Cardiology and Cardiovascular Research
2022; 6(1): 45-49
http://www.sciencepublishinggroup.com/j/ccr
doi: 10.11648/j.ccr.20220601.17
ISSN: 2578-8906 (Print); ISSN: 2578-8914 (Online)

Angiographic Correlates of Acute ST Elevation Inferior Wall Myocardial Infarction with or Without Right Ventricular Involvement

Soumik Ghosh[1, *], Salini Mukhopadhyay[2], Tusharkanti Patra[3]

[1]Department of Cardiology, Institute of Medical Sciences, Banaras Hindu University, Varanasi, India

[2]Department of Anesthesiology, Institute of Postgraduate Medical Education and Research, Kolkata, India

[3]Department of Cardiovascular Science, Institute of Postgraduate Medical Education and Research, Kolkata, India

Email address:

Soumik13joy@gmail.com (S. Ghosh), drsalini2016@gmail.com (S. Mukhopadhyay), doctushar.tp@gmail.com (T. Patra)

[*]Corresponding author

To cite this article:

Soumik Ghosh, Salini Mukhopadhyay, Tusharkanti Patra. Angiographic Correlates of Acute ST Elevation Inferior Wall Myocardial Infarction with or Without Right Ventricular Involvement. *Cardiology and Cardiovascular Research*. Vol. 6, No. 1, 2022, pp. 45-49. doi: 10.11648/j.ccr.20220601.17

Received: March 2, 2022; **Accepted**: March 26, 2022; **Published**: March 31, 2022

Abstract: Objectives: Right ventricular infarction (RVI) poses as an added risk factor in patients presenting with acute ST elevation inferior wall myocardial infarction (IWMI) with considerable high mortality. An early interventional therapeutic strategy after a prompt and accurate non-invasive investigative correlate is needed. Material and methods: We sampled 104 patients diagnosed with inferior wall infarction presenting with angina within 12 hours of angina. Investigations included routine blood investigation, 12 lead and right precordial lead electrocardiography, right ventricular (RV) systolic echocardiographic indices, and coronary angiography. Results: Majority of the patients had angiographic evidence of a dominant distal right coronary artery (RCA) culprit lesion. Those patients having ST elevation in RV4 lead had significantly higher incidences of RVI and high-grade atrio-ventricular (AV) blocks. Elderly diabetic patients with azotemia and deranged liver function predicted RVI among the study population. RV systolic indices like TAPSE was most accurate and S' was found to be most specific in detecting RVI. Chi square test and multivariate regression analysis of echocardiographic parameters like RVDD, RVMPI, and S' proved excellent surrogate non-invasive surrogate markers for specific angiographic culprit lesions. Conclusion: RV systolic echocardiographic indices shows a diagnostic accuracy of variable degrees in detecting right ventricular involvement in IWMI patients and also act as a surrogate marker in predicting the culprit lesion.

Keywords: Inferior Wall Myocardial Infarction, Right Ventricular Infarction, Coronary Angiography, Right Ventricular Systolic Indices, Atrio-ventricular Block

1. Introduction

Inferior wall myocardial infarction (IWMI) is generally regarded as a low-risk entity, compared with anterior wall MI. However, right ventricular infarction (RVI), precordial ST-segment depression and complete atrio-ventricular (AV) block have been proved as the three high-risk subsets in IWMI cases. [1] Isolated RVI is rare and usually occurs in association with IWMI. [2, 3] The incidence is considerably higher in autopsy series. [4] A possible explanation for this discrepancy is the ambiguity in diagnosing the disease occurrence in living cohorts. Currently ST-segment elevation in electrocardiographic lead V4R is the single most powerful predictor of right ventricular involvement in the setting of IWMI. [5] When complicated by RVI, in-hospital mortality is higher, as compared with isolated inferior MI. [6] Early diagnosis and prompt treatment of this association carries considerable prognostic importance.

The clinical consequences of right ventricular involvement were first described by J N Cohn only in 1974, which vary from no hemodynamic compromise to severe hypotension and cardiogenic shock. [7] Diagnosis is based on physical examination, electrocardiography, echocardiography and

coronary angiography. Because the standard 12-lead electrocardiogram is insufficient for the assessment of RV involvement, right-sided precordial leads should always be included. [8] Right ventricular involvement appears to be an independent prognostic factor that dramatically increases in-hospital mortality, due, in part, to a significantly higher risk of hemodynamically compromising arrhythmias. [9]

In this study, we aim to interrogate the angiographic correlates of inferior wall myocardial infarction with or without right ventricular involvement with various electrocardiographic and echocardiographic parameters.

2. Methodology

One hundred and four consecutive patients presenting with acute onset chest for the first time and diagnosed with inferior wall myocardial infarction within 12 hours of onset of angina, who gave positive response to informed consent were included in the study. It was an institutional, observational study conducted in a tertiary care teaching hospital of India over 6 months' duration. Exclusion criteria were previous history of documented angina or myocardial infarction, smoker, known case of bronchial asthma, chronic airway disease or pulmonary hypertension, any valvular or congenital heart disease heart disease.

A brief clinical history was taken and routine blood investigations were sent. Twelve lead electrocardiography (ECG) with right sided leads were obtained in each case. Point of care echocardiography done bedside to assess following parameters: wall motion abnormalities, left ventricular ejection fraction, RV functions like RV mid-ventricular diastolic diameter (RVDD), Tricuspid annular plane systolic excursion (TAPSE), RV Myocardial Performance Index (RVMPI), RV Fractional Area Change (RVFAC), and peak lateral tricuspid annular systolic velocity (S').

Echocardiography was done on Philips EPIQ CVxi (795232) model by the same physician for all patients to minimise inter-observer bias and results noted in data sheets, according to protocol standardised by American Society of Echocardiography. RVDD was considered abnormal with value exceeding 35 mm. Similarly abnormal cut-off values of the following were taken into consideration: TAPSE if less than 16 mm, Pulse Doppler RVMPI if more than 0.4, RVFAC if less than 0.35, S' if less than 0.1.

Coronary angiography was done for each patient as soon as possible but within 24 hours of hospitalisation as a measure of revascularisation procedure with primary percutaneous coronary intervention (PCI) or pharmaco-invasive or rescue PCI strategy. With angiography done, we tried to delineate the culprit vessel responsible for the presenting acute coronary syndrome. Angiogram showing acute vascular flow cut off or subtotal stenosis with less than TIMI 3 flow related perfusion were considered as culprit lesion. Lesions were detailed as the artery involved: whether Right coronary artery (RCA) or left circumflex artery (LCX), whether the culprit vessel is dominant or non-dominant, whether the lesion is at proximal or distal site.

Patients were managed with standard latest guidelines of acute coronary syndrome (ST elevation) as recommended by American College of Cardiology with antiplatelets and antithrombotic agents. Those patients who were given fibrinolysis were sampled for echocardiographic evaluation either before the initiation or during the thrombolytic procedure. All data were tabulated in MS Excel data sheet in tabulated fashion and subjected for statistical analysis with the help of NCSS 2020 statistical calculator.

3. Result

The mean age of the study population was 54 years. Majority of the patients were male (84%). Risk factors like hypertension were noted in 68% patients and 37% had history of diabetes mellitus. On ECG, 72 (69%) patients had a higher amplitude of ST segment elevation in lead III than lead II. On right sided ECG lead interrogation, ST elevation in RV4 was noted in 32 (30%) patients with the diagnosis of inferior wall infarction patients indicating definitive right ventricular involvement. High grade atrio-ventricular (AV) block was seen in 34 patients (32%).

ST segment elevation in RV4 lead had a significant correlation with the trend of ST segment amplitude being higher in lead III than lead II. ($Y^2 = 13.04$, $p = 0.0003$) On chi square analysis, it was also noted that those patients who had ST elevation amplitude in lead III greater than lead II and ST elevation in lead RV4 had more incidence of high-grade AV blocks ($p = 0.001$; $p = 0.039$ respectively).

Multivariate regression analysis of demographic data revealed elderly (age group above 60 years) and diabetic cohort (with history of diabetes or currently having uncontrolled hyperglycaemia) associated with significant incidence of AV block. ($p = 0.02$; $p = 0.001$ respectively). Similarly, on routine blood investigation it was found that high grade AV block were predominantly significant with patients presenting with azotaemia and increased SGPT levels ($p = 0.005$; $p = 0.016$ respectively).

Point of care bedside echocardiography parameters of various RV functions were noted.

Table 1. *RV echocardiographic systolic indices data.*

Parameters	Value	Mean ± SD	Percentage
RVIDD		33 ± 4.2 mm	
Normal	< 35 mm		70%
Abnormal	>= 35 mm		30%
TAPSE		19 ± 3.6 mm	
Normal	>= 16 mm		75%
Abnormal	< 16 mm		25%
RVMPI		0.42 ± 0.12	
Normal	< 0.43		61%
Abnormal	>= 0.43		39%
S'		0.12 ± 0.03	
Normal	>= 0.1 m/s		86%
Abnormal	< 0.1 m/s		14%
IVA		3.36 ± 1.7	
Normal	>= 3 m/s^2		50%
Abnormal	< 3 m/s^2		50%

Considering ST elevation in RV4 lead as confirmed right ventricular infarction (n = 32) among the study population of acute inferior ST elevation myocardial infarction the prevalence of RVI was calculated as 30%. Consequently, the various right sided echocardiographic parameters were analysed statistically to assess their ability to detect RVI in the study cohort.

Table 2. *Diagnostic potential of echocardiographic parameters to detect right ventricular infarction.*

Parameters	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Accuracy
RVIDD	25%	68.1%	25.8%	67.1%	54.8%
TAPSE	31.3%	79.2%	40%	72%	64.4%
RVMPI	28.1%	55.6%	22%	63.5%	47%
S’	12.5%	86%	28.6%	69%	41.3%
IVA	50%	50%	30.8%	69.2%	50%

The above table depicts that TAPSE remains to be the single echocardiographic parameter diagnosing RVI among patients with acute inferior MI with an overall accuracy of 64.4%. However, IVA has the highest sensitivity (50%) to detect RVI when compared to other parameters; and S’ showing the highest specificity (86%)

Coronary angiography of study population was analysed which revealed that the culprit lesion was in Right coronary artery (RCA) in 85 patients (82%), and left circumflex (LCx) artery was involved in the rest. Site and dominance of the culprit lesion and the coronary system was also considered. Of the 104 angiographies analysed, right dominance was noted in 85 patients (82%). Majority of the culprit lesions (55%) were located at distal segment of a dominant RCA system. Thirteen patients in the study population reported with left dominant coronary system having the dominant LCx being the culprit artery.

Table 3. *Coronary angiography characteristics of study population.*

Site of lesion		Rt. dominant	Lt. dominant	N=104
RCA	proximal	22	4	85
	Distal	57	2	
LCx	proximal	2	6	19
	Distal	4	7	
		85	19	

On analysing angiographic results obtained as in above table, it was noted that those patients diagnosed with RVI with ST elevation in RV4 had significantly higher incidence of culprit lesion in RCA than LCx, irrespective of coronary dominance and site of lesion. (Chi square test statistics = 4.47; p = 0.034) ST elevation in RV4 showed a sensitivity rate of 93.7% to detect RCA as the culprit artery in the study population. Moreover, when analysing only dominant RCA lesions, ST segment elevation in RV4 could too statistically indicate the probability of the stenosis location being in the proximal rather than distal segment. (Chi square statistics = 6.76; p = 0.009)

Incidence of high grade AVB were correlated to the culprit artery lesion. Chi square analysis showed that AVBs were significantly predominant in culprit lesions involving RCA than LCx, irrespective of coronary dominance and site of lesion. (Chi square test statistics = 5.19; p = 0.022) The presence of AVB could also statistically predict the site of lesion being in proximal rather than distal, when right dominant RCA is the culprit artery. (Chi square test statistics = 9.69; p = 0.001)

Individual RV echocardiographic indices were compared with angiographic data of the study population. Chi square analysis revealed that an abnormal RVDD value could statistically predict the culprit lesion to be in the distal RCA irrespective of the coronary dominance in comparison to other lesion sub-types. Pulse wave RVMPI values when obtained > 0.43 indicated a proximal stenosis when compared with the cohort of patients having right dominant RCA lesion. Similarly, low S’ measurement significantly correlated with the culprit lesion being found in patients having LCx artery stenosis when compared to RCA lesions with respective coronary dominance.

Table 4. *Echocardiographic parameters as angiographic correlates to predict culprit site lesion (with p values).*

Parameters	RCA V/S LCx		Prox. v/s Dist. RCA		Culprit Segment	
	Chi sq stat	P value	Chi sq stat	P value	Chi sq stat	P value
RVDD	0.85	.35	0.09	.89	11.58	.02
TAPSE	0.19	.66	0.29	.59	6.14	.104
RVMPI	0.07	.79	4.81	.02	2.70	.609
S’	6.55	.01	0.18	.66	6.17	.103
IVA	1.61	.20	0.63	.42	4.32	.364

4. Discussion

Clinical description of right ventricular myocardial infarction was first given by Saunders in 1930 when he reported a case with triad of hypotension, elevated jugular veins, and clear lung fields and extensive RV necrosis in autopsy. [10] The incidence of RVMI in inferior wall MI varies from 20% to 50% in various studies, and in less than 10% of patients RVMI is hemodynamically significant. [11] In-hospital mortality rate for IWMI with RV infarction is 31% compared to 6% in IWMI without RVMI. [12] Right ventricular infarction typically occurs when there is an occlusion of the right coronary artery proximal to the acute marginal branches or of the left circumflex artery in

patients with a dominant left coronary system. [13] Assessment of RV function remains challenging because of the complex geometry of the right ventricle, its asynchronous contraction pattern, and its mechanical interaction of the LV. [14]

An electrocardiogram (ECG) is the first diagnostic test performed in patients complaining of chest pain. If the left ventricle is involved, evidence of inferior lead ischemia/infarction in leads II, III, and AVF is generally evident. Disproportionate ST-elevation in lead III>II is almost pathognomonic for RVMI and warrants further investigation. [15] ST elevation in the V1 lead is also highly suspicious for RVMI and is even more specific when associated with ST depression in lead V2. Overall, conventional left-sided electrocardiography is a poor indicator of RV ischemia/infarction due to the position of the right side of the heart. If right side dysfunction is suspected, a right-sided ECG is the most sensitive and specific, as ST elevation in V4R >1.0 mm has 100% sensitivity, 87% specificity, and 92% predictive accuracy. [16] Conduction abnormalities such as right bundle branch block, bradycardia, or complete heart block can also manifest themselves in the ECG but may also be non-specific. [17-19]

In our study, we sampled 104 patients presented with acute onset chest pain diagnosed with inferior wall ST elevation myocardial infarction presenting within 12 hours of onset angina. All patients were subjected to right sided ECG evaluation along with standard 12 lead rhythm strip. Those having ST segment elevation of at least 1 mm were considered as confirmed cases of RVI among the study population as previous studies have showed 100% sensitivity of RV4 lead to detect RVI. We found that 30% of the patients were having RVI as comparable to previous studies done by Overgaard et al, Ashmawy et al and Klein et al. [20-22] Those diagnosed with RVI had higher incidence of high-grade AV blocks and ST segment elevation in lead III more than lead II. Other risk factors for predicting the incidence of AV block in this population were elderly patients, diabetics, and patients presenting with azotaemia and high SGPT levels. Such predictors of RVI complications were concurrently also reported by El Rabat et al and Chhapra et al. [23, 24].

Meticulous echocardiographic assessment of RV indices was performed in all patients during the acute setting to avoid sampling delay or bias. We incorporated predominantly parameters testing RV systolic function to assess the extent of RV myocardial injury during the event of acute ST elevation inferior wall infarction with or without RVI. It was observed that 30% of the patients had abnormally high RV diastolic dimension signifying RV dilatation. Similarly, 25% patients had abnormally low TAPSE values, 39% had higher RVMPI measurement than the reference value, 14% had lower tricuspid systolic peak velocity S', and 50% of the patients had decreased isovolumic acceleration of tricuspid valve motion during systole among the study population.

However, when the 5 echocardiography indices of RV systolic function were compared with the two group of patients with or without RVI, it was noted that IVA proved to have the best sensitivity and S' to have the best specificity to detect RVI among all patients of the study. Overall accuracy to detect RVI was obtained best from TAPSE measurement of the population. Sethumadhavan et al too studied echocardiographic indices of RV systolic function in their study of two groups of patients presenting with IWMI with or without RVI. It showed that patients with RVI had statistically significant lower values of S' and TAPSE compared to those with no RV involvement. [25]

We sub-classified coronary angiography report according to the culprit lesion in question into the artery involved, the site of the artery affected and the coronary dominance of the patient. Accordingly, 8 groups of culprit lesions were obtained, of which right dominant RCA distal stenosis had the maximum incidence (55%). In our study, majority of the patients (85%) had right dominant coronary anatomy, however 68% of LCx lesions causing IWMI had left dominance system. Proximal dominant RCA stenosis as the culprit lesion was proved to be statistically significant in causation of RVI in our study population. It was also seen that RCA lesions irrespective of the site or dominance were responsible for RVI on chi square statistical analysis. Furthermore, it was also appreciated that high grade AV blocks were also associated with a preponderance of RCA disease, more commonly with right dominant proximal lesions, though acute occlusion of a dominant LCx at its proximal site was also associated with blocks.

Statistical analysis further done to investigate the culprit lesion segment revealed that among the 5 indices of RV systolic function interrogated, a higher RVMPI (Tei's index) value could statistically predict lesion being in the proximal segment when RCA was the culprit vessel but increasing dimension of RVDD indicated distal RCA lesion when compared to the entire cohort with multivariate analysis. A lower peak systolic tissue doppler tricuspid valve velocity S' proved to be a surrogate marker of left dominant LCx as a culprit artery than a right dominant RCA within the study population. In previous studies done by Thirumurugan et al, TAPSE and RVMPI TDI were the echocardiographic indices of RV systolic function as laid down by ASE which predicted proximal RCA lesions in RVI. [26] Mezzulin et al demonstrated that if the lower limit value of S' is considered less than 11.5 cm/s then there is a significant correlation to detect RVI among IWMI cohorts with RVEF < 45%. [27]

5. Conclusion

IWMI with RVI is associated with a poor prognosis especially when elderly and diabetic patients present with renal and hepatic dysfunction and frequently is associated with high grade AV blocks. RV systolic echocardiographic indices like IVA and TAPSE has shown modest accuracy and adequate sensitivity in detecting RVI among patients of IWMI. ST elevation in RV4 proves to be a surrogate marker of predicting proximal dominant RCA lesion, moreover when complicated with conduction blocks. Echocardiographic parameters too were found to predict the culprit lesion. Lower S' values suggested more of proximal dominant LCx

rather than RCA lesion; on the other hand, a high RVMPI value was found to be indicative of a proximal stenosis within RCA culprit vessel cohort, and greater RVDD value predicted for dominant distal RCA lesion on multivariate regression analysis of the entire population of IWMI with or without RVI.

References

[1] Warner MJ, Tivakaran VS. Inferior Myocardial Infarction. [Updated 2021 Dec 28]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan.

[2] Vanajakshamma V, Vyshnavi K, Latheef K et al. Right Ventricular Infarction in Inferior Wall Myocardial Infarction. Indian J Cardiovasc Dis Women-WINCARS 2017; 2: 29–34.

[3] Md. Sohel Khan et al (2021). Clinical Profile of Right Ventricular Infarction in Patients with Acute Inferior Wall Myocardial Infarction. Sch Int J Tradit Complement Med, 4 (10): 174-178.

[4] Ondrus T, Kanovsky J, Novotny T, Andrsova I, Spinar J, Kala P. Right ventricular myocardial infarction: From pathophysiology to prognosis. Exp Clin Cardiol. 2013; 18 (1): 27-30.

[5] Nagam MR, Vinson DR, Levis JT. ECG Diagnosis: Right Ventricular Myocardial Infarction. Perm J. 2017; 21: 16-105. doi: 10.7812/TPP/16-105.

[6] Kukla P, Dudek D, Rakowski T, Dziewierz A, Mielecki W, Szczuka K, Dubiel JS. Inferior wall myocardial infarction with or without right ventricular involvement--treatment and in-hospital course. Kardiol Pol. 2006 Jun; 64 (6): 583-8; discussion 589-90.

[7] Cohn JN, Guiha NH, Broder MI, Limas CJ. Right ventricular infarction. Clinical and hemodynamic features. Am J Cardiol. 1974 Feb; 33 (2): 209-14.

[8] Goldstein JA. Pathophysiology and management of right heart ischemia. J Am Coll Cardiol. 2002 Sep 4; 40 (5): 841-53.

[9] Konstam MA, Kiernan MS, Bernstein D, Bozkurt B, Jacob M, Kapur NK, Kociol RD, Lewis EF, Mehra MR, Pagani FD, Raval AN, Ward C., American Heart Association Council on Clinical Cardiology; Council on Cardiovascular Disease in the Young; and Council on Cardiovascular Surgery and Anesthesia. Evaluation and Management of Right-Sided Heart Failure: A Scientific Statement from the American Heart Association. Circulation. 2018 May 15; 137 (20): e578-e622.

[10] Rajesh GN, Raju D, Nandan D, et al. Echocardiographic assessment of right ventricular function in inferior wall myocardial infarction and angiographic correlation to proximal right coronary artery stenosis. Indian Heart J. 2013; 65 (5): 522-528.

[11] Robert AO, Louis J Dell'italia. Diagnosis and management of right ventricular myocardial infarction. Current Problems in Cardiology, 2004; 29 (1): 6-47.

[12] Kumar V, Sinha S, Kumar P, et al. Short-term outcome of acute inferior wall myocardial infarction with emphasis on conduction blocks: a prospective observational study in Indian population. Anatol J Cardiol. 2017; 17 (3): 229-234.

[13] Maha H. El Sebaie, Osama El Khatib. Right ventricular echocardiographic parameters for prediction of proximal right coronary artery lesion in patients with inferior wall myocardial infarction. Journal of the Saudi Heart Association, 2016; 28 (2): 73-80.

[14] Vignon P, Weinert L, Mor-Avi V et al. Quantitative Assessment of Regional Right Ventricular Function with Color Kinesis. Am J Respir Crit Care Med 1999; 159: 1949–1959.

[15] Jeffers JL, Boyd KL, Parks LJ. Right Ventricular Myocardial Infarction. [Updated 2021 Aug 3]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan.

[16] Albulushi A, Giannopoulos A, Kafkas N, et al. Acute right ventricular myocardial infarction, Expert Review of Cardiovascular Therapy, 2018; 16 (7): 455-464.

[17] Albaghdadi A, Teleb M, Porres-Aguilar M, Porres-Munoz M, Marmol-Velez A. The dilemma of refractory hypoxemia after inferior wall myocardial infarction. Proc (Bayl Univ Med Cent). 2018 Jan; 31 (1): 67-69.

[18] Ashida T, Tani S, Nagao K, Yagi T, Matsumoto N, Hirayama A. Usefulness of synthesized 18-lead electrocardiography in the diagnosis of ST-elevation myocardial infarction: A pilot study. Am J Emerg Med. 2017 Mar; 35 (3): 448-457.

[19] Venkatachalam S, Wu G, Ahmad M. Echocardiographic assessment of the right ventricle in the current era: Application in clinical practice. Echocardiography. 2017 Dec; 34 (12): 1930-1947.

[20] Overgaard C, Fitchett D. Cardiogenic shock from right ventricular infarction. Cardiol Rounds. 2002; 7 (8).

[21] Ashmawy MM, EI-Barbary YH, Hassib WA, Naguib CG. Assessment of Right Ventricular Function in Acute Inferior Wall Myocardial Infarction in Patients Treated with Primary Percutaneous Coronary Intervention. Research Journal of Cardiology, 2016; 9: 8-16.

[22] Klein HO, Tordjman, Ninio R, et al. The early recognition of right ventricular infarction diagnostic accuracy of electrocardiographic lead V4R. Circulation. 1983; 67: 558.

[23] Khaled El-Rabat E, Bastwesy RB, El Meligy NA et al. Predictors of complications among patients with acute inferior and right myocardial infarction. Research in Cardiovascular Medicine. 2019; 8 (4): 99-105.

[24] Chhapra DA, Mahajan SK, Thorat ST. A study of the clinical profile of right ventricular infarction in context to inferior wall myocardial infarction in a tertiary care centre. J Cardiovasc Dis Res. 2013; 4 (3): 170-176.

[25] Sethumadhavan R, Ponnusamy S. Echocardiographic assessment of right ventricular myocardial infarction. J. Evolution Med. Dent. Sci. 2018; 7 (47): 5063-5068.

[26] Thirumurugan P, Rafic Babu AM. Assessment of Right Ventricular function by Echocardiograpy in Inferior wall Myocardial Infarction and Angiographic correlation to Proximal Right Coronary Artery Stenosis. IOSR Journal of Dental and Medical Sciences. 2017 (Sept); 16 (9): 36-40.

[27] Meluzı´n J, Spinarova´ L, Bakala J, et al. Pulsed Doppler tissue imaging of the velocity of tricuspid annular systolic motion; a new, rapid, and non-invasive method of evaluating right ventricular systolic function. Eur Heart J. 2001; 22: 340-348.