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INVESTIGATION ON MIOCARD INFARCTION AND ANALYSIS MACROSCOPIC AND MICROSCOPIC PATTERNS

[1] Saparaliyev Umidjon Xasanovich
[1]Student of Tashkent Medical Academy,Medicine faculty,Uzbekistan
Call: +998900931575 navrozbeksaparaliyev@gmail.com

[2] Ibrohimov Bahodir Rofiqovich
[2]Student of Tashkent Medical Academy,Medicine faculty,Uzbekistan
Call: +998901107036 ibrohimovbohodir62@gmail.com

[3] Xonjonova E'zoza Zayniddinovna
[3] Student of Tashkent Medical Academy,Medicine faculty,Uzbekistan
Call: +998993670609 xonjonovaezoza@gmail.com

[4] Mamayusupova Dilshoda Hayotovna
[4]Student of Tashkent Medical Academy,Medicine faculty,Uzbekistan
Call: +998996382351 mamayusupovadilshoda9@gmail.com

Abstract
As this research includes more important discussion on the theme of myocardial infarction (MI), causes risk factors and common results after being MI, moreover, we tried to clarify what types of MI are dominant feature among the people(taking into age groups, genders and life styles account).This investigation lasted for 4 months and in this case, histological, statistical, morphological methods were in use on 10 people who suffered from MI. Also, we observed cellular alterations during the MI and compared with healthy myocardial cells under the microscope with using latest technological devices. At the end of the research, we got results which represented gender type which had high probability of being MI and influences of secondary diseases on development of MI.

Keywords: Classification of MI, autopsy, macroscopic patterns, coronary artery, spasm, stenocardia, ischemia, morphometria, thrombosis, atherosclerosis.

Introduction

Myocardial infarction is a major cause of death and disability worldwide. Coronary atherosclerosis is a chronic disease with stable and unstable periods. During unstable periods with activated inflammation in the vascular wall, patients may develop a myocardial infarction. Myocardial infarction may be a minor event in a lifelong chronic disease, it may even go undetected, but it may also be a major catastrophic event leading to sudden death or severe hemodynamic deterioration. A myocardial infarction may be the first manifestation of coronary artery disease, or it may occur, repeatedly, in patients with established disease. Information on myocardial infarction attack rates can provide useful data regarding the burden of coronary artery disease within and across populations, especially if standardized data are collected in a manner that demonstrates the distinction between incident and recurrent events. From the epidemiological point of view, the incidence of myocardial infarction in a

population can be used as a proxy for the prevalence of coronary artery disease in that population. Furthermore, the term myocardial infarction has 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 and observational studies. With these perspectives, myocardial infarction may be defined from a number of different clinical, electrocardiographic, biochemical, imaging, and pathological characteristics [1].

Causes and risk factors

More than 80% of acute myocardial infarcts are the result of coronary atherosclerosis with superimposed luminal thrombus. Uncommon causes of myocardial infarction include coronary spasm, coronary embolism, and thrombosis in nonatherosclerotic normal vessels. Additionally, concentric subendocardial necrosis may result from global ischemia and reperfusion in cases of prolonged cardiac arrest with resuscitation. Myocardial ischemia shares features with other types of myocyte necrosis, such as that caused by inflammation, but specific changes result from myocyte hypoxia that vary based on length of occlusion of the vessel, duration between occlusion and reperfusion, and presence of collateral circulation [2]. A positive family history for myocardial infarction (MI) is known to be a major cardiovascular risk factor. The current European guidelines therefore recommend intensified primary prevention for the siblings and children of persons who have had an MI. Although the genes underlying the heritable component of MI were largely unknown previously, the development of new molecular genetic methods, and particularly the advent of genome-wide association (GWA) studies, has led to the discovery of numerous genetic variants that are associated with an elevated risk of MI [3]. Cardiovascular disease has been the leading cause of death in both sexes in developed countries for decades. In general, men and women share the same cardiovascular risk factors. However, in recent trials including both men and women sex specific analyses have raised awareness of sex differences in cardiovascular risk factors due to both biological and cultural differences. Women experience their first myocardial infarction (MI) 6-10 years later than men and a protective effect of their natural estrogen status prior to menopause has been suggested. Female sex hormones have been associated with a less atherogenic lipid profile and a more healthy fat distribution. These differences are attenuated following menopause. Regarding life style the prevalence of smoking is highest in men but female smokers have a relatively higher cardiovascular risk than male smokers. Men are more physically active than women while women have healthier dietary habits. Genetic factors also affect cardiovascular risk but no sex differences have been seen. Increased cardiovascular risk attributed to psychosocial distress is similar in men and women, but since women are more prone to psychosocial distress their burden of disease is greater. Compared with a healthy population the relative risk of MI in a diabetic population is higher in women than in men. No sex difference exists in the prevalence of hypertension but it has an earlier onset in men [5].

Classification of MI

There are generally two MI categories; MI with elevation or non-elevation of ST-segment in electrocardiogram (ECG) known as STEMI and STEMI respectively. This classification is usually adopted for patients with chest ischemic manifestations in order to sake immediate management

including reperfusion therapy. However, there is another classification made according to international consensus into 5 categories (Thygesen et al, 2018). These include the followings:

Type 1 "spontaneous MI"

This type MI is associated with atherosclerotic plaque disruption such as rupture, erosion, fissuring or dissection.It may be complicated by intraluminal thrombosis and distal emboli. There may also be intraplaque hemorrhage through surface disruption of plaque cap (Falk et al, 2013; Bentzon et al, 2014).

Type 2 "ischemic-related MI"

This myocardial infarction is associated with myocardial ischemia. It is caused by an imbalance between supply and demand for O2 to the heart muscle (Chapman et al, 2017). Cases of decreased blood supply include shock, defined by hypotension below 90 mm Hg associated with organ dysfunctions' signs. They also include anemia with hemoglobin less than 5.0 mmol/L, respiratory failure, bradycardia, arrhythmia and coronary artery spasm, dissection or atherosclerosis. On the other hand, increased demands occur in conditions such as tachycardia of twenty minutes or more, and hypertension of systolic pressure over 160 mm Hg associated with ventricular hypertrophy (Saaby et al, 2013; DeFilippis et al, 2019).

Type 3 "sudden unexpected cardiac death without cardiac biomarkers"

In this type, sudden, unexpected death occurs before an elevation of cardiac biomarkers or when evidence is lacking even though the signs and symptoms are suggestive of a myocardial infarction (Thygesen et al, 2018).

Type 4 "percutaneous coronary procedure related MI"

This MI is associated with medical interference including 2 types: Type 4a resulting from percutaneous coronary intervention (PCI) or type 4b that might follow coronary stent placement (Chapman et al, 2017). It may occur per procedural or later on reflecting a complication for the stent or device like stent thrombosis or restenosis. It may be discovered by angiography or postmortem at autopsy (Rahimi et al, 2009; Thygesen et al, 2018).

Type 5 "cardiac surgery-related MI"

In this case, MI is associated with coronary artery bypass grafting(CABG).It has been reported that CABG has higher mortality rate that percutaneous coronary intervention(PCI).Furthermore, there is no further benefit to CABG over PCI[6] Myocardial injury is common in patients without acute coronary syndrome, and international guidelines recommend patients with myocardial infarction are classified by a etiology. The universal definition differentiates patients with myocardial infarction due to plaque rupture (type 1) from those due to myocardial oxygen supply-demand imbalance (type 2) secondary to other acute illnesses. Patients with myocardial necrosis, but no symptoms or signs of myocardial ischemia, are classified as acute or chronic myocardial injury. This classification has not been widely adopted in practice, because the diagnostic criteria for type 2 myocardial infarction encompass a wide range of presentations, and the implications of the diagnosis are uncertain. However, both myocardial injury and type 2 myocardial infarction are common, occurring in more than one-third of all

hospitalized patients. These patients have poor short-term and long-term outcomes with two-thirds dead in 5 years. The classification of patients with myocardial infarction continues to evolve, and future guidelines are likely to recognize the importance of identifying coronary artery disease in type 2 myocardial infarction. Clinicians should consider whether coronary artery disease has contributed to myocardial injury, as selected patients are likely to benefit from further investigation and in these patients targeted secondary prevention has the potential to improve outcomes [7].

Methods of Research

In **statistical method**, overall 10 murders who died from MI were involved and classified according to their gender, age groups and frequency of MI.

Tab 1. Patterns of our research

GENDERS (numbers)		FREQUENCY of MI		AGE GROUPS (cases)		DURATION TIME
Male	Female	Once	More than 1	40-49	4	4 MONTHS
				50-59	2	
7	3	6	4	60-69	3	
				70-79	1	
				80<...	0	

In England acute myocardial infarction was diagnosed in the first physician encounter in 307 496 (69%) of 446 744 admissions with a diagnosis of acute myocardial infarction, in the second or later physician encounter in 52 374 (12%) admissions, and recorded only as a comorbidity in 86 874 (19%) admissions. Patients with comorbid diagnoses of acute myocardial infarction had two to three times the case-fatality rate of patients in whom acute myocardial infarction was a primary diagnosis. 135 950 deaths were recorded as being caused by acute myocardial infarction as the underlying cause of death, of which 66 490 (49%) occurred in patients who were in hospital on the day of death or in the 28 days preceding death. AMI was the primary diagnosis in 32 695 (49%) of these 66 490 patients (27 678 [42%] diagnosed in the first physician encounter and 5017 [8%] in a second or subsequent encounter), was a comorbid diagnosis in 12 118 (18%), and was not mentioned at all in the remaining 21 677 (33%). The most common causes of admission in people who did not have an acute myocardial infarction diagnosis but went on to die of acute myocardial infarction as the underlying cause of death were other circulatory conditions (7566 [35%] of 21 677 deaths), symptomatic diagnoses including non-specific chest pain, dyspnea and syncope (1368 [6%] deaths), and respiratory disorders (2662 [12%] deaths), mainly pneumonia and chronic obstructive airways disease [8].

Morphological Method

Topographic Distribution of MI

According to the myocardial region involved, MI is classified as either regional when it involves the perfusion area of one epicardial artery, or circumferential when it encompasses the largest part of the circumference of the ventricular wall. Regional MI can be either transmural, usually associated with ST

segment elevations on ecg (STEMI), or only subendocardial (non-STEMI). Early reperfusion interrupts the wave front of necrosis, which limits irreversible damage to the subendocardial region only (Fig. 2). The topography of segmental MI corresponds grossly to the perfusion territory of the three large epicardial arteries or, more rarely, one of their branches such as the first diagonal branch or the obtuse marginal artery. Occlusion of the left main stem usually results in immediate death. However, in the presence of extensive collateral circulation, which occurs frequently in chronically ischemic hearts, the association between site/extent of necrosis and the occluded branch is often less obvious ('paradoxical infarction'). Application of post-mortem angiography to the excised heart or during whole body post-mortem CT-angiography(PMCTA) can provide important information on the presence and extent of collaterals between the vascular beds of major epicardial arteries (shown by a retrograde filling pattern), or neovascularization around chronic total occlusions ('bridging collaterals').Circumferential MI is mostly due to an overall fall in coronary perfusion pressure, often in the presence of severe multivessel CAD and involves in many cases only the subendocardial region. Aberrant patterns of ischemic damage such as disseminated or predominantly epicardial locations have been reported in patients who died after resuscitation (see later) or in a setting of septic shock. In the latter, ischemia likely results from inflammation-related microvascular spasm, damage or thrombotic occlusions [10]. Atrial infarctions occur in combination with ventricular infarctions and have variable reported incidence among MI patients ranging from 0.7 to 42%. Isolated atrial infarctions are scarce. The leading cause of atrial MI is coronary atherosclerosis. Pathologic significance is obviously lower than in ventricular infarctions, but for a pathologist, two potential complications of atrial MI should be underlined. The first is mural thrombus formation followed by thromboembolization, mostly pulmonary emboli (> 80% of atrial infarctions are located in right atrium), and the second, the rare cases of atrial rupture that can result in cardiac tamponade [11]

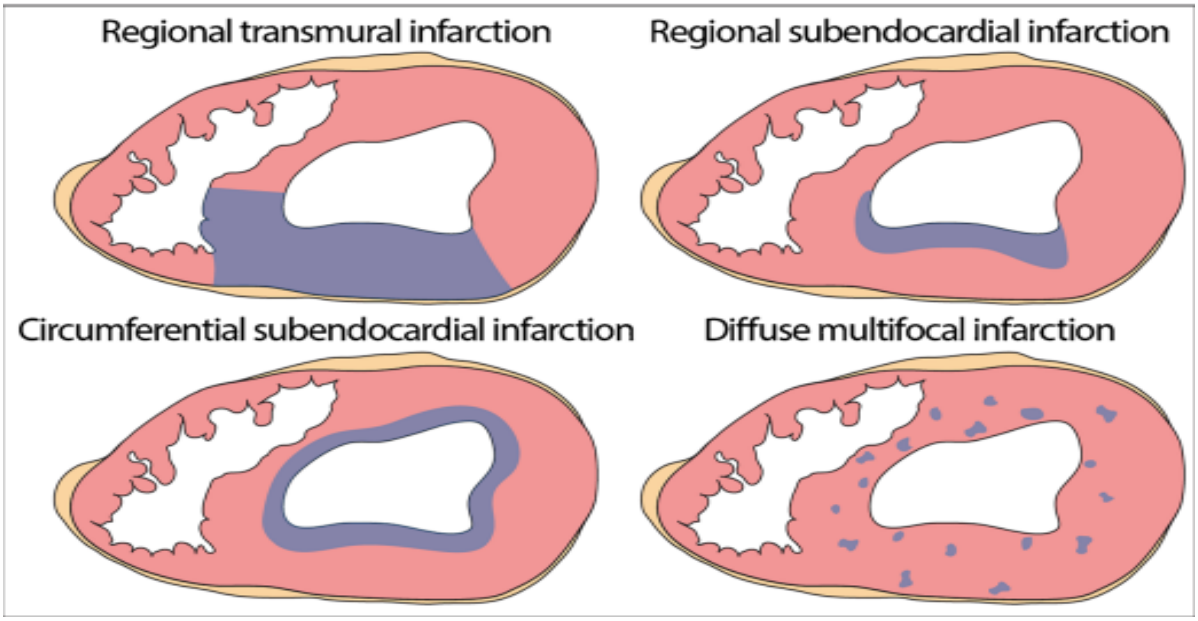
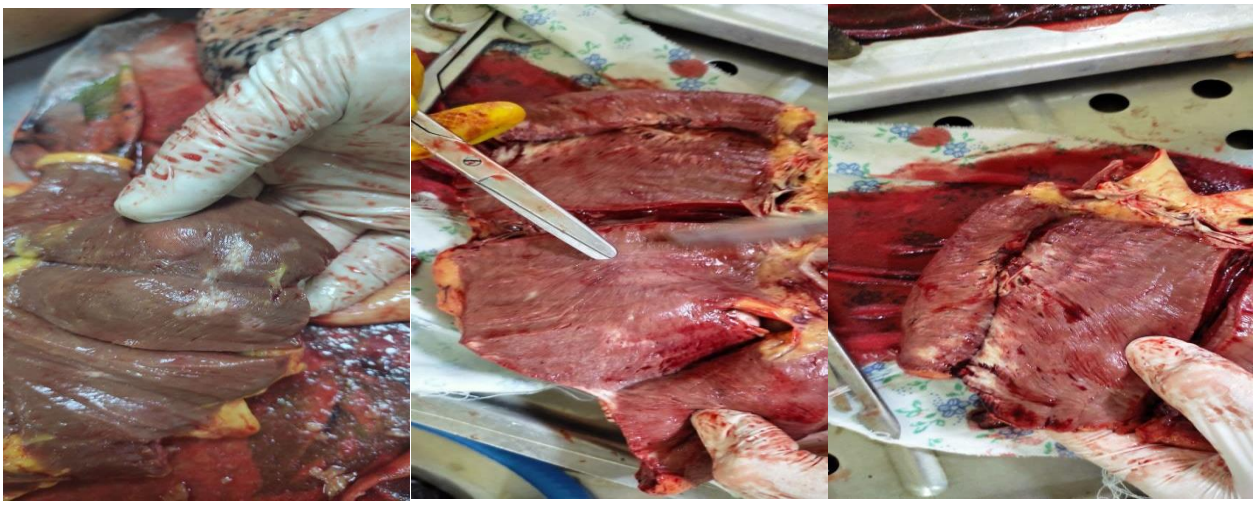


Fig.2 Patterns of topographic distribution of MI in the heart: regional transmural infarction; regional subendocardial infarction; circumferential subendocardial infarction; diffuse multifocal infarction[9]



1 2 3

Fig.2 trans mural MI 1.2 and on 3 it is intramural.

In our cases trans mural type of MI was indicated in two patterns and intramural type of MI had the biggest ratio while subendocardial was only in one statement:

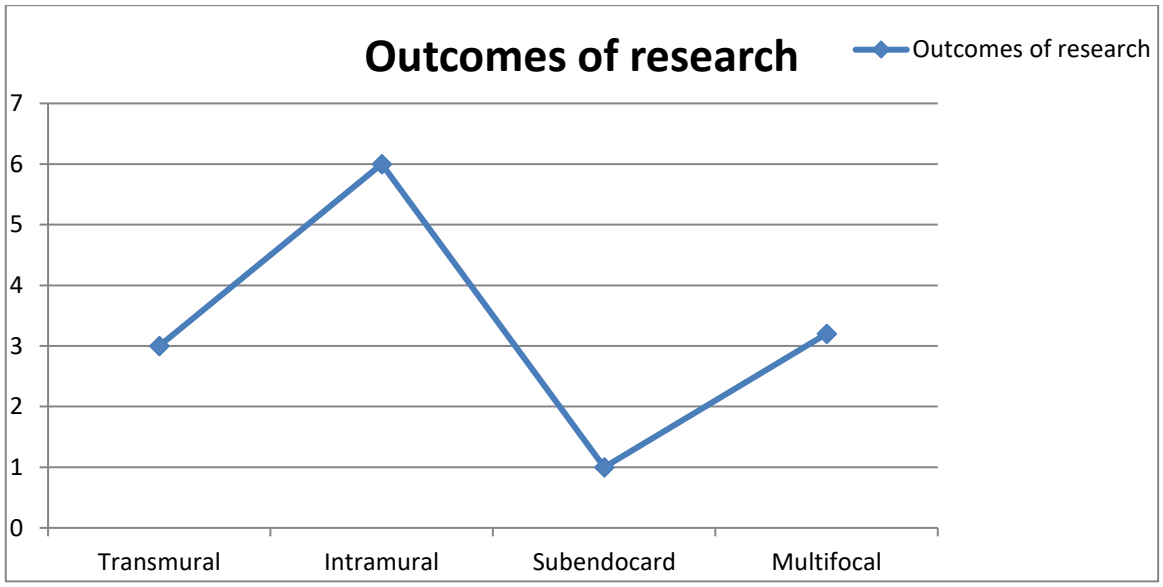


Fig.2. Statistics on topographical distribution of MI

Histological Method

The autopsy was taken from each heart tissue and made micropreparates to analyze under the microscope. After observing, these results were taken:

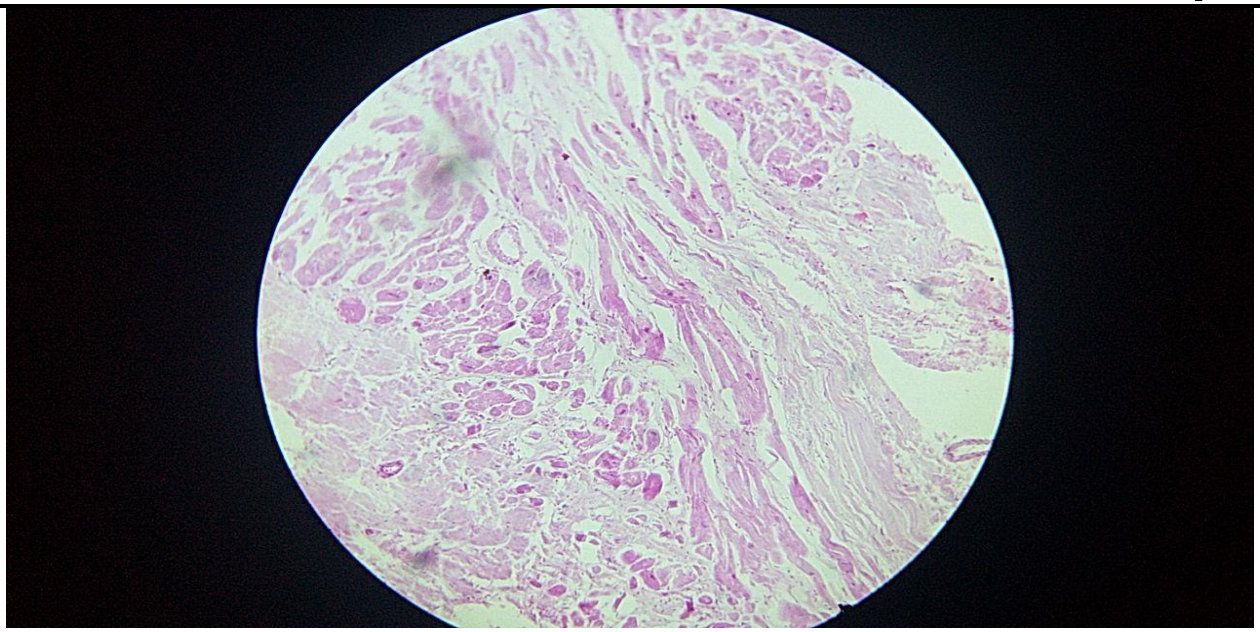


Fig.1 – structure of myocardial cells after MI

After 12 hours when viewed with the naked eye, the myocardium turns white where ischemia is observed and it is determined that the blood is uneven with the veins(before period of necrosis). After 2-4 days, it is determined that the edge of the area with Necrosis has become uneven with blood clots and has acquired a yellowish tint (period of necrosis). In the center of necrosis, the myocardium softens and a slight swelling appears, and this is called myomalacia. Then, vascularization of blood vessels is observed around the foci, and after 6-8 weeks, fibrosis tissue covers the myocardium (period of association) [12].

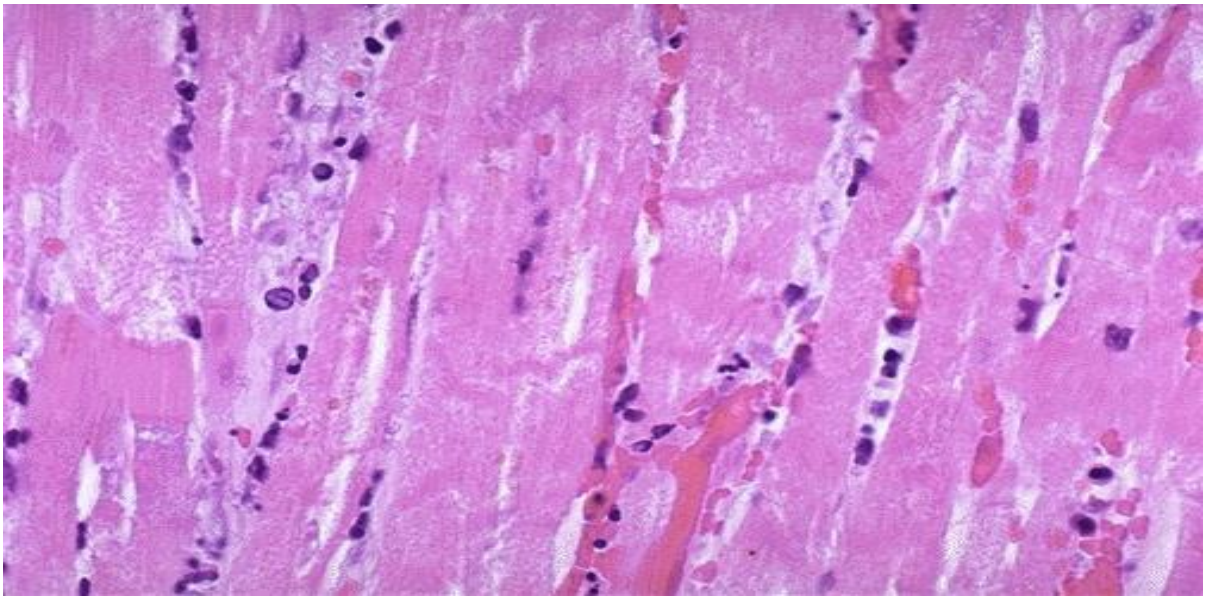


Fig.2 -This is an early acute myocardial infarction. There is increasing loss of cross striations, and some contraction bands are also seen, and the nuclei are undergoing karyolysis. Some neutrophils are beginning to infiltrate the myocardium [13].

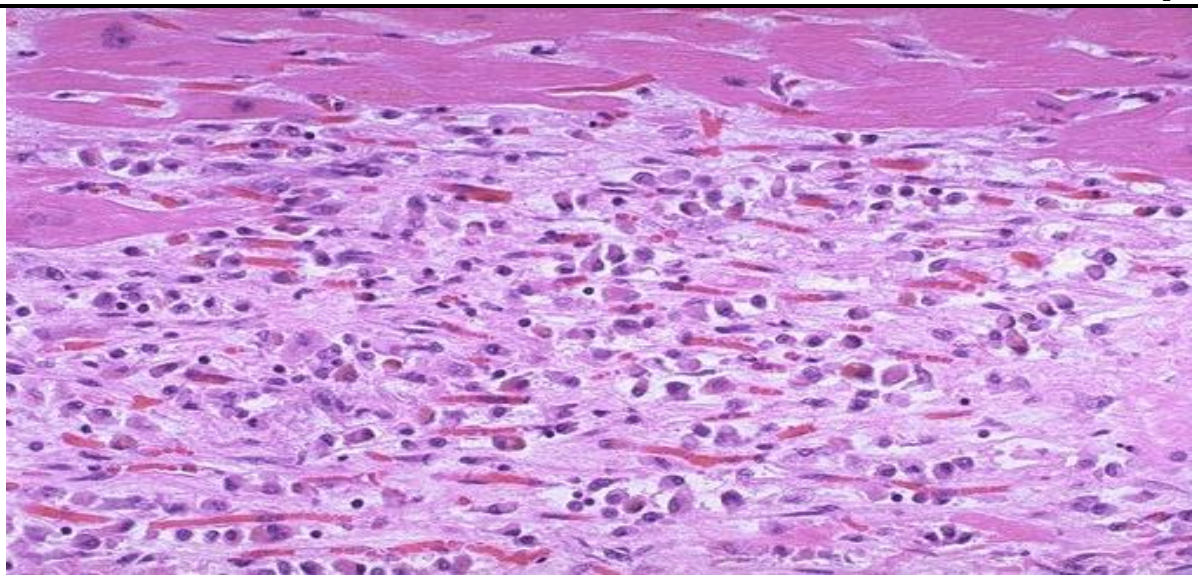


Fig-4. Toward the end of the first week, healing of a myocardial infarction becomes more prominent. Seen here is ingrowth of capillaries along with fibroblasts and macrophages filled with hemosiderin. The granulation tissue seen here is found in abundance from 10 days to 3 weeks following onset of infarction, with a peak around 1 to 2 weeks. Cardiac troponin markers may still be present in the blood up to weeks following the initial ischemic event [15].

In the last method, in morphometric, there the right ventricle of the heart is located between the sucker muscles, and thrombotic masses are detected in the earlobe. Thrombi can be seen in the left ventricular cavity. The rear wall of the left ventricle and ventricles in the aero barrier identify dense foci of many whitish colors, the dimensions of the foci are up to 2.3x1.7x1.4 cm. In the area of the anterior wall and apex of the left ventricle, foci of light yellowish color with a size of 1.3x1.2x0.8 cm, with an enveloping consistency, are determined.

To determine whether left ventricular hypertrophy following myocardial infarction leads to a complete or incomplete reconstitution of myocardial mass, the left coronary artery in rats was ligated and the animals sacrificed 30 days later. Infarcts affecting an average 43% of the ventricle were characterized by a 90% hypertrophic growth of the remaining myocardium that was inadequate for a full restoration of ventricular tissue. Myocyte hypertrophy, evaluated by changes in mean cell volume per nucleus, was again insufficient for a total recovery of the myocyte compartment of the ventricle. These observations suggest that infarcts comprising nearly 50% of the ventricle produce a sufficiently large stress on the spared myocytes to stimulate their maximal hypertrophic reserve capacity. Cardiac muscle cells, however, appear to be unable to offset by cellular hypertrophy alone the loss of mass induced by infarcts of this size. The inadequate compensatory response of the myocytes could be the underlying structural mechanism responsible for impaired ventricular function in large infarcts [16].

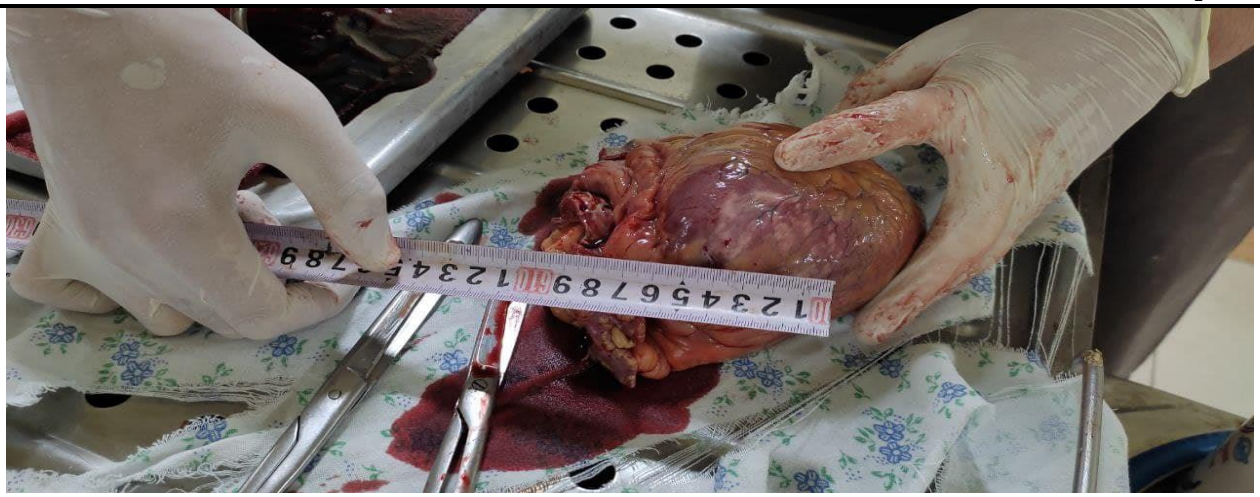


Fig-5. Process of morphometric analysis

Outcomes and discussions of this research

In this experiment, the following results are obtained:

1) Myocardial infarction was at the highest level in 40-49 age groups. In sum: 4 patients(three out of 4 were men)

2)More frequent cases were among the male genders

3)The intramural type of MI was dominant in all statements

4)In all repeated MI cases, the cause of being MI in heart was subendocardial infarction and there thrombi in area of SI which was in left ventricle, also it was a driver of BI(brain infarction) or brain stroke

Most treatment strategies are optimally suited for type 1 MI. It has been shown that both type 1 MI and type 2 MI are independently associated with cardiovascular death.[15,16,21] We confirmed these associations in a large PAD population over a median follow-up of 30 months. The risk of cardiovascular death was high for patients with type 2 MI but appeared to be even higher for those with type 1 MI. Other studies reported opposite trends[16,21] but relied on fewer type 2 MI events, and most patients had prior CAD. Our results highlight the need for further studies to identify and test treatment strategies for type 2 MI to prevent cardiovascular death.

Type 2 MI was independently associated with ALI requiring hospitalization. This information may be helpful for clinicians to identify patients at increased risk for these events. Whether this risk simply reflects advanced atherosclerosis or if MI has a causal relationship is unknown. However, there were only 4 ALI events requiring hospitalization after a first type 1 MI and only 8 after a first type 2 MI. The large confidence intervals indicate that these numbers should be interpreted with caution. Although MI treatment is mostly suited for type 1 MI, it seems unlikely that the infarction itself increases the risk for ALI (eg, through heart failure or emboli). Common risk factors or comorbidities not included in our model might explain this increased risk. Whether multidisciplinary care can avoid ALI in these comorbid patients at risk or whether this increased risk would justify revascularization despite low event rates deserves further studies [17].

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