA stenosis/occlusion had a higher risk for RVMI presence than those without RCA changes. Of note is that in our study the patients with RCA stenosis/occlusion were 4.8 times more likely to have RVMI which was the highest odds among other significant predictive variables. Occasionally, the left circumflex or LAD can be the culprit for RVMI, but exactly the absence of LAD disease had a higher risk for RVMI presence as we showed in our study. Such results were in line with numerous angiographic studies that confirm RCA involvement as IRA in a similar percentage, especially its proximal part [3, 9, 20-23, 25, 26].

Given that RV function is predominantly determined by the RV free wall, which receives blood flow primarily from RV branches of the RCA, in circumstances of RCA occlusion it is imminent to have some degree of RV dysfunction. Furthermore, the results of our study showed that patients with ECG

diagnosed RVMI in comparison to those without had worse RV systolic global and regional function manifested by significantly lower FAC% and TAPSE, larger RV inner dimension, and higher PAPs. In addition, the result of our study revealed enlarged RV mid-dimension as significant predictor of RVMI.

These finding were consistent with the data from the literature that showed deterioration of RV function due to the acute RCA proximal occlusion. Among the data few features were found to be useful in predicting proximal RCA stenosis: enlarged RV mid-dimension, reduced TAPSE, myocardial performance index by tissue Doppler imaging (MPI-TDI) and velocity of the tricuspid annular systolic motion (RV S') as well reduced global and/or free-wall RV longitudinal strain detected either with echocardiography or with cardiac magnetic resonance [26-31].

Therefore, the stenosis/occlusion of RCA as IRA is an extremely important finding in RVMI existence taking into account its capability to induce RV dysfunction which fortunately could be restored with successful complete reperfusion of the RCA. Since the results of the experimental study of Laster et al. [20], who emphasized the deleterious impact of the duration of ischemia on RV function and implicated that the effects of reperfusion on the recovery of RV function could be beneficial, numerous studies in patients with acute RVMI demonstrated that successful complete reperfusion of the RCA, including flow restoration to the major RV branches done in a timely manner mainly, leads to immediate and/or quick improvement and later complete recovery of RV function [23,32,33].

In this respect, Kidawa et al. [34] selected 70 patients out of 153 with ECG signs of RVMI who had proximal occlusion of dominant RCA before the origin of the RV branch and applied echocardiography in order to assess RV function. They found that RV function assessed using RV echocardiographic parameters was less affected in patients with a shorter time from pain onset to the opening of the occluded RCA. Therefore the authors concluded that the time to reperfusion is crucial for the preservation of ventricular function in patients with RVMI, because delayed restoration of TIMI flow grade 3 significantly exposes the RV to dysfunction. Furthermore, in another study by Bowers et al. [21] RVI occurred in 53 (42%) out of 125 patients with acute inferior MI who had occlusion of the proximal part of RCA as a culprit vessel which was why the RV dysfunction appeared. In addition, this study succeed to document a direct correlation between the extent of RV branch flow compromise and the magnitude of RV dysfunction and stressed the value of assessment of RV branch perfusion status.

The importance of the proximity of RCA and its branch perfusion status arises from two extremely important aspects: The first one is the finding that more proximal lesions were predictive of hemodynamic deterioration and very likely worse prognosis as we have already pointed out in this discussion, and the Second one is the tendency of the RVMI to involve the right atrium (RA) as well, which is supplied by branches arising from the proximal RCA and in which circumstances the compensatory augmentation of RA contraction in the enhancement of RV filling and performance will be lost [3, 21, 23, 35, 36].

Limitations of the study

Our study has several potential limitations. First, the diagnosis of RV myocardial involvement was based exclusively on ECG criteria, even though RV myocardial involvement is often considered to be a clinical syndrome with symptoms that indicate its presence. Second, the study population should have been larger, especially with the coronary angiography, which would have provided greater normality in the distribution of patients and would have reduced confidence interval width. Third, despite we have made an adjustment for different confounding factors, undetected and/or unmeasured factors could still exist. And finally, the current study is a single-center study and it would have been better if the results will be confirmed in multicentric studies.

Conclusion

Right ventricular myocardial infarction (RVMI) is frequent in inferior myocardial infarction presenting with typical ECG findings. RCA is the most frequent IRA, especially its proximal part which leads to enhanced risk for RVMI presence.

It was confirmed that whenever RCA occlusion and especially if RV branch flow was compromised, some degree of RV dysfunction emerged. Thus, taking into account the RCA stenosis/occlusion presence and lack of left anterior descending (LAD) presence along with some of the echocardiographic parameters showing RV dysfunction, the odds of RVMI increased with substantial-high sensitivity (90%) and respectful specificity (75%).

Applying immediate and complete reperfusion of RCA and avoiding compromising the side branches is the cornerstone of prompt and profound recovery of RV function which means a better long-term prognosis.

References

1. Andersen HR, Falk E, Nielsen D. Right ventricular infarction: frequency, size, and topography in coronary heart disease: a prospective study comprising 107 consecutive autopsies from a coronary care unit. *J Am Coll Cardiol* 1987;10:1223-32.
2. Kinch J W and Ryan TJ. Right ventricular infarction. *N Engl J Med.*1994;330:1211-7.
3. Goldstein JA. Pathophysiology and management of right heart ischemia. *J Am Coll Cardiol* 2002; 40:841-53.
4. Isner JM, Roberts WC. Right ventricular infarction complicates left ventricular infarction secondary to coronary heart disease. Frequency, location, associated findings, and significance from analysis of 236 necropsy patients with acute or healed myocardial infarction. *Am J Cardiol* 1978;42:885-94.
5. Lopez-Sendon J, Coma-Canella I, Alcasena S, et al. Electrocardiographic findings in acute right ventricular infarction: Sensitivity and specificity of electrocardiographic alterations in right precordial leads V4R, V3R, V_1, V_2, and V_3. *J Am Coll Cardiol* 1985;6:1273–1279.
6. Zehender M, Kasper W, Kauder E, et al. Right ventricular infarction as an independent predictor of prognosis after acute myocardial infarction. *N Engl J Med* 1993;328:981-8.
7. Somers MP, Brady WJ, Bateman DC, et al. Additional electrocardiographic leads in the ED chest pain patient: right ventricular and posterior leads. *Am J Emerg Med.* 2003;21:563–573.
8. Zimetbaum PJ, Josephson ME. Use of the electrocardiogram in acute myocardial infarction. *N Engl J Med* 2003;348:933–40.
9. Verouden NJ, Barwari K, Koch KT, et al. Distinguishing the right coronary artery from the circumflex coronary artery as the infarct-related artery in patients undergoing primary percutaneous coronary intervention for acute inferior myocardial infarction. *Europace* 2009;11:1517-21.
10. Thygesen K, Alpert JS, Jaffe AS, et al on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/ American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction. Fourth universal definition of myocardial infarction (2018). *Eur Heart J* 2018; 00:1-33.
11. Yasuda T, Okada RD, Leinbach RC, et al. Serial evaluation of right ventricular dysfunction associated with acute inferior myocardial infarction. *Am Heart J* 1990;119:816-22.
12. Goldberger JJ, Himmelman RB, Wolfe CL, et al. Right ventricular infarction: recognition and assessment of its hemodynamic significance by two-dimensional echocardiography. *J Am Soc Echocardiogr.* 1991;4:140-6.
13. Jiang, L, Levine RA, Weyman AE. Echocardiographic assessment of right ventricular volume and function. *Echocardiography* 1997*; 14*: 189–206.
14. Horton KD, Meece RW and Hill JC. Assessment of the right ventricle by echocardiography: A primer for cardiac sonographers. *J Am Soc Echocardiogr* 2009;22:776-92.
15. Rudski LG, Lai WW, Afilalo J, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. *J Am Soc Echocardiogr* 2010; 23:685–713.
16. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr* 2015; 28:1–39.e14.
17. Berger PB, Ryan TJ. Inferior myocardial infarction: high-risk subgroups. *Circulation* 1990;81:401-11.
18. Mehta S, Eikelboom J, Natarajan M, et al. Impact of right ventricular involvement on mortality and morbidity in patients with inferior myocardial infarction. *J Am Coll Cardiol* 2001;37:37-4.
19. Jacobs AK, Leopold JA, Bates E, et al. Cardiogenic shock caused by right ventricular infarction. A Report from the SHOCK Registry. *J Am Coll Cardiol* 2003;41:1273-9.

20. Laster SB, Ohnishi O, Safitz JE, Golstein JA. Effects of reperfusion on ischemic right ventricular dysfunction. Disparate mechanism of benefit related to the duration of ischemia. *Circulation* 1994; 90:1398-409.
21. Bowers TR, O'Neill WW, Pica M, and Goldstein JA. Patterns of the coronary compromise resulting in acute right ventricular ischemic dysfunction. *Circulation* 2002; 106:1104-9.
22. Tomala M, Miszalski-Jamka T, Zajdel W, et al. Angiographic result of index PCI determines the presence of right ventricular infarction in patients with acute inferior myocardial infarction. *Int J Cardiovasc Imaging*. 2015; 31(8):1591-601.
23. Bowers TR, O'Neill WW, Grines C, et al. Effect of reperfusion on biventricular function and survival after right ventricular infarction. *N Engl J Med.* 1998*;* 338:933–940.
24. Sianos G, Morel M-A, Kappetein AP, et al. The SYNTAX Score: an angiographic tool grading the complexity of coronary artery disease. *EuriInterv* 2005;1:219-27.
25. Albulushi A, Giannopoulos A, Kafkas N, et al. Acute right ventricular myocardial infarction. *Expert Rev Cardiovasc Ther* 2018;16:455-64.
26. Femia G, French JK, Juergens C, et al. Right ventricular myocardial infarction: pathophysiology, clinical implications and management. *Rev Cardiovasc Med* 2021;22:1229-40.
27. Ashmawy MM, El-Barbary YH, Hassib WA and Naguib CG. Assessment of right ventricular function in acute inferior wall myocardial infarction in patients treated with primary percutaneous coronary intervention. *Res J Cardiol* 2016; 9:8-16.
28. Baghel PK, Tripathi SK, Vehab AA, Aggarwal P. Assessment of right ventricular function in patients with acute myocardial infarction. *Cureus* 2022; 14:e22399.
29. van Veelen A, Elias J, van Dongen IM, et al. Recovery of right ventricular function and strain in patients with ST-segment elevation myocardial infarction and concurrent chronic total occlusion. *Int J Cardiovasc Imaging* 2022;38:631-44
30. Erley J, Tanacli R, Genovese D, et al. Myocardial strain analysis of the right ventricle: comparison of different cardio- vascular magnetic resonance and echocardiographic techniques. *J Cardiovasc Magn Reson* 22(1):51
31. Taha K, Bourfiss M, Te Riele A, et al. A head-to-head comparison of speckle tracking echocardiography and feature tracking cardiovascular magnetic resonance imaging in right ventricular deformation. *Eur Heart J Cardiovasc Imaging* 2021; 22:950-8
32. Zehender M, Kasper W, Kauder E, et al. Eligibility for and benefit of thrombolytic therapy in inferior myocardial infarction: Focus on the prognostic importance of right ventricular infarction. *J Am Coll Cardiol* 1994; 24:362-9.
33. Berger PB, Ruocco NA Jr, Ryan TJ, et al, and the TIMI research group. Frequency and significance of right ventricular dysfunction during inferior wall left ventricular myocardial infarction treated with thrombolytic therapy: Results from the Thrombolysis in Myocardial Infarction (TIMI II) trial. *Am J Cardiol* 1993;71:1148-52.
34. Kidawa M, Kasprzak JD, Wierzchowski T, Krzeminska-Pakula M. Right ventricular function suffers from reperfusion delay: Tissue Doppler Study. *Clin Cardiol* 2010;22:E43-E48.
35. Goldstein JA, Lee DT, Pica MC, et al. Patterns of coronary compromise leading to bradyarrhythmias and hypotension in inferior myocardial infarction. *Coron Artery Dis* 2005; 16: 265-274.
36. Goldstein JA. Hemodynamic complications of right ventricular infarction. Role of the right atrium. *JACC: Case Reports* 2021; 3:1174-6.