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ISCHEMIA MODIFIED ALBUMIN IS A SENSITIVE MARKER OF MYOCARDIAL ISCHEMIA AFTER PERCUTANEOUS CORONARY INTERVENTION

By

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ABSTRACT

Background: Ischemia modified albumin (IMA) is a novel marker of ischemia that is produced when circulating serum albumin contacts ischemic heart tissues. IMA rises in patients who develop myocardial ischemia during percutaneous coronary intervention (PCI).

Objective: To compare blood levels of IMA in angina patients before and after elective PCI.

Patients and methods: This case-control study was conducted during the period from July 2020 to March 2021 on eighty patients with single-vessel coronary artery disease in Military Medical Center at Maadi. Forty five consecutive chronic stable angina patients undergoing PCI for management of single-vessel coronary artery disease included in group (I), and 45 Patients undergoing diagnostic angiography with coronary artery disease included in group (II) (control group).

Results: There was a statistically significant difference between the studied groups regarding IMA levels. There were statistically significant relations of IMA with TIMI level and occurrence of dissection between the studied patients. The best cut-off of IMA in prediction of TIMI II among the studied patients was ≥ 21. The best cutoff of IMA in prediction of no reflow was ≥ 26.5. Regarding the prediction of dissection, the best cutoff of IMA among the studied patients was ≥ 26.5. There were statistically significant positive correlations between IMA levels and all of diastolic blood pressure, predilation ischemic time, deployment ischemic time, total ischemic time and number of stent inserted. Only total ischemic time was significantly independently associated with IMA levels.

Conclusion: IMA is a new sensitive biomarker of PCI-induced ischemia in the PCI setting and may have a role in clinical practice.

Keywords: Coronary artery disease, percutaneous coronary intervention, myocardial ischemia, Ischemia modified albumin, cardiac biomarker.

INTRODUCTION

Percutaneous Coronary Intervention (PCI) has increased dramatically, becoming one of the most common medical interventions performed. The growth of PCIs has been remarkable and will likely be sustained, as new technologies have resulted in improved outcomes. Innovations in PCIs over the past 2 decades have been paralleled by a dramatic reduction in 30-day death,

myocardial infarction, and target-vessel revascularization rates *(Stone et al., 2011)*.

Cardiac biomarkers provide insights into variable physiopathological features such as oxidative stress, inflammation, platelet activation, and neurohormonal activity. Assessment via multi-markers assays may help to adjust treatment according to the underlying physiopathological mechanism *(Aldous, 2013)*.

The troponins are regulatory proteins found in skeletal and cardiac muscle. Three subunits have been identified: troponin I (TnI), troponin T (TnT), and troponin C (TnC). The genes that encode for the skeletal and cardiac isoforms of TnC are identical thus; no structural difference exists between them. However, the skeletal and cardiac subforms for TnI and troponin TnT are distinct, and immunoassays have been designed to differentiate between them.

According to European Society of Cardiology (ESC)/American College of Cardiology Foundation (ACCF)/American Heart Association (AHA)/ World Heart Federation (WHF) guidelines, MI refers specifically to myocardial necrosis due to myocardial ischemia. However, although elevations in the serum levels of TnI, TnT, and CK-MB indicate the presence of injury-associated necrosis of myocardial cells, such elevations do not point to the underlying mechanism of the necrosis. While myocardial necrosis occurs in MI, it can also be a product of predominantly nonischemic myocardial injury, as occurs in association with heart failure, arrhythmia, myocarditis, renal failure, pulmonary embolism, and percutaneous or surgical coronary procedures *(Thygesen et al., 2012)*.

While the benefits of High-sensitivity C-reactive Protein (Hs CRP) testing in a primary setting to screen for ischaemic heart disease is very clear, its use post-acute coronary syndrome or – myocardial infarction is less clear. CRP is elevated post-acute coronary syndrome almost exclusively in the setting of myocardial necrosis indicating the level of myocardial inflammation *(Habib et al., 2010)*.

Interestingly, the addition of N-terminal pro-brain natriuretic peptide (NT-proBNP) and CRP to The Acute Physiology and Chronic Health Evaluation II (APACHE-II) score significantly improved the ability to predict mortality in the intensive care unit *(Wang et al., 2011)*.

The aim of the present study was to compare blood levels of IMA in angina patients before and after elective PCI, and evaluate the correlation of these biomarkers to evidence of transient ischemia induced by PCI balloon inflation.

PATIENTS AND METHODS

This case-control study was conducted during the period from July 2020 to March 2021 on eighty patients with single-vessel coronary artery disease in Military Medical Center at Maadi.Written consent was obtained from family members for the participants. They were classified into:

Group (I): Forty five consecutive chronic stable angina patients undergoing PCI for management of single-vessel coronary artery disease (>70% stenosis in a major

coronary artery with no evidence of significant collateral coronary circulation around the single stenotic vessel).

Group (II) (control group): Forty five patients undergoing diagnostic angiography, with coronary artery disease.

Exclusion criteria:

Patients have other acute or chronic ischemic conditions including stroke, transient ischemic attack, claudication, peripheral vascular disease, kidney failure, shock, or objective evidence of acute myocardial infarction < 72 hours before angiography (unequivocal ECG abnormalities, serial cardiac troponin elevations, or angiographic evidence).

Methods:

All the participants were requested to sign a written informed consent regarding the procedures according to the study protocol and no harm to the patients would be allowed. Every patient record included thorough informations with special emphasis on:

- Age, gender and family history of the patient.
- Special habits (Tobacco use /Alcohol intake) - (current, former or non-smoker).
- Full clinical assessment.
- Routine laboratory data including CBC, serum creatinine, urea, RBS and liver enzymes (ALT and AST).
- Measurement of serum IMA by the albumin cobalt binding (ACB) test on a Roche Cobas MIRA PLUS instrument.
- Percutaneous coronary intervention (PCI).

Statistical analysis:

Data were entered checked and analyzed using Epi-Info version 6 and SPP for Windows version 8. Data were summarized using the arithmetic mean, standard deviation (SD); Analysis Of Variance (ANOVA and LSD), validity of screening test, student t test and Fischer's exact test were used for comparison. Correlation between variables was done using correlation coefficient “r”. Some data that are not normally distributed, were presented as median. Linear regression analysis was applied. P value was considered significant when it was < 0.05.

RESULTS

There was a statistically non-significant difference between the studied groups regarding gender. On the other hand, patients within case group were significantly older than those within control group **(Table 1)**.

Table (1): Comparison between the studied groups regarding demographic criteria

Groups / Patients	Case group N=45 (%)	Control group N=45 (%)	P
Age (year):			
Mean ± SD	62.47 ± 7.53	33.31 ± 4.87	<0.001
Range	47 – 77	24 – 41	
Gender			
Female	10 (22.2)	4 (8.9)	0.14
Male	35 (77.8)	41 (91.1)	

χ2Chi square test, t Independent sample t test

About 26.7% of patients had LAD, and 22.2% of patients had combined LAD and OM **(Table 2)**.

Table (2): Distribution of the studied patients according to culprit artery

	N=45	%
Circumflex artery	3	6.7
LAD	12	26.7
LAD-LCX	2	4.4
LAD-RCA	1	2.2
LAD, CI	1	2.2
LAD, OM	10	22.2
LCX	1	2.2
LCX-OM	2	4.4
LCX, OM	1	2.2
OM	2	4.4
OM, CI	4	8.9
RCA	5	11.1
RCA-PDA	1	2.2

There was a statistically significant difference between the studied groups regarding IMA levels which were significantly higher among case group **(Table 3)**.

Table (3): Comparison between the studied groups regarding IMA

Groups / IMA	Case group N=45 (%)	Control group N=45 (%)	P
Mean ± SD	23.51 ± 6.541	10.978 ± 0.812	<0.001
Range	14 – 38	10 – 12	

t Independent sample t test

Predilation ischemic time ranged from 15 to 200 seconds with median 35 seconds. Deployment ischemic time ranged from 30 to 160 seconds with median 60 second. Stent number ranged from 1 to 4 with median 2. Total ischemic time ranged from 30 to 260 seconds with median 90 seconds **(Table 4)**.

Table (4): Ischemic time among the studied patients

	Mean ± SD	Median	Range
Predilation ischemic time (n=35)	69.46 ± 49.96	35	15 – 200
Deployment ischemic time (n=45)	70.0± 32.96	60	30 – 160
Stent number (n=45)	2.04 ± 0.8	2	1 – 4
Total ischemic time (sec) (n=40)	114.75 ± 70.574	90	30 – 260

Five patients had no reflow, 33.3% had TIMI II while 55.6% of patients had TIMI III **(Figure 1)**.

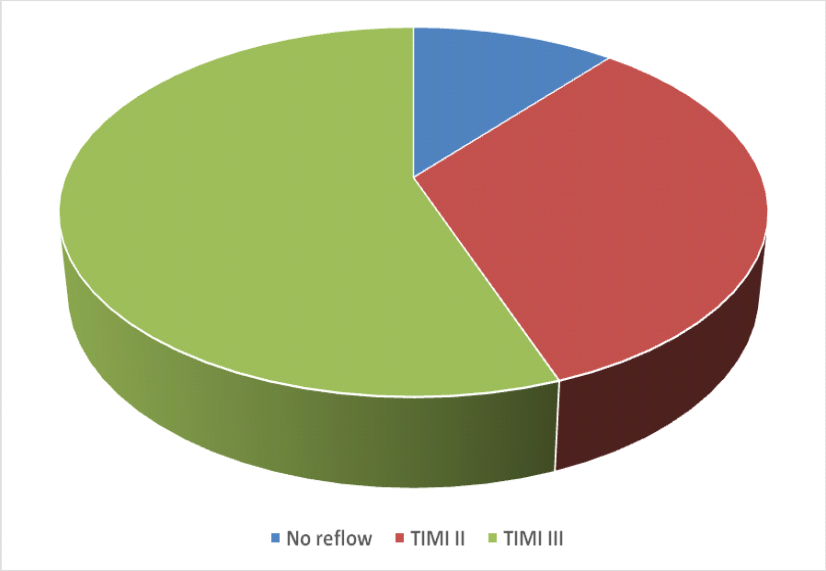

Figure (1): Pie chart showing distribution of the studied patients according to TIMI

There was a statistically significant relation between the studied patients with TIMI and IMA levels. On LSD comparison, difference was significant between each two groups **(Table 5)**.

Table (5): Relation between IMA levels and TIMI among the studied patients

TIMI \ IMA	Mean ±SD	Range	P	
No reflow (No = 5)	32.6 ± 4.04	27 – 37		$P_1$0.029
II (No = 15)	28.53 ± 4.27	19 – 38	<0.001	P_2<0.001
III (No = 25)	18.68 ± 2.79	14 – 27		P_3<0.001

F one way ANOVA test LSD Fisher least significance difference test (posthoc test)

There was a statistically significant relation between the studied patients with TIMI and IMA (significantly higher in patients with dissection) **(Table 6)**.

Table (6): Relation between IMA levels and occurrence of dissection among the studied patients

IMA / Dissection	Mean ±SD	Range	p
No	23.05 ± 6.27	14 – 38	0.025
Yes	33.5 ± 4.95	30 – 37	

t independent sample t test

The best cutoff of IMA in prediction of TIMI II among studied patients was ≥ 21 with area under curve 0.956 with sensitivity 93.3%, specificity 92%, positive predictive value 87.5%, negative predictive value 95.8% and accuracy 92.5% (p<0.001) **(Table 7** and **Figure 2)**.

Table (7): Performance of IMA in prediction of TIMI flow I and II among the studied patients

Cutoff	AUC	Sensitivity	Specificity	PPV	NPV	Accuracy	P
≥ 21.5	0.956	93.3%	92%	87.5%	95.8%	92.5%	<0.001

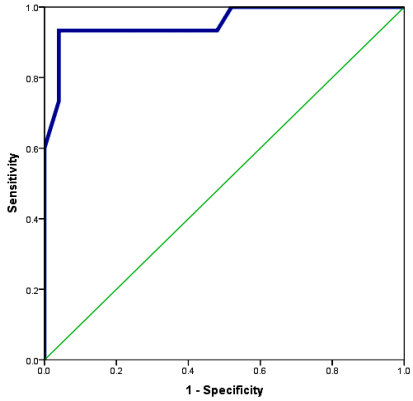

Figure (2): ROC curve showing performanceof IMA in prediction of TIMI flow I and II among the studied patients

The best cutoff of IMA in prediction of no reflow among the studied patients was ≥ 26.5 with area under curve 0.91 with sensitivity 100%, specificity 70%, positive predictive value (PPV) 29.4%, negative predictive value 100% and accuracy 73.3% (p<0.001) **(Table 8** and **Figure 3)**.

Table (8): Performance of IMA in prediction of no reflow among the studied patients

Cutoff	AUC	Sensitivity	Specificity	PPV	NPV	Accuracy	P
≥ 26.5	0.91	100%	70%	29.4%	100%	73.3%	<0.001

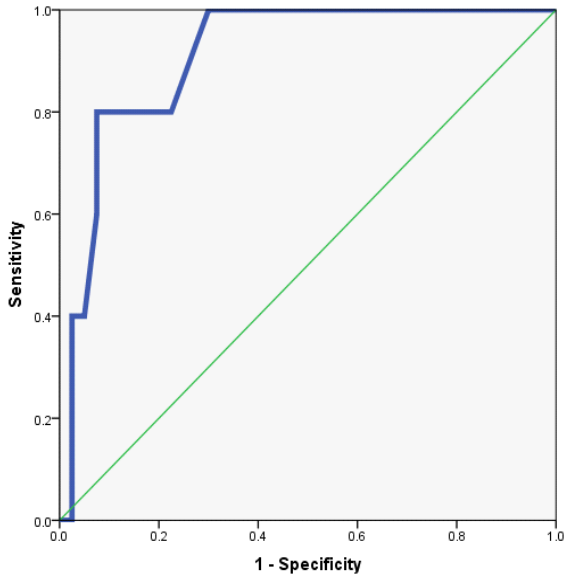

Figure (3): ROC curve showing performanceof IMA in prediction of TIMI flow I and II among the studied patients

The best cutoff of IMA in prediction of no reflow among the studied patients was ≥ 26.5 with area under curve 0.91 with sensitivity 100%, specificity 70%, positive predictive value (PPV) 29.4%, negative predictive value 100% and accuracy 73.3% (p<0.001) **(Table 9** and **Figure 4)**.

Table (9): Performance of IMA in prediction of dissection among the studied patients

Cutoff	AUC	Sensitivity	Specificity	PPV	NPV	Accuracy	P
≥ 29.5	0.907	100%	81.4%	20%	100%	82.2%	<0.001

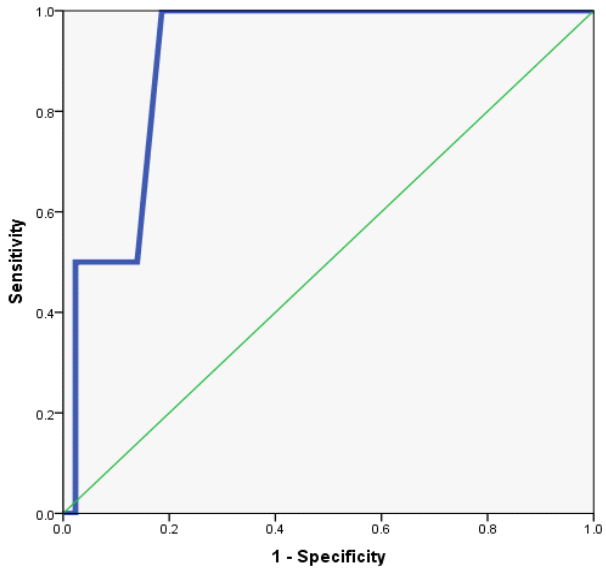

Figure (4): ROC curve showing performanceof IMA in prediction of TIMI flow I and II among the studied patients

There was a statistically significant positive correlation between IMA levels and all of diastolic blood pressure, predilatation ischemic time, deployment ischemic time, total ischemic time and number of stent inserted **(Table 10** and **Figure 5)**.

Table (10): Correlation between IMA and the studied parameters among case group

	R	P
Age	0.28	0.062
SBP	0.239	0.114
DBP	0.477	<0.001
Heart rate	-0.115	0.453
Predilation ischemic time	0.867∞	<0.001
Deployment ischemic time	0.6∞	<0.001
Total ischemic time	0.814∞	<0.001
Number of stent inserted	0.46∞	0.001

r Pearson correlation coefficient
∞Spearman rank correlation coefficient

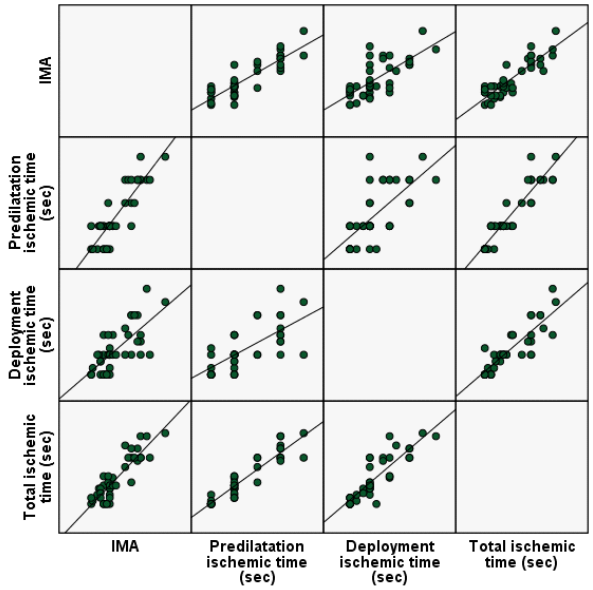

Figure (5): Scatter matrix showing significant positive correlation between ischemic time and IMA levels among case group

Among factors significantly correlated with IMA levels, only total ischemic time significantly independently associated with it (unstandardized β=0.08, 95% CI; 0.064 – 0.096, p<0.001) **(Table 11)**.

Table (11): Linear stepwise regression analysis of factors significantly associated with IMA levels among the studied patients

	Unstandardized Coefficients		Standardized Coefficients	T	p	95.0% Confidence Interval	
	β	Std. Error	B			Lower	Upper
Total ischemic time (second)	.080	.008	.881	10.178	<0.001	.064	.096

DISCUSSION

Many patients presenting to emergency department may present with myocardial ischemia without the onset of myocardial necrosis. However, use of markers for diagnosing ACS is based on the presence of myocardial necrosis as a surrogate indicator for myocardial ischemia. Diagnostic tools including ECG and CK-MB, although remains mainstay for the diagnosis of AMI, have limited role in predicting myocardial ischemia. Thus, a rapidly detectable and highly sensitive marker for myocardial ischemia would be appropriate to identify the patients in the early course of the disease, thus providing opportunity to intervene the progression of myocardial ischemia to myocardial infarction *(Maneewong et al. 2011)*. IMA was developed, later approved by United states food and drug administration, and was found to be very promising for the early detection of myocardial ischemia *(Mishra et al. 2018)*.

Among the many biomarkers, the earliest examined were the myocardial enzymes, several myocardial proteins, peptides, and many other molecules. The latest addition to the repertoire is the microRNAs, which are stable molecules detectable in circulation. About four groups are found to be involved in regulation of circulatory system, and some show promise as specific and early markers of acute coronary syndrome and cardiac dysfunction; miR-155, miR-34a, miR-126, miR-133a, miR-208b. As in other fields of medicine, personalized precise treatment may be possible with the use of microRNAs *(Jacob and Khan, 2018)*.

The mean age of case group was significantly higher than control group in the present study. Similar difference of age between the two groups was also seen in study done by Mishra et al. (2018) that showed the mean age of ACS group was significantly higher than control group. Disparity between ages in our study groups was due difficulty in finding healthy elder individuals. However, our study has shown no observed difference in IMA value between gender, and when studied in different age group.

The results of our study highlighted that serum IMA level was significantly higher in ACS group as compared to the control group after percutaneous coronary intervention, thus supporting the fact that patients with evidence of myocardial ischemia and ACS have reduced cobalt binding to HSA. Various mechanisms have been proposed for the generation of IMA. Cardiac ischemia may induce hypoxia, acidosis, increased free radical damage, membrane energy-dependent sodium and calcium pump disruptions, free iron and copper ion exposure, all of which involves damage of the amino terminal of HSA *(Patil et al., 2013)*.

There have been very little studies assessing the prognostic value of ischemia-modified albumin (IMA). *Nepal et al. (2017)* reported that people with higher IMA above 93.3U/ml showed higher short-term end points and higher 1-year mortality rate that High ischemia modified albumin (IMA) was independent predictor of both of these outcomes.

Levels of IMA were also found to be in chest pain high in another study *(Güldoğan et al., 2017)*. Few studies also recommended that instead of injury and

cellular necrosis markers, such as total CK, CK-MB and Tn-I, IMA is a marker for the early prediction of myocardial ischemia *(Chacko et al., 2017)*. However, total CK, CK-MB were not determined in our present study. Nevertheless, IMA displayed no significant difference and correlation between IMA and the cardiac markers in *(Bonorino et al., 2015)*. It was found that IMA cannot be used unaccompanied for the identification of MI because outcome may hinge on the concentration of serum albumin, which could not be detected in our patients. In addition, *Reddy et al. (2014)* demonstrated that IMA can be an early predictor of Tn-I results after 6-24 hours in patients with ACS, suggesting an association between IMA and Tn-I. To the best of our knowledge, this is the first report of its kind from this region of world highlighting that the novel biomarker has several possible utilities including the diagnosis of many conditions, differentiating IHD from non-ischemic and even prognostic value. Increased levels of IMA evidently forecasted adverse results in patients and increased the hospitalization days *(Nepal et al., 2017)*.

The release of fatty acids in myocardial ischemia results in the binding of fatty acid to albumin, inducing a conformational change in albumin, thus reducing its binding with Co (II) *(Mishra et al., 2018)*. Study has reported that IMA values during balloon angioplasty, showed correlation with the number, pressure and inflation duration, suggesting that IMA reflects to the magnitude and duration of ischemia during percutaneous intervention (PCI), and is not just a simple marker of free radical injury. While in the same study, CK-MB and Mb showed no alterations immediately after PCI.

Exploring the feasibility of IMA values when used alone and along with CK-MB and ECG, the results of IMA was superior, and additive to, when used alone or in various combinations with the conventional diagnostic tools *(Patil et al., 2013)*. The combination of IMA to CK-MB and EGC significantly increased the sensitivities of these diagnostic tools, but the sensitivity of IMA itself did not increase significantly either when used alone (92%) or in total combination with other diagnostic tools (94%), which showed discordance to other studies *(Takhshid et al, 2010)*. They reported in their study a significant increase in the sensitivity of IMA from 84% to 96% when IMA was used in combination with ECG. *Patil et al. (2013)* also reported the sensitivity and specificity of IMA as 88% and 93 %, respectively, and when combined with cTnI the IMA sensitivity increased to 96%. Thus, all these studies suggested the use of IMA in conjunction with cardiac biomarkers and ECG despite the high performance of IMA.

Similarly, *Lin et al. (2011)* compared between the means of IMA before and after percutaneous coronary intervention that showed that IMA was higher after percutaneous coronary intervention than before.

Coronary slow flow is a microvascular disorder characterized by slow progression of the opaque material during coronary angiography, without obstruction of the epicardial coronary arteries, and is an important clinical entity that can cause precordial pain at rest or during exercise *(Yılmaz et al., 2016)*.

IMA results are instrument dependent, thus, it is suggested that each facility perform an independent ROC analysis to define the optimal cut-off values of IMA assay results for their study *(Mishra et al., 2018)*. In our study, ROC curve revealed IMA cut-off of >21.4 in performance of IMA in prediction of TIMI flow I and II with area under curve 0,956 with sensitivity and specificity of 93.3% and 92% respectively. And cutoff value of >26.5 in performance of IMA in prediction of no reflow among study groups. *Mowafy et al. (2013)* reported that there was a statistical significant positive correlation between IMA levels and TIMI risk score of the study patients. Also, there was significant positive correlation between IMA levels and the extent of the coronary artery disease, defined by number of vessels affected in ischemic patients but not the severity of the disease as defined by MGS. As a predictor of mortality, IMA at a level of 9.65 ng/ml had a sensitivity of 66.6% and specificity of 88.6%. Although the mean level of IMA was higher in morbid patients (during the follow-up period) when compared with non-morbid patients, this difference was statistically insignificant.

The study was, however, bounded by its limitations. Due to the lack of "gold standard" test for myocardial ischemia, the diagnostic performance of IMA relied upon the diagnosis made by different attending physicians based on clinical findings and interpretation of the results of standard convention diagnostic tool for myocardial infarction. In addition, due to specificity issues, the study was limited by strict exclusion criteria; as a consequence, the applicability of the IMA assay in the patients presenting in the ED with ACS might be questioned.

CONCLUSION

IMA was significantly influenced by wide range of physiological variables, including exercise and hydration. It may also be elevated in a number of other diseases. It is a sensitive marker of PCI-induced ischemia in the PCI setting, and may have a role in clinical practice.

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الزلال المعدل نتيجة إفتقار الدم كدلالة دقيقة على تروية عضلة القلب بعد قسطرة القلب العلاجية

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خلفيـــة البحـــث: الآلبـــومين المعـــدل بـــنقص التروية هـــو علامـــة جديـــدة لـــنقص التروية يـــتم انتاجـــه عنـــدما يـــتلامس ألبـــومين مـــع أنســـجة القلـــب الاقفاريـــة ويرتفـــع فـــى المرضـــى الـــذين يصـــابون بـــنقص تروية عضـــلة القلـــب أثنـــاء التـــدخل التـــاجى عن طريق الجلد.

الهـــدف مـــن البحـــث: مقارنـــة مســـتويات الـــدم مـــن الآلبـــومين المعـــدل بـــنقص التروية فـــى مرضـــى الذبحـــة الصـــدرية قبـــل وبعـــد التـــدخل التـــاجى الاختيـــارى عـــن طريـــق الجلد.

المرضـــى و طـــرق البحـــث: اجريـــت دراســـة الحـــالات والشـــواهد فـــى الفتـــرة مـــن يوليـــو 2020 إلـــى مـــارس 2021 علـــى ثمـــانين مريضـــا يعـــانون مـــن مـــرض الشـــريان التـــاجى أحـــادى الاوعيـــة فـــى المركـــز الطبـــى العســـكرى بالمعـــادى: 45 مريضـــا متتاليـــا مـــن الذبحـــة الصـــدرية المســـتقرة المزمنـــة خضـــعوا لعمليـــة فـــى الشـــريان التـــاجى لادارة مـــرض الشـــريان التـــاجى أحـــادى الوعـــاء فـــى المجموعـــة (1)، وخضـــع 45 مريضـــا لتصـــوير الاوعيـــة التشخيصـــى ، مـــع مـــرض الشـــريان التاجى فى المجموعة (ll) (مجموعة التحكم).

نتـــائج البحـــث: توجـــد فـــروق ذات دلالـــة أحصـــائية بـــين المجموعـــات المدروســـة فيمـــا يتعلـــق بمســـتويات الآلبـــومين المعـــدل بـــنقص التروية. وقـــد وجـــدت علاقـــات دالـــة إحصـــائيا للآلبـــومين المعـــدل بـــنقص التروية مـــع مســـتوى تخثـــرب الـــدم فـــي احتشـــاء عضـــلة القلـــب وحـــدوث تشـــريح بـــين المرضـــى الخاضـــعين للدراســـة.

وكان أفضل حد قاطع للآلبومين المعدل بنقص التروية فى التنبؤ ب تخثر الدم في احتشاء عضلة القلب بين المرضى الخاضعين للدراسة هو ≤ 21. لكن كان أفضل حد للآلبومين المعدل بنقص التروية فى التنبؤ بعدم اعادة التدفق هو 26.5. فيما يتعلق بالتنبؤ بالتسلخ، فقد كان افضل حد لقطع الآلبومين المعدل بنقص التروية بين المرضى الخاضعين للدراسة هو ≤26.5. وهناك ارتباطات ايجابية ذات ايجابية ذات دلالة أحصائية بين مستويات الآلبومين المعدل بنقص التروية وجميع ضغط الدم الانبساطى، والوقت الاقفارى للميول، والوقت للنشر، والوقت الاقفارى الاجمالى وعدد الدعامة المدخلة. فوكان اجمالى الوقت الاقفارى مرتبطا بشكل مستقل بمستويات الآلبومين المعدل بنقص التروية.

الاستنتاج: الآلبومين المعدل بنقص التروية هو علامة بيولوجية حساسة جديدة لنقص التروية الناجم عن التدخل التاجى عن طريق الجلد فى اعداد التدخل التاجى الاختيارى عن طريق الجلد، وقد يكون له دور فى الممارسة السريرية.

الكلمات الدالة: مرض الشريان التاجى، التدخل التاجى عن طريق الجلد، نقص تروية عضلة القلب، نقص التروية الزلال المعدل، المؤشرات الحيوية للقلب.