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THE LEVEL OF INFLAMMATORY MARKERS IN PATIENTS WITH MYOCARDIAL INFARCTION AFTER PERCUTANEOUS CORONARY INTERVENTION

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Cardiovascular diseases are among the most widespread diseases in the world that affect all ages and sometimes can lead to death. Atherosclerosis, coronary syndrome and myocardial infarction are usually associated with artery occlusion and require percutaneous coronary intervention (PCI) as a non-surgical procedure to restore blood flow to the heart. Inflammatory biomarkers, especially interleukins and cardiac biomarkers, have an important role in diagnosing the state of patients with heart damage. The goal of the study was to estimate the serum levels of interleukins and cardiac biomarkers after PCI to reduce the risk of acute coronary syndrome. The study included 100 persons between the ages of 40 and 69 diagnosed with acute coronary syndrome who had successful PCI and a control group consisting of 50 healthy participants of the same age. The levels of interleukins, creatine kinase MB and myoglobin were measured using an enzyme-linked immunosorbent assay. Troponin and D-dimer levels were measured using immunoassay. It was found that patients before PCI had significantly higher levels of IL-1β, IL-6, IL-8, cardiac troponin I, D-dimer, creatine kinase-MB and myoglobin compared to the control group. One day after PCI, the levels of IL -6, IL-8, cardiac troponin I and D-dimer remained elevated. One week after PCI, the levels of IL-1β, IL-6, IL-8, CK-MB and myoglobin did not show significant differences compared to the control group, while the levels of cardiac troponin I and D-dimer remained higher. Results obtained indicate that in patients after PCI, the levels of interleukins decreased, indicating the reduction of inflammatory processes, but cardiac damage persists to a certain degree, even a week after PCI.

K e y w o r d: percutaneous coronary intervention, myocardial infarction, interleukin, creatine kinase MB, D-dimer, myoglobin.

Myocarditis is an inflammation of the heart that is characterized based on certain criteria in histology, immunology, and immunohistochemistry, as defined by the World Health Organization [1, 2]. Myocarditis is often caused by viral infections, toxic chemicals, and autoimmune processes [3, 4]. Myocardial infarction can be classified depending on the presence or absence of ST-segment elevation on the electrocardiogram (ECG) to ST-segment elevation myocardial infarction (STEMI), which forms up approximately 25–40% of MIs and non-ST Segment Elevation Myocardial Infarction (NSTEMI), which forms up approximately 60–75% of MIs. Acute myocardial infarction is myocardial necrosis resulting from acute obstruction of a coronary artery. Symptoms include chest discomfort with or without dyspnea, nausea, and/or diaphoresis. Diagnosis is determined by electrocardiography (ECG) and the presence or absence of biomarkers [3, 4]. Complete and prolonged occlusion of an epicardial coronary blood vessel typically leads to STEMI, while NSTEMI usually results from severe coronary artery narrowing, transient occlusion, or microembolization of the thrombus [5]. The time standard for percutaneous coronary intervention is 90 min and no more in a hospital equipped

with the tools and equipment necessary to perform percutaneous intervention. A meta-analysis of 23 trials confirmed that performing primary intravenous interventions reduces the mortality rate by 2% [4]. Studies indicate that when the procedure to restore blood flow to the heart, known as percutaneous coronary intervention, is completed in 90 min or less, patients tend to experience less heart damage, face a reduced number of heart-related complications, and have improved chances of survival over time [5]. The duration between a patient's arrival at the hospital and the start of their percutaneous coronary intervention – a heart procedure to clear blocked arteries is critical. Shorter times can significantly lower the risk of further heart complications and are key in improving patient survival rates. In 2014, at the King Faisal Cardiac Centre, one in four patients managed to receive this crucial heart procedure within 90 min of arriving, which is an optimistic outcome for emergency heart care [5, 6]. When the heart muscle does not receive enough air, it hurts. This is called angina pectoris. For people with this medical condition, pain in the chest, neck, shoulder, back, or arm gets worse when they move or are stressed, but it goes away quickly when they rest or take nitroglycerin. A lot of people who have coronary heart disease (CHD) have angina [6, 7]. Major arterial problems are about twice as likely to happen if you have chronic angina. Studies that followed people for 1 to 9 years found that risk factors like getting older, having a serious form of angina, having heart function problems, and having other diseases at the same time (like diabetes and chronic kidney disease) can make the disease worse [8, 9]. The interleukin (IL-1) family of cytokines has long been associated with regulating several cellular processes, such as vascular permeability, leukocyte adhesion, endothelial cell thrombogenic response, and extracellular matrix production [10, 11]. IL-1 is an important factor in the etiology of cardiac remodeling and plays an important part in the activation of the inflammatory response that occurs after a heart attack. As a result, the signaling cascade of IL-1 inhibition may be an effective treatment target for MI patients [12]. IL-5 is a pleiotropic cytokine known to stimulate B cells and eosinophils. In particular, it was shown that human IL-5 increased the reactivity of specific pathogen-stimulated B-cells and was a critical element for terminal eosinophil differentiation as well as eosinophil activation [13, 14]. IL-6 is considered an inflammatory cytokine secreted from monocytes and macrophages in response to the secretion of interleukin 1. It plays an important role in inflammatory processes in the body [15]. Interleukin-8 (IL-8), or CXCL8 based on the latest nomenclature, represents the prototypical chemokine of the CXC subfamily that recruits specific target cells and mediates inflammation and wound healing [16, 17]. Myoglobin can make oxymyoglobin, carboxymyoglobin, or metmyoglobin and works similarly to hemoglobin in that it can reversibly bind oxygen. However, unlike hemoglobin, myoglobin only possesses one oxygen-binding site with a far higher affinity. Therefore, at times of hypoxia, anoxia, or high metabolic activity, myoglobin may either store oxygen or transfer it to muscle cells through the Bohr effect at the tissue level [18, 19]. D-dimer, which is produced when cross-linked fibrin degrades. Nonetheless, due to the specificity of serum D-dimer levels for fibrin, numerous other conditions, including acute NSTEMI, may induce an increase in levels. The most useful biomarkers for the diagnosis of acute NSTEMI and acute coronary syndrome are cardiac troponins [19, 20]. Creatine kinase-MB (CK-MB) is one of the three isoforms of the CK enzyme. CK is not specific for MI since it is found in skeletal, brain, and cardiac tissues. There are instances when CK increases with mild myocardial infarction may not be noticed, suggesting insufficient sensitivity. As a result, the CK test was eventually replaced by the CK-MB. For the last 20 years, CK-MB has been regarded as the gold standard for acute myocardial infarction due to its greater specificity in the heart muscle [20]. Inflammatory biomarkers, especially interleukins and cardiac biomarkers, have an important role in diagnosing and treating the inflammatory state of cardiovascular disease. In our study, inflammatory and cardiac factors were measured and monitored to determine the effect resulting from inflammation and the increased risk of heart disease, especially acute coronary syndrome. Cardiac catheterization or percutaneous coronary intervention (PCI) was first introduced in 1977. The PCI procedure is considered important because of its role in reopening closed blood vessels to save patients' lives and reduce the risk of infection that could lead to death [20]. Increasingly complicated lesions and patients with a history of clinically significant cardiac disease, coronary artery disease risk factors, comorbid diseases, or structural risk factors may now be treated thanks to experience with this method and advancements in technology [21]. The insertion of a catheter into

the blood vessels occurs through the arm or groin. A catheter is inserted through the blood vessels into the heart, specifically into the constricted coronary artery, utilizing fluoroscopy, a specialized form of X-ray imaging. A distended balloon tip covered with a stent is produced once the tip is in position. The stent is enlarged and the plaque is compressed by the balloon tip. After the stent has been positioned and the plaque has been compressed, the balloon is deflated and withdrawn. Stent insertion maintains the artery's open position [20, 21]. The relationship between inflammation and heart disease and catheter intervention is very important to improve patient outcomes and could expand understanding to discover or identify new therapeutic strategies that reduce inflammation or the risk of acute coronary syndrome. The study aims to determine the role of intravenous intervention on the levels of inflammatory factors, especially interleukins.

Materials and Methods

The study examined a cohort of 50 individuals who were in good health (30 males and 20 females) as a control group. Additionally, it included 100 individuals (50 males and 50 females) who had acute coronary syndrome and underwent successful percutaneous coronary intervention (PCI) at the Nasiriya Heart Center. The time following the event after the intravenous intervention was performed depends on the type of disease. It was in ST-segment elevation myocardial infarction (STEMI), so the time frame was 90 to 100 min of medical contact. For non-ST-segment elevation myocardial infarction (NSTEMI) or unstable angina, the time frame was divided into rapid intervention within 2 h and delayed intervention from 24 to 72 h. The study group was divided into two factions. The patients' group was assessed before PCI, 24 h post-PCI, and 1 week post-PCI to obtain their measurements; however, 50 patients refused to participate after one week, so the parameters were determined after 1 week only in 50 patients. The sample population comprised individuals aged 40 to 69. Between November 2021 and April 2022, all patients in this investigation were diagnosed by specialists, and their diagnoses were confirmed by clinical and laboratory tests. Access to the bloodstream is achieved through either the femoral or radial arteries. Ten milliliters of blood were taken from each patient before and after catheterization. Anticoagulant tubes containing sodium citrate were used to transfer 1.8 ml of blood. The sample was subsequently centrifuged at 3000 rpm for 15 min to separate the components, from which the plasma was isolated and stored at -20°C until analysis. As a result of the propensity for these conditions to independently modify the variables of the study. To check the amounts of myoglobin, CK-MB, IL-8, IL-5, and IL-6, an enzyme-linked immunosorbent assay (ELISA) was used, as directed by Sunlog (China). An immunoassay was conducted utilizing the Avis-6 apparatus to quantify the concentrations of troponin and D-dimer according to the company's kits, Boditech Med Inc. Korea. Patients taking treatments for heart disease such as statins and aldosterone antagonists were excluded, and those suffering from autoimmune diseases or malignant tumors were excluded.

Ethical approval. Ethical approval was received from the ethical and research committee of Thi-Qar Directirate Alhabbobi Teaching Hospital (protocol number and date are based on the official facilitation letter number 344, 10/11/2021). Informed consent was obtained from all caregivers of patients who participated in the research.

Statistical analysis. Statistical analysis is often used to analyze quantitative data and provides methods for data description and simple inference for continuous and categorical data. The procedure involves the collection of data, leading to a test of the relationship between two statistical data sets. In this study, all data are presented as mean ± SD. The statistical analyses were performed using SPSS (version 26) and using dependent t-tests (two-tailed) and independent t-tests (two-tailed) for normally distributed variables, whereas the Mann-Whitney and Wilcoxon tests were used for those variables that were not normally distributed. $P < 0.05$ was considered statistically significant.

Results

Socio-demographic characteristics of the study groups. In this study, a comparative analysis was performed between two groups: the control group, which included 50 individuals (age 45.22± 3.12), and the patient group, which included 100 patients (age 47.63 ± 4.82). Regarding gender distribution, which does not indicate the presence of statistically significant differences, regarding the distribution of females, there were 20 females in the control group compared to 50 females in the patient group. The total number of individuals in both groups was 50 in the control group and 100 in the patient group;

there were no significant differences by conventional statistical standards.

Comparison of the parameters between the control group and patients pre-PCI. Before PCI, patients had statistically significantly higher levels of IL-1β, IL-6, IL-8, cardiac troponin I, D-dimer, CK-MB, and myoglobin compared to the control group, which indicates the presence of an inflammatory state and increased myocardial damage in these patients. On the other hand, IL-5 levels were significantly lower in patients compared to the control group. These data provide a profound understanding of the biochemical and inflammatory status of patients in the pre-PCI period (Table 1).

Comparison of the parameters between the control group and patients 1 day post-PCI, and patients 1 week post-PCI. One day after PCI, patients had statistically significant higher levels of IL-6, IL-8, cardiac troponin I, and D-dimer than did the control group. The results showed no statistical significance in the levels of interleukin 1 beta and interleukin 5 in the group one day after PCI compared to the control group. A significant increase in myoglobin levels was observed in patients one day after the PCI procedure compared to the control group. Myocardial injury and inflammatory alterations that can follow PCI were shown in the statistically significant increased levels. One week after PCI, the study found a statistically significant decrease in the levels of IL-1β and IL-6 in patients compared to the control group, while the levels of cardiac troponin I and D-dimer increased statistically significantly. Other components, such as IL-5, IL-8, CK-MB, and myoglobin, showed non-significant differences compared to the control group. These results may indicate that inflammatory processes and cardiac damage persist to a certain degree, even a week after PCI (Table 2).

Table 1. Comparison of the parameters between the control group and patients pre-PCI

Parameters	Control group, $n = 50$	Patients Pre-PCI, $n = 100$
IL-1β, pg/ml	88.41 ± 16.09	123.56 ± 19.66
IL-5, ng/l	11.65 ± 2.76	9.76 ± 2.88
IL-6, ng/l	4.09 ± 0.73	6.44 ± 1.08
IL-8, ngl	7.65 ± 0.12	9.90 ± 1.65
cTnI, ng/ml	0.019 ± 0.002	0.689 ± 0.213
D-dimer, ng/ml	357.63 ± 61.16	523.87 ± 87.86
CK-MB, ng/ml	3.66 ± 1.54	5.92 ± 1.77
Myoglobin, ng/ml	48.52 ± 6.77	71.64 ± 12.82

Note: mean ± SD, $P < 0.001$

Comparison of the parameters between patients pre-PCI and patients one day post-PCI, and patients one week post-PCI. Comparing patients before and one day after PCI, data showed a statistically significant decrease in levels of IL-1β, cardiac troponin I, CK-MB, and myoglobin, as well as a significant increase in the levels of IL-5, IL-8, and D-dimer. There were no statistically significant differences in IL-6 levels between the two groups. These findings suggest important biochemical and inflammatory changes that occur after PCI that may

Table 2. Comparison of the parameters between control group and patients one day post-PCI and one week post-PCI

Parameters	Control group, $n = 50$	Patients one day post-PCI, $n = 100$	*P* value	Patients one week post-PCI, $n = 100$	*P* value
	mean ± SD	mean ±SD		mean ±SD	
IL-1β, pg/ml	88.41 ± 16.09	85.92 ± 13.52	0.423	81.66 ± 10.53	<0.01
IL-5, ng/l	11.65 ± 2.76	11.12 ± 1.32	0.998	10.87 ± 1.54	0.443
IL-6, ng/l	4.09 ± 0.73	6.85 ± 1.09	<0.001	4.12 ± 0.32	0.345
IL-8, ng/l	7.65 ± 0.12	11.43 ± 2.06	<0.001	8.43 ± 1.03	0.098
cTnI, ng/ml	0.019 ± 0.002	0.399 ± 0.188	<0.001	0.025 ± 0.007	<0.001
D-dimer, ng/ml	357.63 ± 61.16	866.52 ± 165.74	<0.001	481.94 ± 145.87	<0.01
CK-MB, ng/ml	3.66 ± 1.54	4.21 ± 0.65	0.234	4.62 ± 1.54	0.963
Myoglobin, ng/ml	48.52 ± 6.77	61.64 ± 12.74	<0.05	55.09 ± 10.55	0.456

Table 3. Comparison of the parameters between patients pre-PCI and patients one day post-PCI, and patients one week post-PCI

Parameters	Patient pre-PCI, *n* = 100	Patients one day post-PCI, *n* = 100	*P* value	Patients one week post-PCI, *n* = 100	*P* value
	mean ± SD	mean ±SD		mean ±SD	
IL-1β, pg/ml	123.56 ± 19.66	85.92 ± 13.52	<0.001	81.66 ± 10.53	<0.001
IL-5, ng/l	9.76 ± 2.88	11.12 ± 1.32	<0.001	10.87 ± 1.54	0.001
IL-6, ng/l	6.44 ± 1.08	6.85 ± 1.09	0.876	4.12 ± 0.32	<0.001
IL-8, ng/l	9.90 ± 1.65	11.43 ± 2.06	<0.001	8.43 ± 1.03	< 0.01
cTnI, ng/ml	0.689 ± 0.213	0.399 ± 0.188	<0.001	0.025 ± 0.007	<0.0001
D-dimer, ng/ml	523.87 ± 87.86	866.52 ± 165.74	<0.001	481.94 ± 145.87	0.086
CK-MB, ng/ml	5.92 ± 1.77	4.21 ± 0.65	<0.01	4.62 ± 1.54	<0.001
Myoglobin, ng/ml	71.64 ± 12.82	61.64 ± 12.74	<0.01	55.09 ± 10.55	<0.001

be of interest in evaluating and managing the effects of this intervention. When comparing biochemical measurements between patients before and one week after PCI, the results showed statistically significant decreases in the levels of IL-1β, IL-6, IL-8, cardiac troponin I, CK-MB, and myoglobin, while IL-5 levels increased. No statistically significant difference appeared in D-dimer levels. These results indicate a significant improvement in inflammation and myocardial damage over time after PCI, which may have a positive impact on the overall health status of patients (Table 3).

Discussion

In this study, when comparing IL-1β levels between the control group and patients, there was a highly significant increase ($P < 0.001$) in serum IL-1β among patients pre-PCI. One day post-PCI, IL-1β dropped to show a non-significant difference compared to healthy. Weeks post-PCI also showed a non-significant difference compared to healthy. When comparing the IL-1β levels of patients one week, one day, and before PCI, a substantial and statistically distinguishable difference ($P < 0.001$) was seen among the patient groups. The study conducted by Bai et al. demonstrated that there is a large discrepancy in the levels of IL-1 beta between the patient group and the control group due to the role of IL-1 in causing inflammation, as high levels can be clear evidence of an increased risk of infection, and this is consistent with our current research [22]. The results of some studies showed that levels of IL-1β were high in the group of patients with cardiovascular disease compared to the control group, and the levels were at their peak approximately half an hour after suffering a heart attack, but after intravenous intervention, levels of IL-1 decreased after three days [23]. According to the findings by Li and colleagues in 2020, the levels of a substance in the blood known as IL-1β were measured before conducting the percutaneous coronary intervention, a heart procedure. It turned out that there was no notable statistical difference in IL-1β levels between patients who later experienced a re-narrowing of their arteries (restenosis) and those who did not [24]. IL-1β is part of the IL-1 family and is a key player in triggering inflammation. It is produced primarily by immune cells such as macrophages and monocytes. In instances of tissue damage within the heart, IL-1β is released in large quantities as a reaction to the inflammation. The presence of IL-1β, along with other complements and cytokines, actively contributes to creating reactive oxygen species (ROS), which can increase the harm to the heart muscle, exacerbating the damage [25]. Following ischemic damage, the heart experiences remodeling and repair via an inflammatory response. The cryopyrin inflammasome is responsible for coordinating the cellular amplification of the inflammatory response following tissue injury. Toxic and injury-causing substances trigger the inflammasome. As a result, it causes the activation of IL-1β by cutting pro-IL-1β with caspase-1, which is the enzyme responsible for the inflammasome's effects. IL-1β promotes the attraction of white blood cells to the injured heart muscle, which is a key feature of the widespread inflammatory reaction

and enhances the generation of chemokines and cytokines [26]. The current data about the correlation between IL-5 and ischemic heart disease is insufficient. The current investigation found that patients before PCI had significantly decreased IL-5 levels compared to healthy controls ($P < 0.001$). When comparing the control group with the groups who received PCI one day and one week later, it was seen that IL-5 levels reverted to normal [27]. This indicates the role of intravenous intervention in reducing the levels of cytokines, especially those that cause inflammation, which helps reduce the resulting risks that may be caused by immune inflammatory factors. At the same time, some studies have shown an inverse relationship between IL-5 levels and cardiovascular disease, according to Knutsson et al. [28].

These investigations have demonstrated that splenectomy has a proatherogenic impact, but the transfer of B1 cells can counteract this impact. Furthermore, this counteracting effect is contingent upon the B1 cells' capacity to manufacture IgM antibodies [29]. One important trigger is IL-5, which is released from type 2 innate lymphoid cells (ILC2). However, the exact steps that B1 cells take to make danger-associated molecular pattern-specific IgM antibodies are still not well understood [30]. In the study, when comparing IL-6 between the control group and patients, we noticed a highly significant increase in serum IL-6 among patients pre-PCI compared to the control group ($P < 0.001$). One day post-PCI, IL-6 continued to rise to show a significant difference compared to healthy ($P < 0.001$). One week post-PCI, in spite of IL-6 decreasing, it still showed a significant difference compared to healthy. Based on the research conducted by Groot et al., the initial concentration of IL-6 was determined to be 3.7 pg/ml. The concentration increased fourfold within 24 h, reaching 10.3 pg/ml ($P < 0.001$). However, it then decreased to 1.8 picograms per milliliter ($P < 0.001$) after two weeks [31]. In the study conducted by Tøllefsen et al., an examination was conducted on 256 people suffering from myocardial infarction. It was found that the levels of interleukin 6 increased at the beginning of the injury until 24 h had passed, and then a decrease in the levels of interleukin was observed after four months. The decline reaches its peak. Regarding the study conducted by Sun et al., there was an increase in IL-6 levels in restenosis compared to people who were in good health and had an appropriate response to treatment [31, 32]. Cytokines play an important role in causing inflammation, especially IL-6, as they lead to an increase in blood viscosity and the number of platelets. It can affect the deposition of fibrinogen in the blood vessels and lead to atherosclerosis. It has an important role in the development of cardiovascular diseases [33, 34].

A study found that IL-8 levels increase in people with cardiovascular disease. On the contrary, some studies have shown high levels of IL-8 after the intravenous intervention, and approximately a week after the intravenous intervention, the interleukin levels continued to rise A study conducted by Correia et al. showed the presence of different levels of IL-8 before and after the intravenous intervention. Also, there are statistically significant differences in the levels of interleukin in the blood of people with cardiovascular disease compared to the control group, according to Lima et al. and Khalid et al. [35, 36]. Monocytes are attracted to the subendothelial space by IL-8. Furthermore, it increases the likelihood of plaque formation and also impacts arterial smooth muscle cells by inducing cell division and attracting other cells [37-39]. The study revealed that compared to the control group, the patients group before PCI had higher D-dimer levels. D-dimer levels rose in the patient group statistically substantially more than in the control group one day after the PCI procedure. In the patient group one week after the PCI procedure, there was also a significant increase. These results corroborate those of Gong et al. [40]. The results of the study conducted by Gong et al. showed that D-dimer levels can predict cardiac events that affect patients. The study included 2410 patients whose angiography proved the existence of coronary artery disease (CAD) [40, 41]. Meta-analysis study conducted by Biccirè et al. concluded that increased D-dimer was linked to in-hospital and short- and long-term complications in ACS patients. D-dimer was also higher in patients with the no-reflow phenomenon. D-dimer may be used to identify ACS patients who still have a thrombotic risk [42]. D-dimer levels reflect increased coagulation and fibrinolytic activity. High amounts of D-dimer are linked to some unhealthy processes, such as thrombosis and plaque necrosis. During the chronic phase of atherosclerosis, changes can be seen in how the blood clots and how fibrin breaks down. The development of thrombi at the site of atherosclerotic lesions, especially on a plaque that has ruptured, is a key part of atherothrombosis [43]. The levels of CK-MB and myoglobin were higher in the patients

who had PCI than in the comparison group. One day after the PCI treatment, there was a statistically significant rise in myoglobin levels in the patient group compared to the control group. However, there were no statistically significant changes in CK-MB levels. It also showed no statistical significance in the group of patients one week after the PCI procedure. These results are consistent with Montaser et al. and Zhoa et al. [44, 47]. Based on the research conducted by Montaser et al., a median concentration of CK-MB and myoglobin was found to be 6.5 ng/l that is higher in the MI group than in the control group (1.4 ng/l) ($P < 0.001$) [44]. People who have high levels of CK-MB (≥4.730 ng/ml) and have coronary heart disease are more likely to die, according to research results by Wu et al. [45]. The results showed that people who have coronary heart disease have high CK-MB levels, according to the research conducted Wu et al. [45]. High levels of CK-MB can lead to death [46]. The decrease in CK-MB and myoglobin levels after PCI is evidence of the important role of percutaneous coronary intervention on cardiac biomarkers. The study conducted by Zhoa et al. also proved that myoglobin levels were high in all study groups with heart disease, especially coronary artery syndrome [47]. Although the results indicated that all patient groups' cardiac troponin levels increased significantly more than those of the control group both before and after the PCI operation, these findings are in line with Baulk et al. [48]. Studies abound that show troponins start to show up in the blood 4–10 h after AMI starts [48]. Troponin levels peak between 12 and 48 h, after which they stay high for 4 to 10 days. Sampled 6–12 h after acute chest pain onset, the sensitivity for troponin T and I detection approaches 100% [49]. In the cardiac sarcomere, actin-thin filaments bind to 92–95% of troponin, leaving 5–8% free in the myocyte cytoplasm [50]. The early releasable troponin pool' (ERTP) is composed of free, unconstrained cTn. When considering the numerous mechanisms of troponin release into the bloodstream, the ERTP concept is useful. It is believed that ERTP is released immediately after myocyte damage; furthermore, under the assumption of normal renal function, this would be promptly eliminated. In contrast, the degradation of structurally bound cTn occurs gradually and over the course of several days, resulting in a more consistent and steady release of troponin [51].

Conclusion. In conclusion, the results showed no statistical evidence of differences in age and gender between the two groups. As for the levels of interleukin before performing the therapeutic intervention, interleukin levels (IL-1β, IL-6, and IL-8) increased significantly pre-PCI compared to the control group, while IL-5 decreased significantly and this indicates the presence of inflammation, but after performing the intravenous intervention, the levels of some interleukins decreased to levels that show the absence of statistical evidence such as IL-1β, IL-5, and IL-6 in the one day after PCI compare to the control group, and IL-5, IL-6, and IL-8 in the week after PCI compare to the control group, and this indicates the important role that intravenous intervention plays in enhancing cardiovascular health and reducing the rate of inflammatory factors, and this is what was concluded in this article. The practical importance of knowing the role of PCI and its effect on reducing risk factors and inflammatory factors is an important stage in getting rid of health problems in the future. The data and results can also be used to understand the pathophysiology and important dynamics of inflammatory and cardiac factors for the purpose of developing an anti-inflammatory treatment or immunotherapy despite some difficulties, such as the polymorphic effect of interleukins.

Conflict of interest. The authors have completed the Unified Conflicts of Interest form at http://ukrbiochemjournal.org/wp-content/uploads/2018/12/coi_disclosure.pdf and declare no conflict of interest.

РІВЕНЬ МАРКЕРІВ ЗАПАЛЕННЯ У ПАЦІЄНТІВ ІЗ ІНФАРКТОМ МІОКАРДА ПІСЛЯ ЧЕРЕЗШКІРНОГО КОРОНАРНОГО ВТРУЧАННЯ

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Серцево-судинні захворювання є одними з найпоширеніших захворювань у світі, які вражають будь-який вік і іноді призводять до смерті. Атеросклероз, коронарний синдром та інфаркт міокарда зазвичай пов'язані з оклюзією

артерій і вимагають черезшкірного коронарного втручання (ЧКВ) як нехірургічної процедури для відновлення кровотоку до серця. Важливу роль у діагностиці стану пацієнтів із ураженням серця відіграють біомаркери запалення, особливо інтерлейкіни та серцеві біомаркери. Мета дослідження полягала в оцінці сироваткових рівнів інтерлейкінів і серцевих біомаркерів після ЧКВ для зниження ризику гострого коронарного синдрому. У дослідженні взяли участь 100 осіб віком від 40 до 69 років із гострим коронарним синдромом, які перенесли успішне ЧКВ, і контрольна група, що складалася з 50 здорових учасників такого ж віку. Рівні інтерлейкінів, креатинкінази МВ та міоглобіну вимірювали за допомогою імуноензимного аналізу. Рівні тропоніну та D-димеру оцінювали імуноензимним методом. Показано, що пацієнти перед ЧКВ мали значно вищі рівні IL-1β, IL-6, IL-8, серцевого тропоніну I, D-димеру, креатинкінази-МВ та міоглобіну порівняно з контрольною групою. Через добу після ЧКВ, рівні IL-6, IL-8, серцевого тропоніну I та D-димеру залишалися підвищеними. Через тиждень після ЧКВ, рівні IL-1β, IL-6, IL-8, CK-MB і міоглобіну не виявили суттєвих відмінностей, тоді як рівні серцевого тропоніну I та D-димеру залишалися підвищеними. Таким чином, у пацієнтів після ЧКВ, рівень інтерлейкінів знижується, що вказує на зменшення запальних процесів, але ураження серця певною мірою зберігається навіть через тиждень після процедури.

Ключові слова: черезшкірне коронарне втручання, інфаркт міокарда, інтерлейкіни, креатинкіназа МВ, D-димер, міоглобін.

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