Diagnostic Value of Ischemia-Modified Albumin in Terms of Acute Coronary Syndromes in Chronic Renal Patients Admitted to Emergency Department with Chest Pain and/or Equivalent Symptoms

Göğüs Ağrısı ve/veya Anjinal Eşdeğer Semptomlarla Acil Servise Başvuran Kronik Böbrek Hastalarında İskemi-Modifiye Albüminin Akut Koroner Sendromlar Açısından Tanısal Değeri

İrfan Gökçek[1], Bedriye Müge Sönmez[2], Çiğdem Yücel[3], Mebrure Beyza Gökçek[4], Fatih Alper Ayyıldız[5]

[1] University of Health Science Kayseri City Education and Research Hospital, Emergency Medicine Specialist, Kayseri, Turkey

[2] University of Health Science Dışkapı Yıldırım Beyazıt Education and Research Hospital, Emergency Medicine Specialist,Ankara, Turkey

[3] University of Health Science Gülhane Education and Research Hospital, Medical Biochemist Ankara, Turkey

[4] Kayseri Provincial Health Directorate, Family Physician Specialist, Kayseri, Turkey

[5] University of Health Science Eskişehir City Education and Research Hospital, Emergency Medicine Specialist, Eskişehir, Turkey

ABSTRACT

Introduction: This study aimed to reveal the diagnostic value of Ischemia-Modified Albumin (IMA) in terms of acute coronary syndromes in chronic renal patients admitted to emergency department with chest pain and equivalent symptoms.

Methods: Our study is a descriptive cross-sectional research and included 125 chronic renal patients admitted to emergency department with chest pain and/or equivalent symptoms and 96 healthy volunteers as the control group.

Results: In our study, acute coronary syndrome (ACS) was detected in 12 (9.6%) of the patients. Mean IMA level was 0.72±0.17 absorbance units (ABSU) in the group with Chronic renal disease (CRD)+ACS, 0.71±0.16 ABSU in patients with CRD but without ACS, and 0.47±0.14 ABSU in the control group. A statistically significant difference was found between the groups. While no difference between chronic renal patients in whom ACS developed and chronic renal patients in whom ACS did not develop was recorded in terms of IMA. IMA value of both patients with CRD and chronic renal patients in whom ACS developed was significantly higher than that of the healthy control group. In our study, no correlation was found between troponin levels and IMA.

Conclusion: IMA level increases in both CRD and ACS. Therefore, it was determined that IMA level did not significantly increase although ACS developed depending on already high IMA level in patients with CRD. We believe IMA cannot be safely used for the diagnosis of ACS in patients with CRD due to the low specificity rate and absence of an optimal threshold value.

Key words: Ischemia Modified Albumin, Acute Coronary Syndrome, Chronic Renal Disease

ÖZET

Giriş: Bu çalışmanın amacı; göğüs ağrısı ve eşdeğer semptomlar ile acil servise başvuran kronik böbrek hastalarında İMA'nın akut koroner sendromlar açısından tanısal değerliliğini ortaya koymaktır.

Yöntemler: Çalışmamız tanımlayıcı kesitsel tipte bir araştırma olup; göğüs ağrısı ve/veya eşdeğer semptomlar ile acil servise başvuran 125 kronik böbrek hastası ve kontrol grubu olarak da 96 sağlıklı gönüllü çalışmaya dahil edilmiştir.

Bulgular: Çalışmamızda hastaların 12'sinde (%9,6) AKS saptandı. Çalışmamızda KBH+AKS'li grubun İMA düzeyi ortalaması 0,72±0,17 ABSUs, KBH'li olup AKS'si olmayan hastaların İMA düzeyi ortalaması 0,71±0,16 ABSUs ve kontrol grubunun İMA düzeyi ortalaması 0,47±0,14 ABSUs olduğu saptandı. Gruplar arasında istatistiksel olarak anlamlı farklılık saptandı (p<0,05). AKS gelişen kronik böbrek hastaları ve AKS gelişmeyen kronik böbrek hastaları arasında İMA açısından fark saptanmazken (p: 0,859); hem KBH'li hastaların hem de AKS gelişen kronik böbrek hastalarının İMA değeri sağlıklı kontrol grubuna göre anlamlı olarak yüksekti (p<0,05). Çalışmamızda troponin düzeyleri ve İMA arasında korelasyona rastlanmadı (p>0,05). KBH'li hastalarda İMA için EAA 0,877 cm2 pozitif kabul edildiği 0,05 ABSUs, değerinde sensitivite %61,5 ve spesifite % 89,6; AKS'li hastalarda İMA için EAA 0,876 cm2 pozitif kabul edildiği 0,05 ABSUs, değerinde sensitivite %83,3 ve spesifite % 61,5; KBH'li hastalarda AKS gelişimindeki İMA için EAA 0,524 cm2 pozitif kabul edildiği 0,05 ABSUs, değerinde sensitivite %83,3 ve spesifite % 9,7 olarak saptandı.

Sonuç: Gerek KBH'de gerekse AKS'de İMA düzeyi artmaktadır. Bu nedenle; KBH'li olgularda İMA'nın halihazırda yüksek olmasına bağlı olarak, AKS gelişmiş olsa dahi İMA düzeyinin anlamlı olarak yükselmediği belirlendi. Gerek spesifitenin düşük olması, gerek optimal eşik değerin bulunamamış olması nedeni ile KBH'li hastalarda; İMA'nın AKS tanısı için güvenli olarak kullanılamayacağı kanısındayız.

Anahtar Kelimeler: İskemi-modifiye albümin, akut koroner sendrom, kronik böbrek hastalığı

Corresponding author: İrfan Gökçek, University of Health Science Kayseri City Education and Research Hospital, Kayseri, Türkiye
E-mail: irfangokcek@yahoo.com
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Authors: İrfan Gökçek (ORCID: 0000-0002-5612-8527), Bedriye Müge Sönmez (ORCID: 0000-0003-3970-8922), Çiğdem Yücel (ORCID: 0000-0003-2647-440X), Mebrure Beyza Gökçek, (ORCID: 0000-0002-6320-4102), Fatih Alper Ayyıldız (ORCID: 0000-0002-2502-1030)

INTRODUCTION

Coronary artery disease and acute coronary syndromes are the most common causes of death during adulthood. Electrocardiography (ECG), physical examination, serum biomarkers and imaging methods are used to diagnose acute coronary syndrome in patients admitted to the emergency department with chest pain and/or equivalent symptoms (1). ST elevation monitored on 12-lead ECG during admission is gold standard (2). However, ECG is not diagnostic in all patients and serum markers are of great importance for risk assessment. Therefore, high troponin I or T values are diagnostic for acute myocardial infarction (AMI). Increase in troponin I and T is important for detection of necrosis that occurs in myocardial cells and their sensitivity is quite high (3,4). But false positivity may occur in troponin values in the presence of comorbid pathologies, which may be confusing for the diagnosis of acute coronary syndrome (3).

Chronic renal disease (CRD) is one of the most hazardous comorbid conditions for AMI. The leading cause of death in these patients is cardiac pathologies especially due to coronary artery disease (5). In studies, abnormalities in electrocardiography (ECG) due to CRD, false cardiac biomarker positivity and cardiac biomarker positivities due to reasons other than AMI have been revealed (6).

Ischemia-modified albumin (IMA) has recently been defined and is a type of albumin protein increasing in cases of ischemia developing in the body. This biochemical marker has been analyzed in many diseases that occur with ischemia, particularly in coronary ischemia (7,8). This study aimed to reveal the diagnostic value of IMA and whether it could be used as an exclusion criterion in terms of acute coronary syndromes in chronic renal patients through comparing it with troponin.

METHODS

For this study, ethics committee approval was obtained from the ethics committee of Ankara Numune Training and Research Hospital, dated 12.07.2017 and numbered E-Kurul-E-17-1462.

The study was prospectively performed with 125 CRD patients admitted to the Emergency Department of Ankara Numune Training and Research Hospital with chest pain or equivalent symptoms within 5 months and 96 healthy controls after it was approved by the local ethics committee. The people in the healthy group had no chronic diseases. There was no smoking or substance use.

After the consents of 125 CRD patients admitted with typical chest pain or equivalent symptoms were obtained blood samples were collected to measure IMA level. Collected blood samples were centrifuged and their serums were separated. Then, adequate amount of serum sample separated for biochemical analysis was stored at -80^{o}C in the refrigerator until the analysis process.

Normal reference intervals for albumin value were accepted between 3.5-5.5 gr/dl. Ischemia-modified albumin was measured with colorimetric method developed by Bar-Or et al. (9). A total of 50 µl from 1 g/l cobalt chloride solution was added into the patient serum of 200 µl, mixed and incubated at room temperature for 10 minutes. Then, 50 µl from 1.5 g/l DTT solution was added, mixed and incubated at room temperature for 2 minutes. After that, 1 ml from 9.0 g/l NaCl solution was added. Sample blanks were similarly prepared without adding DTT. Absorbance of test mixtures was measured at 470 nm. The results were reported in absorbance units (ABSU). Normal reference intervals were accepted between 0-0.5 ABSU.

Patients who refused to give consent, patients under the age of 18, patients with liver pathology, patients with pathology related to albumin synthesis or that

Table 1. Demographic features of ACS in patients with CRD

			Acute Coronary Syndrome		p
		All patient group Mean±SD/n(%)	**Yes Mean±SD/n(%)**	**No Mean±SD/n(%)**	
Age		72.7±12.4	71.3±10.4	72.9±1.0	0.509*
Gender	**Male**	58 (46.4)	7 (58.3)	51 (45.1)	0.383**
	Female	67 (53.6)	5 (41.7)	62 (54.9)	
CRD Stage	**Stage 1**	2 (1.6)	0	2(1.8)	0.859*
	Stage 2	10 (8.0)	2(16.7)	8 (7.1)	0.662**
	Stage 3	52 (41.6)	6 (50)	46(40.7)	
	Stage 4	35 (28.0)	2(16.7)	33 (29.2)	
	Stage 5	26 (20.8)	2(16.7)	24 (21.2)	
DM		68 (54.4)	8 (66.7)	60 (53.1)	0.544**
HT		46 (36.8)	4 (33.3)	42 (37.2)	0.793**
CAD		25 (20)	3 (25)	22 (19.5)	0.649**
Troponin 1			297.2±30.7	66.1± 6.5	<0.001*
Troponin 2			465.8±49.8	465.8±49.8	<0.001*
Troponin 3			831.9±79.9	831.9±79.9	<0.001*

**Mann Whitney U test, **Chi-square test. Abb. CRD: Chronic renal disease, CAD: Coronary arter disease, DM: Diabetes mellitus, HT: Hypertansion.*

would cause excessive consumption of albumin, patients with active ischemia or another ischemic occurrence in their medical history, patients who were considered to have another ischemic occurrence in their preliminary diagnosis and patients with acute infection were excluded from the study.

Statistical Analysis

The data were analysed with Windows 22 version of SPSS software program. Distribution of the variables was assessed with Kolmogorov Smirnov test. Quantitative data were expressed as mean and standard deviation while the number of patients (n) and percentile were used for the expression of qualitative data. Student-t test and ANOVA test were used in the analysis of numerical parametric data and Mann Whitney U and Kruskal Wallis tests were used in the analysis of numerical nonparametric data. Chi-square test was used in the analysis of qualitative data. Spearman's Correlation test was used in comparison of quantitative data. ROC curve was used in calculating the area under the curve (AUC), cut-off values and specificity and sensitivity rates of the data. $p<0.05$ was accepted as the statistically significant value.

RESULTS

A total of 221 people were included in the study. Of these, 96 were healthy people. The mean age was 66±8.2 years. %43.7 percent were male. 125 people had chronic kidney disease. The mean age of this group was 72.7±12.4 years and no relationship between age and ACS development was found in patients with CRD ($p>0.05$). Of the patients with CRD, 58 (46.4%) were male. Of the patients included in the study, 2 (1.6%) were Stage I, 10 (8%) were Stage II, 52 (41.6%) were Stage III, 35 (28%) were Stage IV and 26 (20.8%) were Stage V. No relationship was found between CRD stage and incidence of ACS

development (p>0.05). In our study, 68 (54.4%) patients had DM, 46 (36.8%) had HT and 25 (20%) had CAD as comorbid diseases. No statistically significant relationship was found between the incidence of comorbid diseases and incidence of ACS development (p>0.05). Twelve (9.6%) of the patients with CRD in our study were diagnosed with ACS and their troponin levels were significantly high (p<0.05) (Table 1).

No correlation was found between age and IMA level in all patient group (p>0.05) (Table 2).

Table 2. Relationship between age and IMA level

	Age	
	R	p*
IMA	-0.119	0.188

**Spearman's correlation test. Abb.IMA: İschemia-modified albumin)*

Mean IMA level of male patients in our study was 0.71±0.15 ABSU and mean IMA level of female patients was 0.71±0.17 ABSU. No difference was found between the groups in terms of IMA level (p>0.05) (Table 3).

Table 3. Relationship between gender and IMA

	Male (n:58) Mean±SD	Female (n:67) Mean±SD	p*
IMA	0.71±0.15	0.71±0.17	0.914

**Student t test test. Abb. IMA: Ischemia-modified albumin*

In our study, mean IMA level was 0.65±0.08 ABSU in Stage I patients, 0.73±0.16 ABSU in Stage II patients, 0.71±0.13 in Stage III patients, 0.73±0.20 ABSU in Stage IV patients and 0.69±0.17 ABSU in Stage V patients. No difference was found in terms of CRD stage and IMA level (p>0.05) (Table 4).

Table 4. Relationship between CRD stage and IMA

CRD stage	n(%)	Mean±SD	p*
Stage I	2 (1.6)	0.65±0.08	0.877
Stage II	10 (8.0)	0.73±0.16	
Stage III	52 (41.6)	0.71±0.13	
Stage IV	35 (28.0)	0.73±0.20	
Stage V	26 (20.8)	0.69±0.17	

**ANOVA test. Abb.CRD: Chronic renal disease.*

In our study, mean albumin level was 3.9±0.5 gr/dl in the group with ACS, 3.7±0.5 gr/dl in the group with CRD but without ACS and 4.4±0.4 gr/dl in the control group. A statistically significant difference was found between the groups (p<0.05). While no difference was found between patients in whom ACS developed and patients in whom ACS did not develop in the patient group in terms of albumin (p:0.065) albumin level was significantly lower in both patients with CRD and patients with CRD in whom ACS developed compared to the control group (p<0.05) (Figure 1).

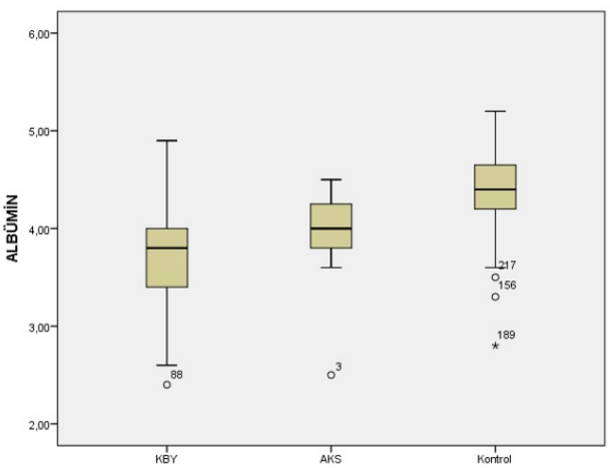

Figure 1. Comparison of albumin level between the groups (Kruskal Wallis test was used in comparison of all groups and Mann Whitney U test was used in two-group comparisons)

In our study, mean IMA level was 0.72±0.17 ABSU in patient group with CRD in whom ACS developed, 0.71±0.16 ABSU in patient group with CRD but without ACS and 0.47±0.14 ABSU in the control group. A statistically significant difference was found between the groups (p<0.05). While no difference was found between patients in whom ACS developed and patients in whom ACS did not develop in the patient group in terms of IMA level (p:0.859) IMA value was significantly higher in both patients with CRD and patients with CRD in whom ACS developed compared to the control group (p<0.05) (Fig. 2).

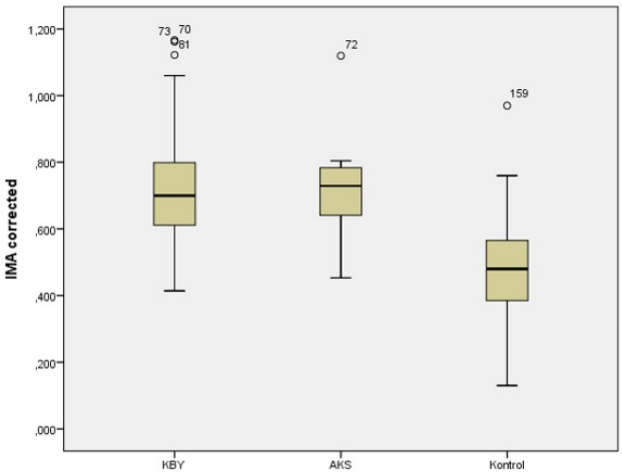

Figure 2. Comparison of IMA levels among the groups (Comparison of all groups was performed with ANOVA test and two-group comparisons were performed with Student t test.)

No correlation was found between Troponin and IMA levels in our study (p>0.05) (Table 5).

Table 5. Comparison between Troponin and IMA levels

		All patient group		Acute Coronary Syndrome	
		r	p*	r	p*
Troponin-1		-0.165	0.065	-0.280	0.379
	N	113		12	
Troponin-2		-0.084	0.392	-0.343	0.276
	N	93		12	
Troponin-3		0.122	0.454	-0.183	0.637
	N	31		9	

**Spearman's correlation test. Abb. IMA: Ischemia-modified albumin*

No relationship was found between history of DM, HT and CAD and IMA levels in our study (p>0.05) (Table 6).

Table 6. Comparison of IMA level with the presence of a comorbid disease

	Acute Coronary Syndrome		p*
	Yes	No	
DM	0.70±0.17	0.72±0.16	0.600
HT	0.70±0.18	0.72±0.15	0.687
CAD	0.73±0.23	0.71±0.14	0.416

**Student t test. Abb. CAD: Coronary arter disease, DM: Diabetes mellitus, HT: Hypertansion,.*

At ABSU value of 0.05 in which AUC value of 0.877 cm^2 was accepted as positive for IMA in patients with CRD, sensitivity rate was 61.5% and specificity rate was 89.6%. At ABSU value of 0.05 in which AUC value of 0.876 cm^2 was accepted as positive for IMA in patients with ACS, sensitivity rate was 83.3% and specificity rate was 61.5%. At ABSU value of 0.05 in which AUC value of 0.524 cm^2 was accepted as positive for IMA in the development of ACS in patients with CRD, sensitivity rate was 83.3% and specificity rate was 9.7% (Fig. 3).

DISCUSSION

End-stage renal failure which is the last stage of CRD is one of the most hazardous comorbidities for AMI. The leading cause of death in these patients is cardiac pathologies especially due to coronary artery disease (5). Although patients with CRD have no ACS their high troponin values accompanied by symptoms such as malaise, fatigue or shortness of breath cause physicians to be confused and lead to late diagnosis. There may be difficulties in interpreting ECG as a result of cardiac hypertrophies caused by increasing comorbidities due to CRD in these patients, ECG abnormalities due to developing electrolyte disturbances, conduction defects and used drugs.

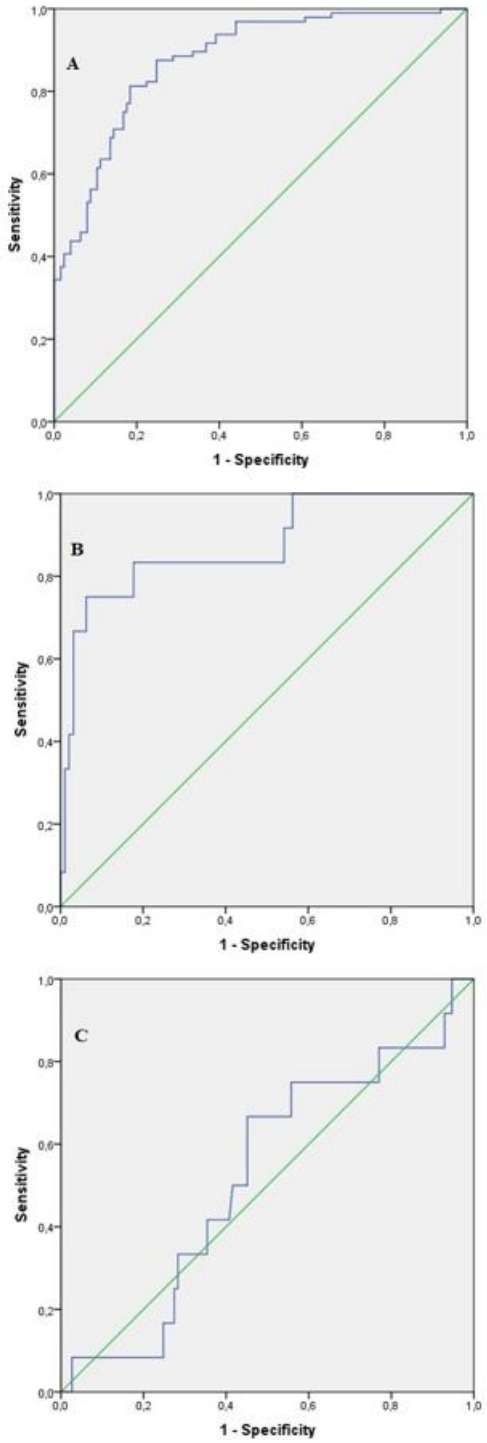

Figure 3. ROC curves of the groups (A: relationship between the group with chronic renal disease(CRD) and control group in terms of ischemia-modified albumin(IMA) level, B: diagnosticity of IMA in the development of acute coronary syndrome(ACS) in patients with CRD)

Cardiac biomarkers in these patients may also give false positive results. Therefore, a cardiac marker with both high sensitivity and specificity for ACS is crucial in patients with chronic renal disease.

High troponin values in patients with CRD even if ACS is not present, leaves the physician in dilemma with symptoms such as weakness, fatigue and shortness of breath in these patients and causes a delay in diagnosis. In this study, we evaluated whether we can use IMA values as exclusion criteria in ACS patients with CRD.

Studies have revealed that age and gender do not change IMA (10,11,12). In our study, our patient group was at seventh decade and 46.4% were male and no relationship was found between IMA and age and gender. We think IMA is not affected by demographic factors such as age and gender as it increases and fails over time as a result of ischemia.

Elevation in IMA concentrations is used as an early marker revealing myocardial ischemia in the evaluation of patients with ACS (9,13). A study performed by Roy et al., high IMA values in patients who had retrosternal pain with normal ECG and troponin findings revealed that IMA was an independent marker for ACS (14). In a study performed by Yaylalı, 64% of the patients admitted with chest pain were diagnosed with ACS and IMA values of patients with ACS were higher (15). A study performed by Çevik et al. reported that IMA did not make a significant contribution in patients whose AMI diagnosis was confirmed (16). In our study, IMA level was significantly higher in patients with ACS compared to the control group.

In a study performed by Yaylalı, IMA and troponin levels were compared in patients with chest pain independent from ACS and false positivity rate of IMA was found high (15). In a study performed by Sharma et al., patients with high IMA levels similarly had high troponin levels (17). A study performed on patients diagnosed with ACS by Guven reported that IMA had

no superiority on cardiac markers in differentiating ACS patients from the control group. However, they stated that the exclusion rate of troponin and IMA combination in patients with ACS was higher (18). In our study, troponin value was high in AMI patients and no correlation was found between IMA and troponin. In several studies, although IMA values are considered to be used for early diagnosis and treatment in patients with ACS as they increase in ischemic conditions this issue is still controversial. Abnormality in albumin metabolism due to CRD in all patients in the patient group with CRD and low number of patients with ACS may have caused the correlation between IMA and troponin to be low. On the other hand, while IMA became positive even in ischemia its positivity in troponin necrosis may have caused this relationship to be disrupted.

In a study performed by Turedi et al., IMA levels both before dialysis and after dialysis in dialysis-dependent patients were found higher in end-stage renal patients who underwent hemodialysis compared to the healthy individuals (19). In our study, IMA level of patients with CRD was high. No relationship was found between IMA level and CRD stage. Protein losses developing due to CRD, CRD's effects that lead to ischemia in other systems such as hematological disorders in particular and kidney damage resulting in ischemia may have caused IMA level to increase. We believe therapies the individuals receive in different stages, replacements (blood, albumin) and other pathologies cause the relationship between CRD stage and IMA to become insignificant.

Cardiovascular disease is the most important cause of morbidity and mortality in patients with CRD (20). A study in which myocardial ischemia was evaluated in patients with ESRD, dobutamine stress echocardiography and IMA levels were assessed in the patient and IMA level of the group with positive dobutamine stress echocardiography was found significantly high and it was concluded that IMA was a positive marker showing myocardial ischemia in patients with ESRD (21). In our study, IMA level of CRD patients in whom ACS developed and did not develop was similar. We believe IMA level is not high in presence of ACS because CRD leads to different levels of ischemia depending on atherosclerosis in the whole body particularly coronaries in patients and causes nutritional problems in tissue level such as anemia, there are stable/unstable angina patients who are not diagnosed among the patients and most importantly, CRD itself is an ischemic process. Moreover, we think abnormality in albumin metabolism caused by CRD also plays a role in the process.

Albumin is a protein with a half-life of 19-20 days, plays a protective role with some different mechanisms against the development of cardiovascular disease and is a negative acute phase reactant (22). Studies state that albumin values decrease in CRD and ACS (23,24). In our study, albumin value was lower in patients with CRD and patients with ACS+CRD compared to the control group, however, albumin value in patients with CRD and ACS+CRD was similar. This may be due to protein loss developing in urine during chronic process in patients with CRD and protein leak in dialysis-dependent patients. In addition, albumin is a negative acute phase reactant, which may have caused it to decrease both in ACS and CRD.

It is known that DM causes an increase in ACS because it is more commonly the cause of atherosclerosis (25). A study performed by Piwowar et el. reported that presence of diabetes increased IMA level (26). In a study performed by Kiraz, patients with DM were evaluated and the results revealed that IMA level was higher in patients with CRD (27). In a multicenter study in our country, the relationship between newly diagnosed HTs and IMA was assessed and no difference was found (28). In our study, the most common comorbidity was DM followed by HT

whether with ACS or not and no relationship was found between comorbidities and IMA. The absence of a relationship between comorbidities and IMA may be because all the patients had CRD and these patients whether with a comorbidity or not may have high IMA or because these diseases had no relationship with ischemia in acute period.

IMA often gave false negative results and its specificity was low in the PRIMA study (29). In the study performed by Yaylalı, when cut-off value for IMA was 0.4482 in ACS patients with normal Troponin I value sensitivity of IMA value was 67.2% and its specificity was 41.0% during admission (15). In a study performed on patients with ACS by Sharma et al., sensitivity of serum IMA level test was 83% and specificity was 69% (17). In our study, sensitivity of IMA was 61.5% and specificity was 83.3% at the cut-off value of 0.05 while its sensitivity was 83.3% and specificity was 9.7% in the development of ACS in patients with CRD. This suggests that IMA at high sensitivity can be diagnostic in ACS and CRD, however, its high sensitivity but low specificity in patients with CRD gives the impression that although IMA helps the diagnosis of ACS its value is low.

Limitations

The low number of patients with ACS in our study is the most important cause of limitation. Not knowing the level of microvascular and macrovascular effects caused by the patients' comorbidities; may have affected the relationship of comorbidities with İMA Since it has been reported in some studies that dialysis affects the IMA level, the limitation of our study is that patients with CRD were not recorded in the dialysis program and this relationship could not be evaluated.

As we mentioned earlier in our study, while IMA increases significantly in the blood at earlier periods compared to troponin, it remains much shorter than troponin. Another limitation in our study; serum IMA level in CRD patients with ACS may not be significantly higher than CRD patients who do not develop ACS because of the prolongation of the time between the onset of chest pain or equivalent symptoms and the time of admission to the hospital. Another limitation is that all patients with CRD in the patient group have comorbid conditions.

CONCLUSION

In conclusion, IMA level increases in both CRD and ACS. Therefore, it was determined that IMA level did not significantly increase although ACS developed depending on already high IMA level in patients with CRD. We believe IMA cannot be safely used for the diagnosis of ACS in patients with CRD due to low specificity rate and absence of an optimal threshold value.

Informed Consent: Informed consent was obtained from patients who participated in this study.

Conflict of Interest: Authors declared no conflict of interest.

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Ethical Approval: For this study, ethics committee approval was obtained from the ethics committee of Ankara Numune Training and Research Hospital, dated 12.07.2017 and numbered E-Kurul-E-17-1462.

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