ASSOCIATION OF RIGHT VENTRICULAR INFARCTION WITH INFERIOR WALL MYOCARDIAL INFARCTION

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ABSTRACT

Background: The right ventricular infarction occurs frequently in association with inferior myocardial infarction and occurs separately in rare patterns. It is related to higher morbidity and mortality rates.

Aim of study: To measure the prevalence of right ventricular infarction among patients with inferior wall myocardial infarction and identifying the characteristics related to right ventricular infarction.

Patients and methods: A cross sectional study carried out in Coronary Care Unit of Azadi Teaching hospital in Kirkuk through the period from 1st of January to31st of October, 2017 on convenient sample of 150 inferior wall myocardial infarction patients. The data were collected by publisher through direct interview and filling of a prepared questionnaire.

Results: Right ventricular infarction was present among 39.3% of patients with inferior wall myocardial infarction. There was a highly significant association between older age of self-employed patients with inferior wall myocardial infarction and right ventricular infarction ($p<0.001$). The main significant risk factors for right ventricular infarction among inferior wall myocardial infarction patients were family history of heart diseases and obesity ($p<0.001$, $p=0.01$). Inferior wall myocardial infarction patients with right ventricular infarction were significantly had higher complicated outcome than myocardial infarction patients with absence of right ventricular infarction ($p=0.002$).

Conclusions: The prevalence of right ventricular infarction in inferior myocardial infarction is within normal range of previous literatures.

INTRODUCTION

1.1. Coronary artery diseases

Cardiovascular disease (CVD) is one of the most common causes of mortality and morbidity across the globe. Coronary artery disease (CAD) is a major cause of death in Western countries, and it is becoming a major cause of death in developing countries. This increase may be due to the rising prevalence of many CAD risk factors, such as obesity and diabetes, which are the most important risk factors [1].

Coronary artery disease "CAD" is predominately manifest in older individuals, but the disease process begins in the young. Although the prevalence of coronary atherosclerosis in young adults is difficult to estimate, there are data on the risk of developing CAD at an older age based on risk factors present while young. Prior literature emphasizes that cigarette smoking, cocaine use, diabetes and dyslipidemia are prominent risk factors in the development of early atherosclerosis [2].

Atherosclerosis, the underlying cause of most heart attacks and strokes, is characterized by the development of arterial plaque. The development of atherosclerosis involves several complex processes, including the infiltration and oxidation of low-density lipoprotein (LDL) within the arterial wall. Oxidized LDL (ox-LDL) has been shown to be a major contributor to the dangerous accumulation of lipids beneath the vascular endothelium. Within the intima the ox-LDL particles can be engulfed by macrophages, which in turn comprise the core of arterial plaque [3].

The term acute coronary syndrome (ACS) refers to any group of clinical symptoms compatible with acute myocardial ischemia and covers the spectrum of clinical conditions ranging from unstable angina (UA) to non–ST-segment elevation myocardial infarction (NSTEMI) to ST-segment elevation myocardial infarction (STEMI). Unstable angina and NSTEMI are closely related conditions: their pathophysiologic origins and clinical presentations are similar, but they differ in severity. A diagnosis of NSTEMI can be made when the ischemia is sufficiently severe to cause myocardial damage that result in the release of a biomarker of myocardial necrosis into the circulation (cardiac-specific troponins T or I, or muscle and brain fraction of creatine kinase [CK-MB]) [3, 4].

In contrast, the patient is considered to have experienced UA if no such biomarker can be detected in the bloodstream hours after the initial onset of ischemic chest pain. Unstable angina exhibits 1 or more of 3 principal presentations: (1) rest angina (usually lasting >20 minutes), (2) new-onset (<2 months previously) severe angina, and (3) a crescendo pattern of occurrence (increasing in intensity, duration, frequency, or any combination of these factors). Each year in the United States, approximately 1.36 million hospitalizations are required for ACS (listed either as a primary or a secondary discharge diagnosis), of which 0.81 million are for myocardial infarction (MI) and the remainder are for UA. Roughly two-thirds of patients with MI have NSTEMI; the rest have STEMI [4].

1.2. Myocardial infarction

Myocardial infarction (MI) can be recognized by clinical features, electrocardiographic (ECG) findings , elevated values of biochemical

markers (biomarkers) of myocardial necrosis, and by imaging, or may be defined by pathology. It is a major cause of death and disability worldwide. MI may be the first manifestation of coronary artery disease (CAD) or it may occur, repeatedly, in patients with established disease. Information on MI rates can provide useful information regarding the burden of CAD within and across populations, especially if standardized data are collected in a manner that distinguishes between incident and recurrent events. From the epidemiological point of view, the incidence of MI in a population can be used as a proxy for the prevalence of CAD in that population. The term 'myocardial infarction' may have major psychological and legal implications for the individual and society. It is an indicator of one of the leading health problems in the world and it is an outcome measure in clinical trials, observational studies and quality assurance programs. These studies and programs require a precise and consistent definition of MI [5].

In 2000, the First Global MI Task Force presented a new definition of MI, which implied that any necrosis in the setting of myocardial ischemia should be labeled as MI [6]. These principles were further refined by the Second Global MI Task Force, leading to the Universal Definition of Myocardial Infarction Consensus Document in 2007, which emphasized the different conditions which might lead to an MI [7]. This document, endorsed by the European Society of Cardiology, the American College of Cardiology Foundation, the American Heart Association, and the World Heart Federation, has been well accepted by the medical community and adopted by the World Health Organization [8]. However, the development of even more sensitive assays for markers of myocardial necrosis mandates further revision, particularly when such necrosis occurs in the setting of the critically ill, after percutaneous coronary

procedures or after cardiac surgery. The Third Global MI Task Force has continued the Joint efforts by integrating these insights and new data into the current document, which now recognizes that very small amounts of myocardial injury or necrosis can be detected by biochemical markers and/or imaging [5].

1.1.1. Pathological characteristics of myocardial ischemia and infarction

MI is defined in pathology as myocardial cell death due to prolonged ischemia. After the onset of myocardial ischemia, histological cell death is not immediate, but takes a finite period of time to develop—as little as 20 min, or less in some animal models [9]. It takes several hours before myocardial necrosis can be identified by macroscopic or microscopic post-mortem examination. Complete necrosis of myocardial cells at risk requires at least 2-4 h, or longer, depending on the presence of collateral circulation to the ischemic zone, persistent or intermittent coronary arterial occlusion, the sensitivity of the myocytes to ischemia, pre-conditioning, and individual demand for oxygen and nutrients [7]. The entire process leading to a healed infarction usually takes at least 5-6 weeks. Reperfusion may alter the macroscopic and microscopic appearance [5].

1.1.2. Biomarker detection of myocardial injury with necrosis

Myocardial injury is detected when blood levels of sensitive and specific biomarkers such as cTn or the MB fraction of creatine kinase (CKMB) are increased [7]. Cardiac troponin I and T are components of the contractile apparatus of myocardial cells and are expressed almost exclusively in the heart. Although elevations of these biomarkers in the blood reflect injury leading to necrosis of myocardial cells, they do not

indicate the underlying mechanism [10]. Various possibilities have been suggested for release of structural proteins from the myocardium, including normal turnover of myocardial cells, apoptosis, cellular release

of troponin degradation products, increased cellular wall permeability, formation and release of membranous blebs, and myocyte necrosis [11]. Regardless of the pathobiology, myocardial necrosis due to myocardial ischemia is designated as MI [5].

1.1.3. Clinical features of myocardial ischemia and infarction

Onset of myocardial ischemia is the initial step in the development of MI and results from an imbalance between oxygen supply and demand. Myocardial ischemia in a clinical setting can usually be identified from the patient's history and from the ECG. Possible ischemic symptoms include various combinations of chest, upper extremity, mandibular or epigastric discomfort (with exertion or at rest) or an ischemic equivalent such as dyspnea or fatigue [8]. The discomfort associated with acute MI usually lasts 20 min. Often, the discomfort is diffuse—not localized, nor positional, nor affected by movement of the region—and it may be accompanied by diaphoresis, nausea or syncope. However, these symptoms are not specific for myocardial ischemia. Accordingly, they may be misdiagnosed and attributed to gastrointestinal, neurological, pulmonary or musculoskeletal disorders. MI may occur with atypical symptoms—such as palpitations or cardiac arrest—or even without symptoms; for example in women, the elderly, diabetics, or post-operative and critically ill patients [7]. Careful evaluation of these patients is advised, especially when there is a rising and/or falling pattern of cardiac biomarkers [8].

1.1.4. Clinical classification of myocardial infarction

For the sake of immediate treatment strategies, such as reperfusion therapy, it is usual practice to designate MI in patients with chest discomfort, or other ischemic symptoms that develop ST elevation in two

contiguous, as an 'ST elevation MI' (STEMI). In contrast, patients without ST elevation at presentation are usually designated as having a 'non-ST elevation MI' (NSTEMI). Many patients with MI develop Q waves (Q wave MI), but others do not (non-Q MI). Patients without elevated biomarker values can be diagnosed as having unstable angina.

1.1.5. Myocardial distribution of three main coronary arteries

A. 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 [12].

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 [12].In these patients, the left ventricular apical

portion is supplied by the posterior descending branch of the RCA or LCx, which is larger and longer than usual [9].

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 branches of LCx are obtuse marginals,which vary from one to three in number and supply the lateral free wall of the left ventricle [13].

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 [12].

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 [13].

1.2. Anterior wall myocardial infarction

An anterior wall myocardial infarction — also known as anterior wall MI, or AWMI, or anterior ST segment elevation MI, or anterior STEMI — occurs when anterior myocardial tissue usually supplied by the left anterior descending coronary artery suffers injury due to lack of blood supply. When an AWMI extends to the septal and lateral regions as well, the culprit lesion is usually more proximal in the LAD or even in the left main coronary artery. This large anterior myocardial infarction is termed an extensive anterior [14]. The ECG findings of an acute anterior myocardial infarction wall include [15]:

1. ST segment elevation in the anterior leads (V3 and V4) at the J point and sometimes in the septal or lateral leads, depending on the extent of the MI. This ST segment elevation is concave downward and frequently overwhelms the T wave. This is called "tombstoning" for obvious reasons; the shape is similar to that of a tombstone.
2. Reciprocal ST Segment depression in the inferior leads (II, III and aVF). According to the American College of Cardiology/American Heart Association guidelines for STEMI, there must be "new ST segment elevation at the J point in at least two contiguous leads of ≥ 2 mm (0.2 mV) in men or 1.5 mm (0.15 mV) in women in leads V2-V3 and/or of ≥ 1 mm (0.1 mV) in other contiguous chest leads or the limb leads." This means 1 millimeter in any two contiguous leads, except leads V2 or V3, where the elevation must be 2 mm in men or 1.5 mm in women [15].

1.3. Posterior wall myocardial infarction

Posterior infarction accompanies 15-20% of STEMIs, usually occurring in the context of an inferior or lateral infarction. Isolated posterior MI is less common (3-11% of infarcts). Posterior extension of an inferior or lateral infarct implies a much larger area of myocardial damage, with an increased risk of left ventricular dysfunction and death. Isolated posterior infarction is an indication for emergent coronary reperfusion. However, the lack of obvious ST elevation in this condition means that the diagnosis is often missed. Posterior MI is suggested by the following changes in V1-3 [12]:

- Horizontal ST depression.
- Tall, broad R waves (>30ms).
- Upright T waves.
- Dominant R wave (R/S ratio > 1) in V2.

1.4. Lateral wall myocardial infarction

The lateral wall of the LV is supplied by branches of the left anterior descending (LAD) and left circumflex (LCx) arteries. Infarction of the lateral wall usually occurs as part of a larger territory infarction, e.g. anterolateral STEMI. Isolated lateral MIs are less common, but may be produced by occlusion of smaller branch arteries that supply the lateral wall, e.g. the first diagonal branch (D1) of the LAD, the obtuse marginal branch (OM) of the LCx, or the ramus intermedius. Lateral MI is a stand-alone indication for emergent reperfusion. Lateral extension of an anterior, inferior or posterior MI indicates a larger territory of myocardium at risk with consequent worse prognosis. Recognition of lateral MI [12]:

- ST elevation in the lateral leads (I, aVL, V5-6).
- Reciprocal ST Depression in the inferior leads (III and aVF).
- ST elevation primarily localised to leads I and aVL is referred to as a high lateral MI.

1.5. Inferior 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 > lead II and more than 1 mm ST-segment depression in leads I and aVL [16]. 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 [17].

One of the important clues that suggest 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 [12]. 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 [18]. Associated ST segment depression in leads V 1 and V2 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 [12].

It has been observed that various ECG and angiographic characteristics did have significant impact on sensitivity of ECG criteria for localizing IWMI [19]. 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 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) [19].

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 [20].

1.5.1. Right ventricular myocardial infarction

Right ventricular infarction (RVI) rarely occurs in isolation with approximately between one-third and half of the patients with inferior-wall myocardial infarction (IWMI) having some RV involvement. The reported incidence of RV infarction varies widely, depending on the criteria and methodology of the study in question. RV infarction can be defined pathologically, haemodynamically, echocardiographically, electrocardiographically, or by cardiac magnetic resonance (CMR). In this manuscript, we first discuss the incidence and diagnosis of RV infarction, with focus on the non-invasive modalities, such as CMR. Then, we aim to provide an overview of its prognosis with respect to changes between the fibrinolytic and mechanical reperfusion era. Finally, we discuss the management of RV infarction focusing on biventricular interdependence, which is expected to promote the understanding of optimal volume replacement, early revascularization, and utilization of newly developing treatment modalities [21].

A. Incidence

RVMI is usually associated with inferior wall MI and, in practice, does not exist in isolation. The occurrence of RV impairment depends primarily on the location of the MI, which ranges from rare cases in the anterior heart wall to more common locations (depending on the type of diagnostic method used) such as in the inferior wall in 24% to 50% of cases. The relatively small percentage of RVMIs may be explained by several factors: lower oxygen requirements of the RV due to its smaller muscle mass and workload; increased blood flow during diastole and systole; more extensive collateralization of the RV, primarily from the left coronary system; and diffusion of oxygen from intra chamber blood through the thin wall of the RV and into the Thebesian veins [22].

The clinical sequelae of RVMI vary widely, and range from no hemodynamic compromise to severe hypotension and cardiogenic shock depending on the extent of RV ischemia. According to the literature, approximately 25% to 50% of RV infarctions are hemodynamically significant [23].

B. Diagnosis

Physical examination

It is important to consider a diagnosis of RVMI, particularly in the presence of an inferior wall MI. The typical triad observed on physical examination is hypotension occurring with jugular vein distention and clear lungs. Preserved left ventricular (LV) function confirms the diagnosis. A tricuspid regurgitation murmur, Kussmaul's sign (an increase in inspiratory central venous pressure, visible as jugular vein distention) and pulsus paradoxus are signs of significant hemodynamic effects due to RV ischemia. In some cases, these symptoms are not present at admission and do not occur until diuretics or nitrates are administered [24].

Electrocardiography

Because RVMIs are usually associated with an inferior wall MI, evaluation using standard 12-lead electrocardiography (ECG) often reveals corresponding ST segment elevations in leads II, III and aVF. Disproportionate ST segment elevation with greater ST elevation in lead III than in lead II is pathognomonic for an RVMI, and RV involvement should be fully and carefully considered [27]. Because standard 12-lead ECG images mainly assess the LV, right-sided precordial leads should always be used. These can show ST segment elevation across the entire right precordium from V1R through V6R; a sole ST segment elevation in lead V4R >1.0 mm is a reliable marker of an RV infarction, with 100% sensitivity, 87% specificity and 92% predictive accuracy [28]. Furthermore,

higher ST segment elevations in V4R have been found to be independent predictive factors for more significant RV dysfunction and higher mortality rates [29].

Two-dimensional echocardiography

This technique can show RV dilation with depressed systolic function and RV free wall dyskinesia with paradoxical septal motion. A Doppler examination of specific pulmonary regurgitation patterns can add hemodynamic insight and confirm the diagnosis of RV involvement, especially in cases of technically inadequate two dimensional images [25].

Radionuclide ventriculography and 99mtc pyrophosphate myocardial scintigraphy

These noninvasive techniques may be used to detect RV dysfunction. A dilated right ventricle with hypokinesia, akinesia or dyskinesia of its free wall associated with a depressed RV ejection fraction and a normal or only mildly depressed LV EF are indicative of an RVMI. A 99mTc pyrophosphate myocardial scintigraphy examination requires proper timing, and the scans are usually not diagnostic until 72 h after the onset of symptoms. Excessive uptake of the radionuclide in non cardiac structures (chest wall, bone, cartilage) can lead to issues regarding the interpretation of the acquired images [26].

Hemodynamic examination

Hemodynamic examination using right-sided cardiac catheterization may reveal a disproportionate elevation of right-sided filling pressures compared with left-sided filling pressures. The generally accepted criteria for hemodynamically-significant RVMIs originate from an autopsy/hemodynamic study by Lopez-Sendon et al and include right

atrial pressure (RAP) >10 mmHg, a RAP to pulmonary capillary wedge pressure (PCWP) ratio >0.8, or RAP within 5 mmHg of the PCWP. However, with concomitant and significant LV dysfunction, the close relationship between the RAP and the PCWP is not preserved, although the RAP will continue to be elevated [22].

Coronary angiography

Angiography often reveals occlusion of the right coronary artery (RCA) proximal to the acute marginal branch , while more proximal occlusions usually suggest more extensive necrosis of the posterior and, potentially, the anterior RV myocardial wall. In patients with left coronary artery dominance, a left circumflex coronary artery (LCX) occlusion may also be found. Although uncommon, RV involvement may be present in patients with an occlusion in the left anterior descending artery. Cabin et al studied 97 hearts with anterior MIs and found that 13% were RVMIs [22].

C. Complications

RVMIs are more often complicated by all types of arrhythmias compared with 'simple' inferior or anterior wall LVMIs [30]. Barrillon et al were the first to recognize the significantly higher risk of severe conduction disorders in patients with RV involvement [22]. Complete atrio-ventricular (AV) or sinoatrial blocks occurred in one half of cases in which ST segment elevation or a QS pattern in V3R and/or V4R were present. On the other hand, these complications were found in only 14% of cases in which these signs were absent. In a prospective study of 200 consecutive patients with acute inferior wall MI, Zehender et al demonstrated a higher incidence of sustained ventricular tachycardia (16% versus 8%; P=0.08) and ventricular fibrillation (21% versus 9%; P=0.05) in patients with

ECG signs of RV involvement. Significantly higher incidences of complete AV block (17% versus 4%; P=0.06) and severe bradycardias (9% versus 3%; P=0.09) with pacing requirements (18% versus 3%; P=0.01) have been reported in cases of RVMI [22]. Mehta et al [30] studied complications and prognoses in a large number of patients hospitalized for anterior (n=971) and inferior MI with (n=491) or without (n=638) RV involvement. Anterior wall MI was associated with the highest overall and in-hospital mortality rates, while the number of arrhythmias (ventricular fibrillation, sustained ventricular tachycardia and high-degree AV blockade) was the highest in patients with inferior wall MI with RV involvement [30].

D. Therapy

RV ischemia may lead to systolic and diastolic dysfunction, resulting in a serious deficit in LV preload with a subsequent drop in cardiac output and consequent systemic hypotension. Adequate filling (preload) of the impaired RV is thus crucial to maintain sufficient RV output volume and LV function [31]. Initial therapy, therefore, requires the administration of sufficient volume to increase RV filling; at the same time, it is critically important to avoid drugs that cause venodilation and a decrease in RV filling (eg, nitrates, diuretics). Treatment is generally recommended to begin with a volume challenge of 300 mL to 600 mL normal saline over 10 min to 15 min through a central line or through a large-bore peripheral intravenous site [32]. However, some studies have indicated that volume loading may not increase cardiac output [33]. This may be due to variable initial volume status among patients. Some patients may be relatively volume-depleted and could benefit from a volume infusion, while others who present with a normal intravascular volume show no changes in cardiac index or blood pressure following a fluid load because the RV

preload is already at a maximum for maintaining RV stroke output [26]. Invasive hemodynamic monitoring is, therefore, recommended, because further infusion may be harmful if additional increases in RV volume prevent sufficient LV filling via interventricular interactions and intrapericardial pressure equalization. Based on hemodynamic monitoring studies, exceeding a RAP or PCWP of 20 mmHg is generally not recommended [22].

Restoration of sufficient coronary blood flow represents the only treatment that addresses the underlying problem, and early reperfusion improves RV performance as well as the clinical course and survival [34]. Bowers et al studied clinical outcomes and RV function using two-dimensional echocardiography in 53 patients with acute RVMI before and after reperfusion therapy. The authors reported dramatic recovery of RV performance and excellent clinical outcomes after early and complete reperfusion of the RCA using primary percutaneous coronary intervention. In contrast, unsuccessful reperfusion was associated with impaired recovery of RV function, persistent hemodynamic compromise and high mortality rates. Early reperfusion was also crucial in preventing ventricular arrhythmias, which were observed much more frequently in patients with unsuccessful coronary reperfusion [35]. In patients with refractory hypotension and low cardiac output, intra-aortic balloon counterpulsation may be beneficial. Although IABC does not directly influence RV performance, it can increase coronary perfusion pressure and thereby improve RV function, particularly if the RCA has been recanalized. Furthermore, the performance of a dysfunctional RV is largely dependent on LV septal contraction, which can be improved using IABC [34].

E. Prognosis

RV involvement significantly increased mortality in patients with inferior wall MI , although it does not achieve the same rates observed in anterior wall MI [30]. Zehender et al studied 200 consecutive patients admitted with acute inferior wall MI . RV involvement presenting with an ST elevation in lead V4R was found to be a highly negative predictive factor of both in-hospital mortality (31% versus 6%) and all major in-hospital complications (64% versus 28%) [22]. These included sustained ventricular tachycardia, ventricular fibrillation, myocardial rupture, second- and third-degree AV block requiring cardiac pacing, re-infarction and cardiogenic shock. Jacobs et al [36] evaluated 933 patients with acute MI presenting with cardiogenic shock due to either predominant RV (n=49) or LV failure (n=884). Despite the younger age, shorter time to shock diagnosis, higher prevalence of single-vessel coronary disease, lower prevalence of previous MI and similar revascularization outcomes in patients with RV failure, there was no significant difference in mortality between the groups (53.1% versus 60.8%; P=0.296), representing an unexpectedly high mortality rate among the RV shock patients. On the other hand, recently published research by Foussas et al[37] found no significant difference in long-term mortality between patients with and without a RVMI. Therefore, the poor in-hospital prognosis for patients with RVMIs appears to be mainly due to the increased risk of life-threatening arrhythmias [30]. Proper monitoring and appropriate antiarrhythmic treatment until hospital discharge plays a key role in the overall prognosis and survival of patients [22].

Aim of study

To measure the prevalence of right ventricular infarction among patients with inferior wall myocardial infarction and identifying the characteristics related to right ventricular infarction.

PATIENTS&METHODS

Study design & settings

A cross sectional study carried out in Coronary Care Unit (CCU) of Azadi Teaching hospital in Kirkuk through the period from 1st of January to31st of October, 2017.

Population of the study

All patients with inferior wall myocardial infarction (MI) presented to CCU of Azadi Teaching hospital in Kirkuk were included in the study population.

Inclusion criteria

The main inclusion is Inferior MI.

Exclusion criteria

1. Anterior wall MI.
2. Double wall infarction.

Sampling

A convenient sample of 150 inferior wall MI patients presented to CCU of Azadi Teaching hospital was selected after their approval to participate in the study.

Data collection

The data were collected by publisher through direct interview and filling of a prepared questionnaire. The questionnaire form was designed by publisher which include the following**:**

1. Socio-demographic characteristics: Age, gender and occupation.
2. Clinical risk factors of MI: Smoking, HT, DM, old IHD, family history of heart diseases and obesity.
3. Examination findings of inferior wall MI patients: General examination, baseline PR, BP and JVP.
4. Outcome of inferior wall MI patients.
5. Number of patients diagnosed with right ventricular infarction (RVI).

Inferior wall MI was diagnosed by a Specialist in Internal Medicine by ECG and cardiac markers. After taking full history and examination of inferior wall MI patients in CCU, ECG was done by ECG equipment (GE mac 1600) and echocardiography was also done by physician of certified in echocardiography by the equipment (Echo Philips-c x50).The diagnosis of RVI was done by a Specialist in Internal Medicine depending on clinical history and examination, ECG.

Ethical considerations:

- Approval was taken from Azadi Teaching hospital administration.
- Oral consent was taken from the patients.
- The publisher and supervisor treat the patients accordingly.

Statistical analysis

All patients' data entered using computerized statistical software; Statistical Package for Social Sciences (SPSS) version 22 was used. Descriptive statistics presented as (mean ± standard deviation) and frequencies as percentages. Kolmogorov Smirnov analysis verified the normality of the data set. Chi square test was used for comparison between categorical data (Fishers exact test was used when expected variable were less than 2% of total number of variables). Independent sample t-test was used to compare between two means. The level of significance set as ≤ 0.05.

RESULTS

A total of 150 patients admitted to Coronary Care Unit (CCU) with inferior wall myocardial infarction (MI) were included in this study with mean age of 60.4±7.8 years;6.7% of them were in age group 40-49 years, 24% of them in age group 50-59 years, 56% of them in age group 60-69 years and 13.3% of them were in age group ≥70 years. Males were more than females with male to female ratio as 3.5:1. The occupation of studied MI patients was distributed as followings; 22% housewives, 5.3% public servants, 37.4% self employed, and 35.3% retired. All these findings were shown in table 1 and figures 1, 2.

Table 1: Socio-demographic characteristics of inferior wall MI patients.

Variable	**No.**	**%**
Age mean±SD (60.4±7.8 years)		
40-49 years	10	6.7
50-59 years	36	24.0
60-69 years	84	56.0
≥70 years	20	13.3
Total	150	100.0
Gender		
Male	117	78.0
Female	33	22.0
Total	150	100.0
Occupation		
Housewife	33	22.0
Public servant	8	5.3
Self employed	56	37.4
Retired	53	35.3
Total	150	100.0

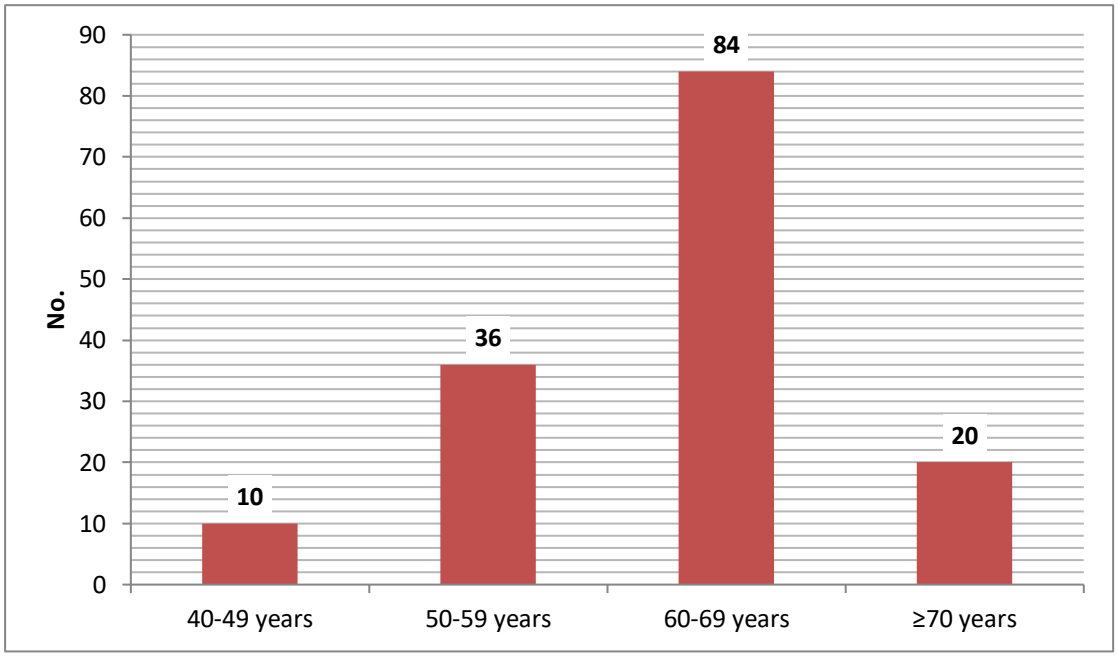

Figure 1: Age distribution.

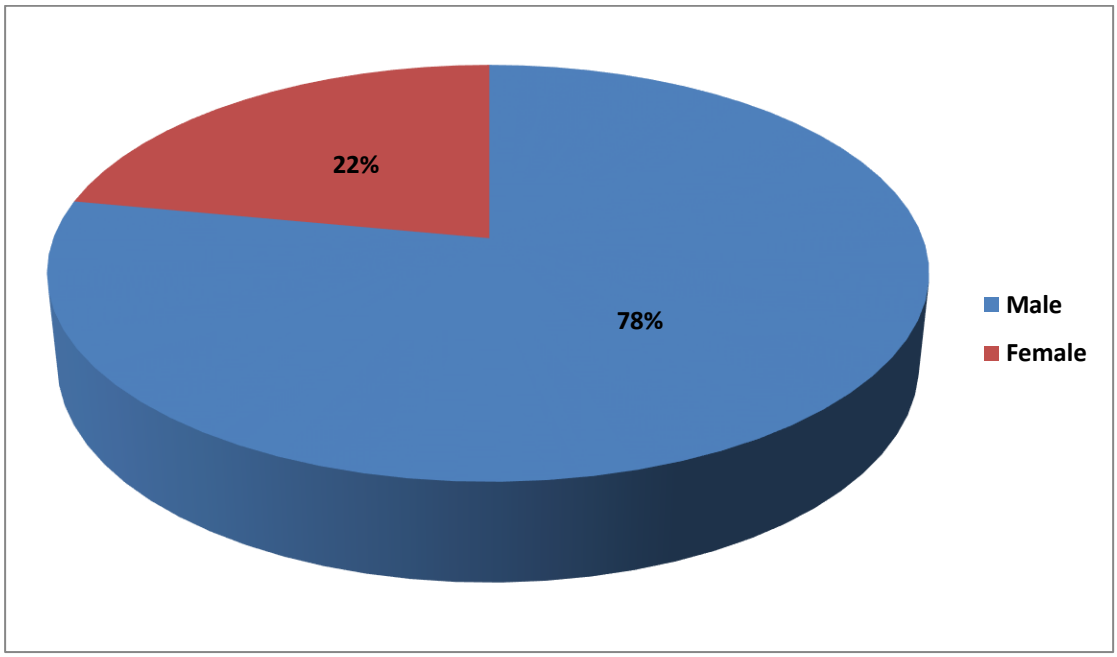

Figure 2: Gender distribution.

High proportion (79.3%) of inferior MI patients were smokers, 36.7% had HT history, 55.3% had DM history, only one patient had old IHD, 8% had positive family history of heart diseases and 2.7% of them had obesity history. All these findings were shown in table 2 and figure 3.

Table 2: Clinical risk factors of inferior wall MI patients.

Variable	**No.**	**%**
Smoking		
Yes	119	79.3
No	31	20.7
Total	150	100.0
HT		
Yes	55	36.7
No	95	63.3
Total	150	100.0
DM		
Yes	83	55.3
No	67	44.7
Total	150	100.0
Old IHD		
Yes	1	0.7
No	149	99.3
Total	150	100.0
Family history of heart diseases		
Positive	12	8.0
Negative	138	92.0
Total	150	100.0
Obesity		
Yes	4	2.7
No	146	97.3
Total	150	100.0

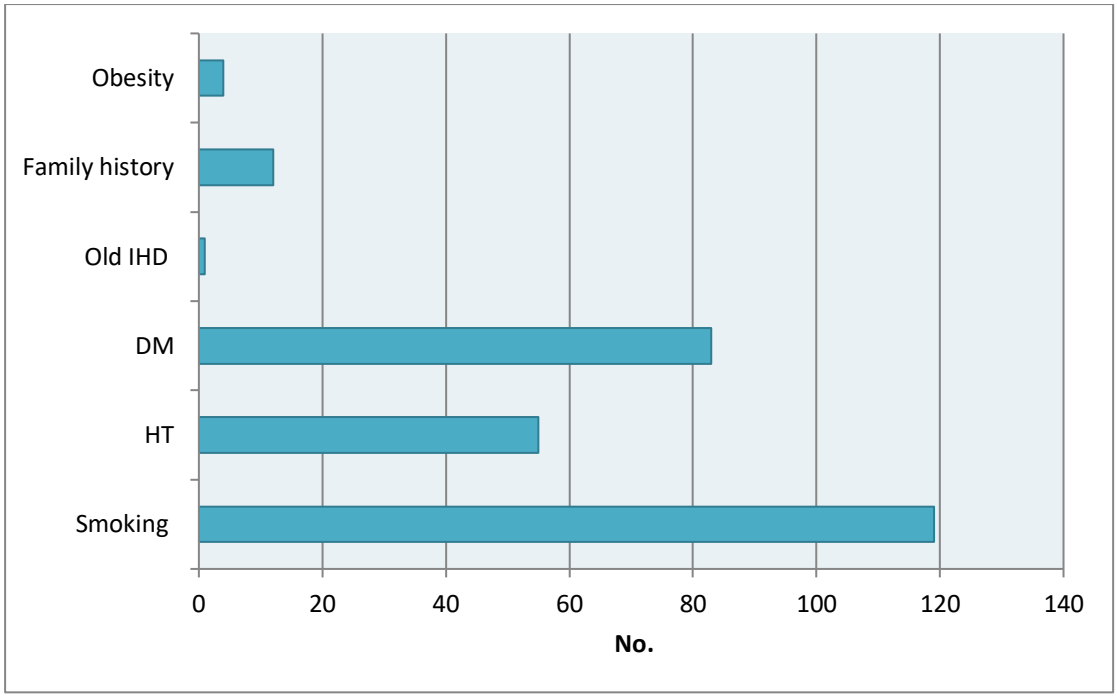

Figure 3: Risk factors of inferior wall MI patients.

All inferior wall MI patients were conscious. Mean baseline PR of inferior wall MI patients was 76.9±15.5 b/m, 8.7% of them had slow PR and 12% of them had rapid PR. Mean blood pressure of inferior wall MI patients was 128/78.4±22.3/15.5 mmHg, 18% of them had low BP (below 100\50 mmgh) and 2.7% of them had high BP (above 140\90 mmgh). The JVP was increased among 12% of inferior wall MI patients. All these findings were shown in table 3 and figure4.

Table 3: Examination results of inferior wall MI patients.

Variable	No.	%
General examination		
Conscious	150	100.0
Total	150	100.0
Baseline PR mean±SD (76.9±15.5 b/m)		
Slow	13	8.7
Normal	119	79.3
Rapid	18	12.0
Total	150	100.0
BP mean±SD (128/78.4±22.3/15.5 mmHg)		
Low	27	18.0
Normal	119	79.3
High	4	2.7
Total	150	100.0
JVP		
Normal	132	88.0
Increased	18	12.0
Total	150	100.0

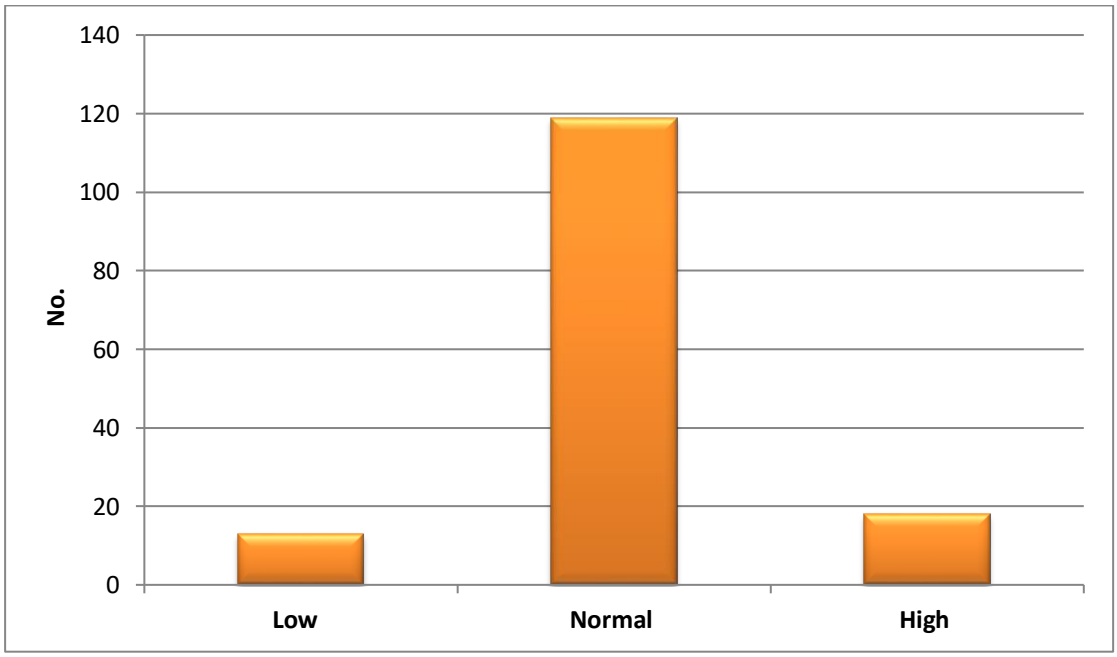

Figure 4: Baseline PR of inferior wall MI patients.

All the inferior wall MI patients had ST elevation in lead II,III, AVF and 39.3% of inferior wall MI patients had ST elevation in lead VR3,VR4,VR5,VR6 at baseline ECG. The troponin test was positive among 78.7% of inferior wall MI patients and CK-M test was positive among 14% of them. Echocardiography findings revealed Inferior wall hypokinesia among 62% among inferior wall MI patients. All these findings were shown in table 4 and figure 5.

Table 4: Investigation results of inferior wall MI patients.

Variable	**No.**	**%**
Baseline ECG: ST elevation in lead II, III,AVF		
Yes	150	100.0
Total	150	100.0
Baseline ECG: ST elevation in lead vr3, vr4, vr5, vr6		
Yes	59	39.3
No	91	60.7
Total	150	100.0
Troponin		
Positive	118	78.7
Negative	32	21.3
Total	150	100.0
CK-M		
Positive	21	14.0
Negative	129	86.0
Total	150	100.0
Echocardiography findings		
Normal	57	38.0
Inferior wall hypokinesia	93	62.0
Total	150	100.0

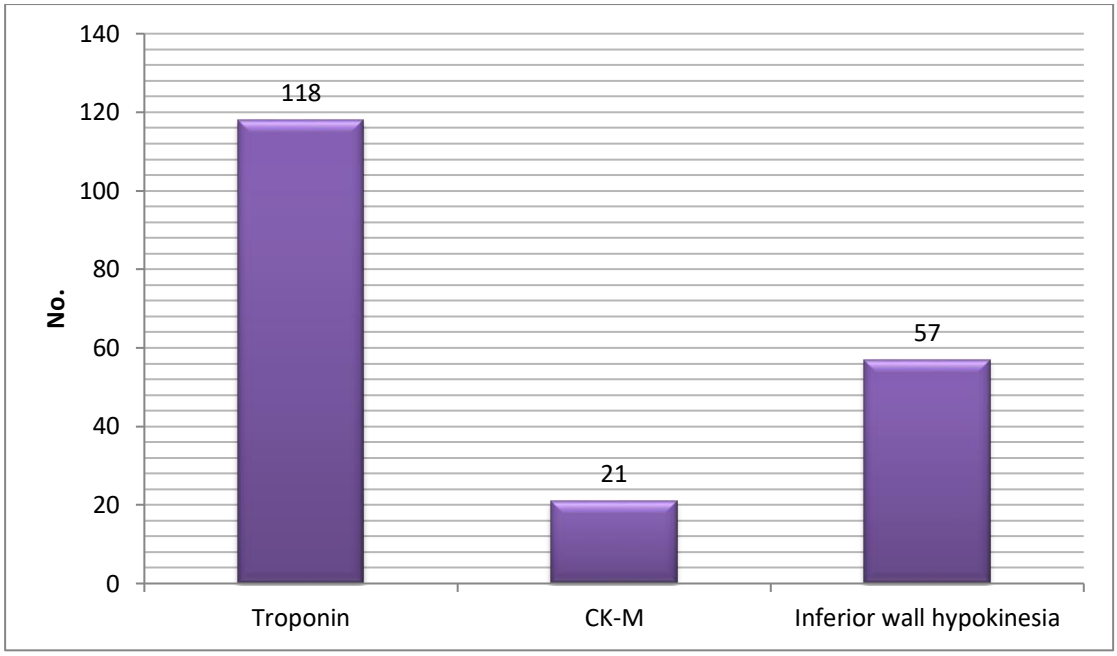

Figure 5: Investigation results.

More than two thirds (72.7%) of inferior wall MI patients had normal outcome, while 9.2% of them had cardiogenic shock, 4% atrial fibrillation , 4% bradycardia, 2.7% heart failure, 2.7% ventricular fibrillation , 2.7% hypotension and 2% bradycardia and hypotension. Right ventricular infarction was present among 39.3% of patients with inferior wall MI. All these findings were shown in table 5 and figure6.

Table 5: Outcome of inferior wall MI patients. .

Variable	No.	%
Outcome		
Normal	109	72.7
AF	6	4.0
Heart failure	4	2.7
Bradycardia	6	4.0
VF	4	2.7
Hypotension	4	2.7
Bradycardia and hypotension	3	2.0
Cardiogenic shock	14	9.2
Total	150	100.0
Right ventricular infarction (RVI)		
Present	59	39.3
Absent	91	60.7
Total	150	100.0

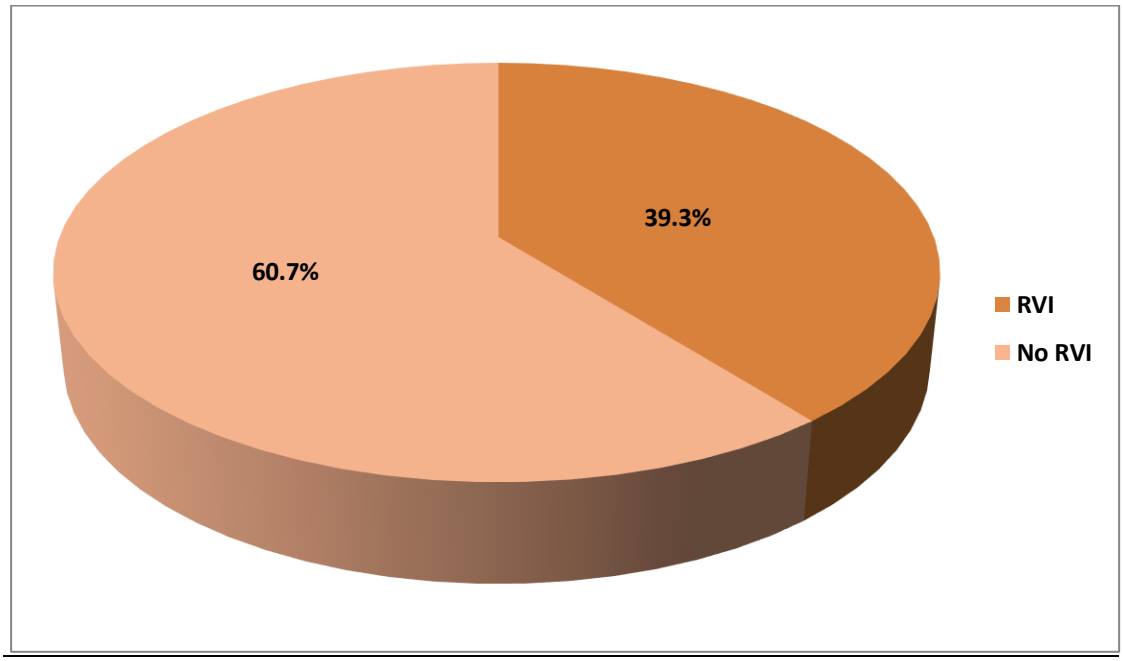

Figure 6: Prevalence of RVI among inferior wall MI patients.

There was a highly significant association between elderly age patients with inferior wall MI and RVI ($p<0.001$). No significant differences between inferior wall MI patients with RVI and those with absence of RVI regarding gender. A highly significant association was observed between self-employed inferior wall MI patients and RVI ($p<0.001$). All these findings were shown in table 6 and figure 7.

Table 6: Distribution of socio-demographic characteristics of MI patients according to RVI.

Variable	**RVI**		**No RVI**		χ^2	**P**
	No.	**%**	**No.**	**%**		
Age					33.5	**<0.001***
40-49 years	10	16.9	44	48.4		
50-59 years	25	42.4	41	45.0		
60-69 years	13	22.0	6	6.6		
≥70 years	11	18.6	0	-		
Gender					0.6	0.4**
Male	44	74.6	73	80.2		
Female	15	25.4	18	19.8		
Occupation					25.8[F]	**<0.001***
Housewife	15	25.4	18	19.8		
Public servant	8	13.6	0	-		
Self employed	27	45.8	29	31.9		
Retired	9	15.3	44	48.4		

**Significant, **Not significant*, [F]*Fishers exact test*.

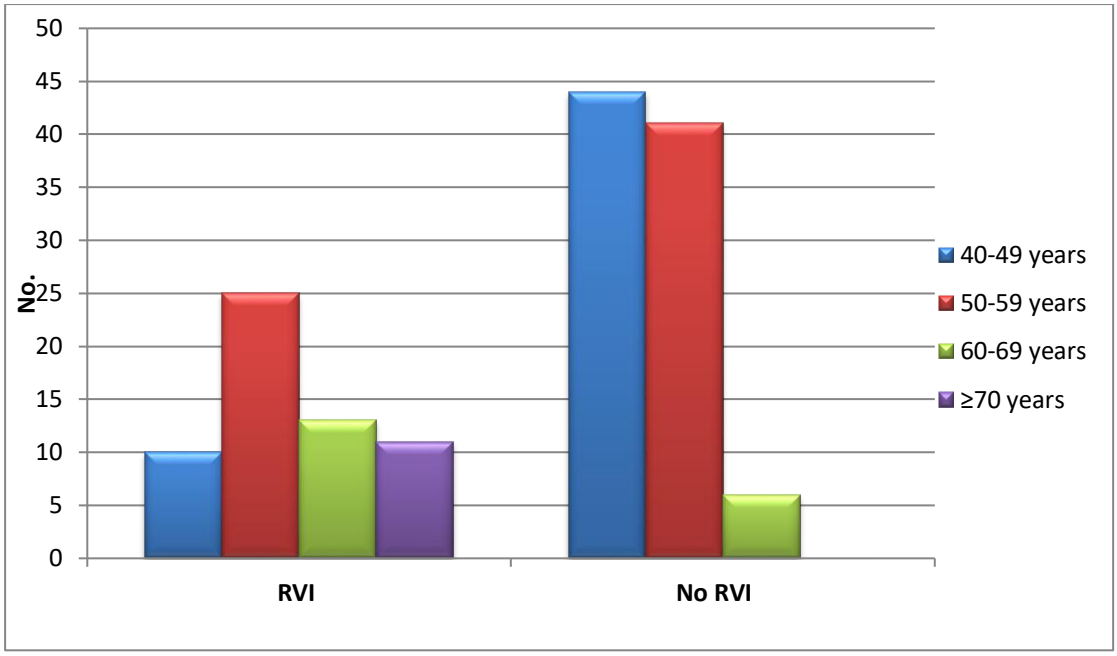

Figure 7: Distribution of age according to RVI.

No significant differences between inferior wall MI patients with RVI and those with absence of RVI regarding smoking and old IHD. There was a significant association between hypertensive MI patients and absence of RVI ($p<0.001$). A significant association was observed between diabetic MI patients and absence of RVI ($p<0.001$).The main significant risk factors for RVI among inferior wall MI patients were family history of heart diseases and obesity ($p<0.001$, $p=0.01$). All these findings were shown in table 7 and figure 8.

Table 7: Distribution of clinical risk factors of MI patients according to RVI.

Variable	RVI		No RVI		χ^2	P
	No.	%	No.	%		
Smoking					0.1	0.7**
Yes	46	78.0	73	80.2		
No	13	22.0	18	19.8		
HT					29.4	**<0.001***
Yes	6	10.2	49	53.8		
No	53	89.8	42	46.2		
DM					18.1	**<0.001***
Yes	20	33.9	63	69.2		
No	39	66.1	28	30.8		
Old IHD					1.5^F	0.2**
Yes	1	1.7	0	-		
No	58	98.3	91	100.0		
Family history of heart diseases					20.1^F	**<0.001***
Positive	12	20.3	0	-		
Negative	47	79.7	91	100.0		
Obesity					6.3^F	**0.01***
Yes	4	6.8	0	-		
No	55	93.2	91	100.0		

**Significant, **Not significant*, ^{F}*Fishers exact test*.

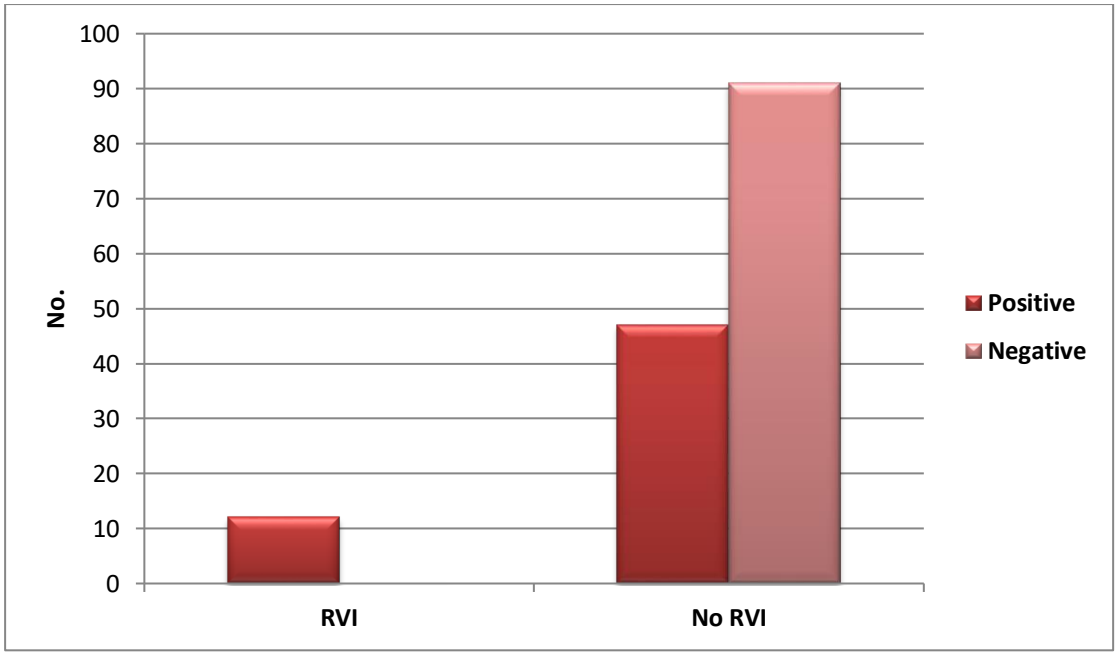

Figure 8: Distribution of family history of heart diseases according to RVI.

There was a significant association between Slow PR of patients with inferior wall MI and RVI (p=0.003). Inferior MI patients with low blood pressure were significantly associated with RVI (p<0.001). A highly significant association was observed between increased JVP and inferior wall MI patients with RVI (p<0.001). All these findings were shown in table 8 and figure 9.

Table 8: Distribution of examination results of MI patients according to RVI.

Variable	RVI		No RVI		χ^2	P
	No.	%	No.	%		
Baseline PR					11.5	**0.003***
Slow	10	16.9	3	3.3		
Normal	46	78.0	73	80.2		
Rapid	3	5.1	15	16.5		
BP					2.8	**<0.001***
Low	22	37.3	5	5.5		
Normal	37	62.7	77	84.6		
High	0	-	9	9.9		
JVP					22.7^F	**<0.001***
Normal	41	69.5	91	100.0		
Increased	18	30.5	0	-		

**Significant, **Not significant*, ^{F}*Fishers exact test*.

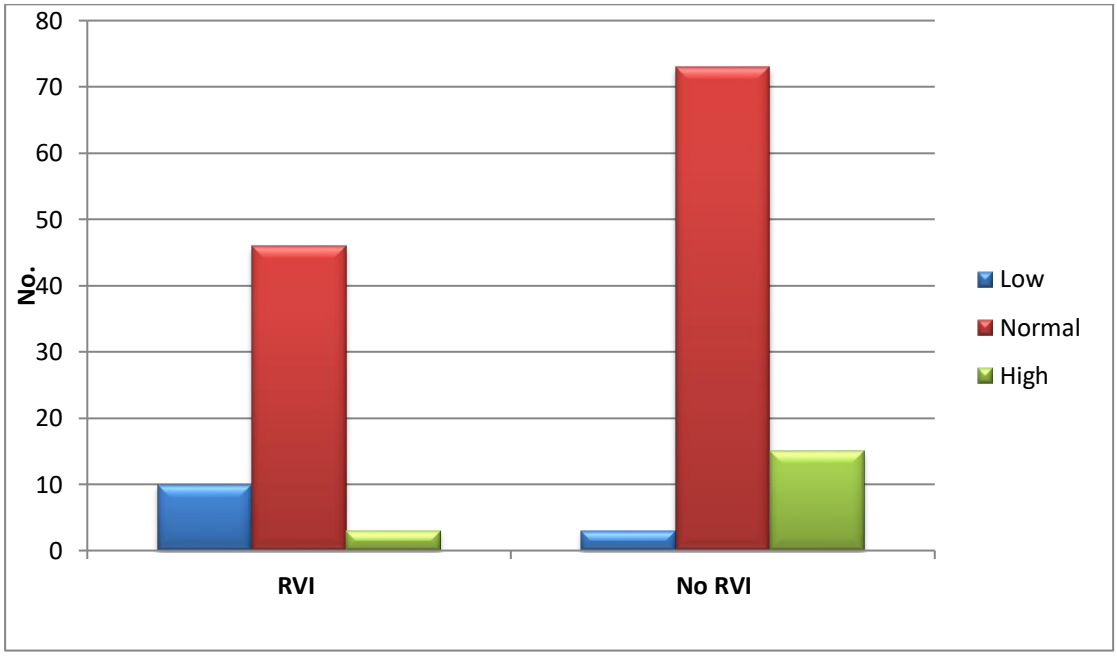

Figure 9: Distribution of baseline PR according to RVI.

The ST segment elevation in lead vr3, vr4, vr5, vr6 by baseline ECG was significantly found among RVI patients (p<0.001), while troponin test was significantly associated with inferior wall MI patients with absence of RVI (p<0.001), on other hand, CK-M test was significantly associated with RVI patients among MI (p=0.001). Inferior wall hypokinesia detected by echo was significantly higher among inferior wall MI patients with absence of RVI (p=0.02). All these findings were shown in table 9.

Table 9: Distribution of investigation results of MI patients according to RVI.

Variable	**RVI**		**No RVI**		χ^2	**P**
	No.	**%**	**No.**	**%**		
Baseline ECG: ST elevation in lead vr3, vr4, vr5,vr6					150	**<0.001***
Yes	59	100.0	0	-		
No	0	-	91	100.0		
Troponin					62.7	**<0.001***
Positive	27	45.8	91	100.0		
Negative	32	54.2	0	-		
CK-M					10.5	**0.001***
Positive	15	25.4	6	6.6		
Negative	44	74.6	85	93.4		
Echocardiography findings					5.1	**0.02***
Normal	29	49.2	28	30.8		
Inferior wall hypokinesia	30	50.8	63	69.2		

**Significant*.

Inferior wall MI patients with RVI were significantly higher complicated outcome than MI patients with absence of RVI (p=0.002). All these findings were shown in table 10.

Table 10: Distribution of outcome of MI patients according to RVI.

Variable	RVI	No	χ^2	P		
	No.	%	No.	%		
Outcome					22.7[F]	**0.002***
Normal	33	55.9	76	83.5		
AF	0	-	6	6.6		
Heart failure	3	5.1	1	1.1		
Bradycardia	3	5.1	3	3.3		
VF	0	-	4	4.4		
Hypotension	3	5.1	1	1.1		
Bradycardia and hypotension	3	5.1	0	-		
Cardiogenic shock	14	23.7	0	-		

**Significant, [F]Fishers exact test*.

As shown in table 11 and figure 10, a significant association was observed between higher age mean and RVI ($p<0.001$). There was highly significant association between slower baseline PR and RVI ($p<0.001$). Means systolic and diastolic blood pressure of MI patients with RVI were significantly lower than BP means of MI patients with absence of RVI ($p<0.001$, $p=0.001$).

Table 11: Distribution of patients characteristics means according to RVI.

Variable	RVI	No RVI	t-test	P
	Mean±SD	Mean±SD		
Age	62.7±5.2	56.8±9.6	2.2	**<0.001***
Baseline PR	68.7±15.1	82.2±13.3	8.1	**<0.001***
Systolic BP	118.5±19.4	134.1±22.1	15.7	**<0.001***
Diastolic BP	73.4±14.2	81.6±15.5	15.7	**0.001***

**Significant*.

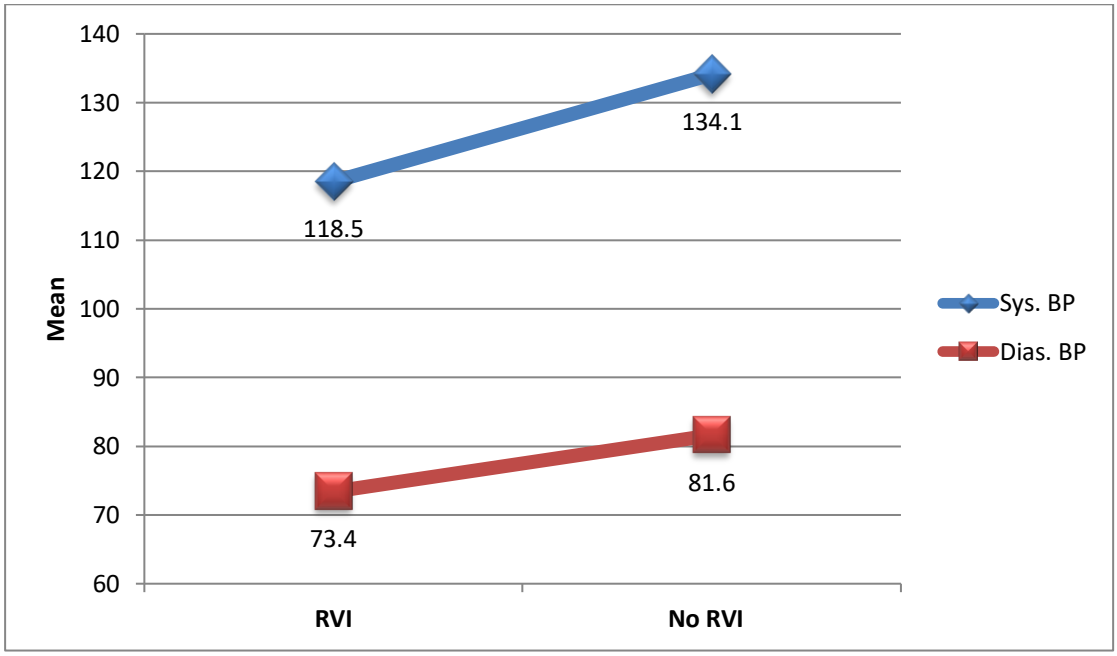

Figure 10: Distribution of BP according to RVI.

DISCUSSION

Globally, myocardial infarction (MI) prevalence in developing countries will soon be similar to MI prevalence in developed countries. MI deaths mostly are due to pathophysiological changes and complications of MI. To prevent these changes and complications, MI patients need strict and precise identification of the MI status and rapid complete management to decrease rates of morbidity and mortality[38].

Present study showed that the prevalence of right ventricular infarction (RVI) among inferior MI patients was 39.3%. This prevalence is close to RVI prevalence among inferior MI patients of 40% reported by Ravikeerthy et al [39] study in India. However, our study prevalence is lower than results of previous studies conducted in Iran (75.8%) [40] and Pakistan (48.5%) [41]. Azhar et al [42] study in Saudi Arabia reported prevalence of RVI among inferior MI patients as 30%. These discrepancies in RVI prevalence might be attributed to differences in quality of coronary intensive care, diagnostic accuracy and MI intensity in addition to differences in sample size and methodological designs between different studies. In Iraq, Sallman study [43] compared the outcome of inferior MI patients with and without RVI and found that higher incidence of complications and higher mortality rates among inferior MI patients with RVI. In previous meta-analysis study conducted by Hamon et al [44] in France, the right ventricular infarction among

inferior MI patients is highly related to many complications such as ventricular arrhythmias, cardiogenic shock, atrioventricular block and many mechanical complications.

The prevalence of right ventricular infarction among inferior MI patients is ranging between 10-50%[45].

The isolated right ventricular infarction RVI is rare (3-5%) as the right ventricle is less susceptible to ischemia and the oxygenation is low due to small muscles mass structure in addition to fact that the coronary perfusion of right ventricle presents in both systole and diastole[46].Different diagnostic criteria were used to recognize RVI among inferior MI patients that reach a maximum prevalence of about 50%[47].The reasons for this relationship between RVI and inferior MI are due to sharing the same blood supply between posterior wall of the right ventricle and the inferior wall of the left ventricle which is commonly the right coronary artery[48]. There are other reasons for uncommon isolated right ventricular infarction like full intercoronary collateral system, thinness of right ventricle wall and throttling effect of left ventricle[49].

Hemodynamic instability sometimes occurs among patients with inferior MI due to left ventricular dysfunction and right ventricular infarction[50].Despite high rates of in-hospital mortality related to right ventricular infarction among inferior MI patients, [51]low concerns and interests were focused on this disorder.The reasons of low interest were1)The ignorance in hemodynamic instability effect of RVI after inferior MI 2) The rapid change of RVI patients from normal status to shock status that is resistant to treatment 3) The effect of RVI is hidden by effect of left ventricular MI hemodynamic instability[52].The right-side precordial leads are essential in detection of right ventricular infarction depending on ST-segment elevation in lead $V_4R \geq 1.0$ mm. This ST

elevation in lead V_4R had 100% sensitivity, 87% specificity, and 92% predictive accuracy in diagnosis of RVI[53].

The main limitation of right precordial ST-segment elevation is its absence among half of RVI patients within 12 hours after pain. The ST-segment elevation in V_4R is related to right ventricle ejection fraction[54].

Our study showed a highly significant association between elderly age patients with inferior wall MI and RVI ($p<0.001$). This finding is consistent with results of Bueno et al [55] study in Spain which stated that among patients with inferior MI, the RVI incidence increased with increase of the patient's age in addition to that the complications of RVI are increased among elderly age patients. Inconsistently, Kukla et al [56] study in Poland revealed no significant difference in mean age of inferior MI patients with RVI and inferior MI patients without RVI. This inconsistency might be due to difference in sample size and designs between studies.

No significant differences were observed between inferior MI patients with and without RVI regarding patients' gender. This finding is inconsistent with results of Obradovic et al [57] study in Serbia which confirmed that women were at higher risk of RVI than men.

The self-employed inferior MI patients were significantly at higher risk of RVI than other patients ($p<0.001$). This is similar to results of Ginghina et al [58] study in Romania which documented that hard physical activity, low socioeconomic status and psychological stress are risk factors for RVI among inferior MI patients.

The smoking in this study was not significantly related to RVI among inferior MI patients. This finding coincides with results of Zornoff et al [59] study in USA. Another American study by Alemu et al [60] found that cigarette smoking increase the risk of inferior MI.

Current study showed that hypertension and diabetes mellitus were not significant risk factors of RVI. This finding is inconsistent with results of Akram et al [61] study in Iran and Roifman et al [62] study in Canada which stated that hypertension and diabetes mellitus are the major risk factors for development of RVI among inferior MI patients. This inconsistency might be attributed to fact that high proportions of inferior MI patients in present study were hypertensive and diabetics.

Family history of heart diseases in our study was significantly associated with RVI in inferior MI ($p<0.001$). This is similar to results of Namana et al [63] study in USA which stated that family history of ischemic heart diseases is significant risk factor for RVI in inferior MI patients, on other hand, Ravikeerthy et al [39] found that positive family history of heart diseases is a risk factor for isolated RVI.

In present study, the obesity is significantly associated with RVI in inferior MI patients ($p=0.01$). This finding is in agreement with Lavie et al [64] study in USA which reported that obesity is a common risk factor for RVI in inferior MI patients and the reduction of weight reduced the risk of RVI for more than 50% chance.

The significant clinical characteristics of RVI in inferior MI patients were slow PR, low BP and increased JVP. These signs are similar to results of Ondrus et al [65] study in Czech Republic and Khalid et al [66] study in Pakistan. Niaki et al [40] study in Iran revealed that the common frequent symptoms and signs of RVI in inferior MI were chest pain, disturbed consciousness, hypotension and elevated JVP.

ECG changes of RVI patients in present study was significantly ST segment elevation in lead vr3, vr4, vr5, vr6 by baseline ($p<0.001$), that is similar to findings of Jeremiah study [67] in USA which represented the ST elevation in lead vr3, vr4, vr5, vr6 is pathognomonic for RVI in inferior MI.

Elevated level of CK-MB test was significantly associated with RVI patients among inferior MI ($p=0.001$). Shemirani et al [68] study reported that CK-MB test is the best diagnostic choice for isolated RVI after ECG. The troponin test and echocardiography in our study was significantly detected inferior MI patients without RVI. The European society of cardiology stated that echocardiography and troponin test is beneficial in diagnosis of RVI in inferior MI patients in addition to diagnosis of inferior MI and its complications[69].

Current study showed that the complications were more common among patients with RVI in inferior MI with frequently significant complication of cardiogenic shock ($p=0.002$). This is similar to results of Inohara et al[70] study in Japan which documented that RVI in inferior MI is ended with higher rates of morbidity and mortality. Similarly, Bari et al [71] found that

cardiogenic shock is associated significantly with RVI in inferior MI patients.

CONCLUSIONS

- The prevalence of right ventricular infarction in inferior myocardial infarction is within normal range of previous literatures.
- The right ventricular infarction in inferior myocardial infarction was more likely to be increased among elderly age patients.
- The common risk factors of right ventricular infarction in inferior myocardial infarction were positive family history of heart diseases and obesity.
- The clinical presentation of right ventricular infarction in inferior myocardial infarction was slow pulse rate, low blood pressure and increased jugular venous pressure with electrocardiography is appropriate diagnostic choice.
- The rate of complications of right ventricular infarction in inferior myocardial infarction is high especially cardiogenic shock.

RECOMMENDATTIONS

- ❖ Instructing junior doctors in coronary care units and emergency department on prevalence and characteristics of right ventricular infarction in inferior myocardial infarction for early detection and treatment.
- ❖ Instructing junior doctors in coronary care unit to consider RVI as a cause of hypotension among other causes of hypotension in MI patients for which treatment is differ (intravenous fluids).
- ❖ Supporting the facilities of coronary care unit to prevent the complications of right ventricular infarction.
- ❖ Further multi-centers studies on prevalence and diagnosis of right ventricular infarction in inferior myocardial infarction must be supported.

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