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Predictive Value of Serum Albumin in Patients with Acute Coronary Syndrome

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Abstract:

Background: In patients with acute coronary syndrome (ACS), the presence of hypoalbuminemia has been associated with increased severity of coronary lesions, no-reflow, increased in-hospital, and long-term mortality as well as development of heart failure. Our study aimed to investigate the predictive value of serum albumin and short-term mortality in patients with ACS.

Methods: This prospective cohort study was carried out on patients who were admitted to the emergency department or coronary care unit with a diagnosis of ACS.

Results: Serum albumin can significantly predict mortality at cut-off 3.34 with 71.4% sensitivity, 62% specificity, 33.3% positive predictive value (PPV), 89.1% negative predictive value (NPV) and 64% accuracy (P value= 0.006). Serum albumin can significantly predict the mortality at cut-off 3.44 with 76.2% sensitivity, 57% specificity, 32% PPV, 90% NPV and 61% accuracy (P value= 0.007).

Conclusions: Serum albumin at cut off 3.34 is a promising predictor of short-term mortality in ACS patients. These support the potential utility of serum albumin as a simple, cost-effective biomarker for identifying patients at higher risk of mortality following an ACS event. Furthermore, the study found that patients with hypoalbuminemia had significantly higher levels of creatinine and lower levels of high-density lipoprotein cholesterol, hemoglobin, and peak troponin I compared to the patients with normal serum albumin.

Keywords : Serum Albumin, Acute Coronary Syndrome, Thrombolysis in Myocardial Infarction, Left Ventricular Ejection Fraction

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Introduction:

Acute coronary syndrome (ACS) refers to a group of conditions that include ST-elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), and unstable angina. It is a type of coronary heart disease (CHD), which is responsible for one-third of total deaths in people older than 35. Some forms of CHD can be asymptomatic, but ACS is always symptomatic [1].

Long-term mortality remains high in patients presenting with ACS despite advances in diagnosis and treatment. Therefore, identification of high-risk patients in this patient group is of prime importance. To date, various risk scores and biomarkers have been used for this purpose [2].

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Albumin is the major protein in human plasma and the most abundant protein in the extracellular compartment. Albumin has multiple important physiological functions including maintenance of plasma osmotic pressure and capillary permeability, a ligand for many endogenous and exogenous compounds and it also affects the pharmacokinetics of several drugs [3].

In addition, low serum albumin is associated with increased adverse clinical outcomes and prognosis in cancer, heart failure (HF), stroke, and stable coronary artery disease (CAD). Similarly, in patients with ACS, the presence of hypoalbuminemia has been associated with increased severity of coronary lesions, no-reflow, increased in-hospital, and long-term mortality as well as development of HF [4, 5].

Until now, several studies have documented the prognostic value of SA in patients with stable CAD, STEMI and pulmonary embolism. In this research, the authors also concluded that admission SA level was a strong and independent predictor of all-cause long-term mortality in patients with ACS. Although the research was interesting and well-conducted, we have some comments [6].

Current European and North American guidelines recommend risk stratification of ACS cases using available risk scores, such as the thrombolysis in myocardial infarction score and the global registry of acute coronary events score, to determine the in-hospital and 6-months risk of death and myocardial infarction in patients with ACS following an index event [7].

Besides that, a recent study revealed that these scores had an adequate predictive power for long-term mortality in patients who were diagnosed and hospitalized with ACS [8].

Our study aimed to investigate the predictive value of serum albumin and short-term mortality in patients with ACS.

Patients and Methods:

This prospective cohort study was carried out on patients who were admitted to the emergency department or coronary care unit with a diagnosis of ACS old aged ≥18 years, both sexes.

The study was done after approval by the Institutional Review Board of Sohag University, Sohag, Egypt. An informed written consent was obtained from the patient or their relatives.

The exclusion criteria were patients with a history of chronic liver disease, nephrotic syndrome, or other conditions known to affect serum albumin levels, patients receiving albumin infusions or other treatments that could potentially interfere with serum albumin

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levels, patients with incomplete medical records or missing laboratory data and patients who declined to participate in the study.

All patients were subjected to complete history taking, clinical presentation, laboratory findings [serum albumin level, cardiac enzymes and inflammatory markers] and imaging results (coronary angiography and echocardiography).

A diagnosis of unstable angina pectoris (UAP)/NSTEMI will be made based on electrocardiographic prominent T-wave inversion or ST-segment depression and/or positive biomarkers of necrosis (e.g., troponin, creatine kinase-MB in the absence of ST segment elevation and in an appropriate clinical setting [anginal equivalent or chest discomfort]).

Mainstay drug treatment for UAP/NSTEMI will include antiplatelet agents, b-blockers, angiotensin converting enzyme inhibitors/angiotensin receptor blockers, and statins. Hypertension (HTN) was defined as previous use of antihypertensive medications, a systolic blood pressure of >140 mm Hg, or a diastolic blood pressure of >90 mm Hg on at least 2 separate measurements.

Diabetes mellitus (DM) was defined as preexisting diagnosis of DM with established antidiabetic diet/treatment or a fasting venous blood glucose level of 126 mg/dL on 2 occasions in previously untreated patients.

Hypercholesterolemia was defined as a total cholesterol level of 200 mg/dL.

Current smoking was defined as regular consumption of cigarettes.

Severe HF was defined as a left ventricular ejection fraction (LVEF)<20%.

Biochemical Measurements:

Serum albumin levels will be measured on admission using a Beckman Coulter CX 9 clinical autoanalyzer (Beckman Coulter Inc). The reference range of serum albumin will be accepted as 3.5 to 5.5 g/dL. Biochemical measurements will be performed using standard methods. All laboratory results will be obtained within 48 hours after admission.

Other laboratory tests, including cardiac biomarkers (e.g., troponin, creatine kinase-MB), lipid profile, and renal function tests, will be also recorded.

Patients will be divided into 2 groups based on the presence of hypoalbuminemia, and both clinical and laboratory characteristics will be compared between these 2 groups. Survival status of the patients will be determined based on hospital records or telephone interviews.

Follow-up and Outcome Measures:

Patients will be followed for a minimum of [e.g., 3 months] after the index ACS event. The primary outcome will be a composite of major adverse cardiovascular events, including all-cause mortality, non-fatal myocardial infarction, and target vessel revascularization.

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Secondary outcomes will include individual components of the primary composite outcome, as well as cardiovascular mortality, HF hospitalization, and the development of chronic kidney disease or the need for renal replacement therapy.

Statistical analysis

Statistical analyses will be performed using SPSS 16.0 (SPSS Inc). Continuous variables were presented as mean + standard deviation and categorical variables will be expressed as percentages (%). Independent samples T test or Mann-Whitney U test will be used for continuous variables and the w2 test will be used for categorical variables. Correlations will be determined using Pearson correlation coefficient. The prognostic value of serum albumin for the primary and secondary outcomes will be evaluated using Cox proportional hazards regression models. Albumin will be treated as both a continuous variable and a categorical variable (with cutoff values determined based on quartiles or clinically relevant thresholds). Multivariable models will be constructed to adjust for potential confounders, including age, sex, comorbidities, ACS type, and other relevant clinical and laboratory variables. A two-sided p-value<0.05 will be considered statistically significant.

Results

The age was significantly higher in the hypoalbuminemia group than in the normal group (P value=0.007). While gender, smoking, comorbidities (DM, HTN and dyslipidemia), history of CAD, and left anterior descending artery (LAD) culprit coronary artery were insignificantly different between both groups. **Table 1**

Table 1: Age, gender, smoking, DM, HTN, dyslipidemia, history of CAD and LAD culprit coronary artery between the studied groups

		Hypoalbuminemia	Normal	P-value
Age (years)		62.8 ± 7.6	57.9 ± 9.9	**0.007***
Gender	**Male**	23 (46.0%)	32 (64.0%)	0.070
	Female	27 (54.0%)	18 (36.0%)	
Smoking		14 (28.0%)	17 (34.0%)	0.517
DM		17 (34.0%)	15 (30.0%)	0.668
HTN		30 (60.0%)	23 (46.0%)	0.161
Dyslipidemia		30 (60.0%)	27 (54.0%)	0.545
History of CAD		14 (28.0%)	11 (22.0%)	0.488
LAD culprit coronary artery		22(44.0%)	17 (34.0%)	0.305

Data is presented as mean ± SD, frequency (%), *significant as p value ≤0.05, DM: Diabetes mellitus, HTN: Hypertension, CAD: Coronary artery disease, LAD: Left anterior descending artery.

LVEF, total cholesterol, low-density lipoprotein (LDL) cholesterol, leukocytes, lymphocytes, neutrophils, platelets, C-reactive protein (CRP) and baseline troponin I were

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insignificantly different between both groups. Creatinine was significantly higher in the hypoalbuminemia group than in the normal group (P value=0.007). High-density lipoprotein (HDL) cholesterol, hemoglobin and peak troponin I were significantly lower in the hypoalbuminemia group than in the normal group (P value<0.05). **Table 2**

Table 2: Laboratory findings of the studied groups

	Hypoalbuminemia	Normal	p-value
LVEF	51.4 ± 9.8	52.0±9.3	0.740
Creatinine (mg/dL)	1.24±0.78	0.91±0.26	**0.007***
Serum albumin (g/dL)	3.13±0.20	3.85±0.23	-
Total cholesterol (mg/dL)	183.0 ± 14.3	195.0±46.0	0.082
LDL cholesterol (mg/dL)	115.8±45.6	119.3±35.2	0.664
HDL cholesterol (mg/dL)	32.8±6.9	37.0±7.9	**0.006***
Hemoglobin (g/dL)	12.5±1.5	13.4±1.7	**0.008***
Leukocytes (103 /mL)	9.5±2.8	10.0±2.3	0.367
Lymphocytes (103 /mL)	2.0± 1.0	2.4±1.1	0.136
Neutrophils (103 /mL)	6.6± 2.7	6.3±2.0	0.575
Platelets (103 /mL)	239± 68	248±69	0.506
CRP (mg/L)	2.4± 2.1	1.8±1.4	0.099
Baseline troponin I (ng/mL)	2.7± 2.3	3.3±2.5	0.213
Peak troponin I (ng/mL)	6.5± 4.3	8.9±4.8	**0.011***

Data is presented as mean ± SD, *significant as p value ≤0.05, LVEF: Left ventricular ejection fraction, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, CRP: C-reactive protein.

Patients received treatments (aspirin, clopidogrel, statin, b-blocker and angiotensin-converting enzyme inhibitor (ACE-I)/ angiotensin II receptor blocker (ARB) were insignificantly different between both groups. **Table 3**

Table 3: Secondary prevention pharmacotherapy for cardiovascular diseases

	Hypoalbuminemia	Normal	P-value
Aspirin	49(98.0%)	46(92.0%)	0.362
Clopidogrel	46(92.0%)	41(82.0%)	0.234
Statin	48(96.0%)	44(88.0%)	0.269
b-blocker	48(96.0%)	45(90.0%)	0.436
ACE-I/ARB	45(90.0%)	44(88.0%)	0.749

Data is presented as frequency (%), ACE: Angiotensin-Converting Enzyme Inhibitor, ARB: Angiotensin II Receptor Blocker.

Mortality was significantly higher in the hypoalbuminemia group than the normal group (p=0.007). Serum albumin was insignificantly different between died and alive patients. Serum albumin can significantly predict mortality at cut-off 3.34 with 71.4% sensitivity, 62%

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specificity, 33.3% PPV, 89.1% NPV and 64% accuracy (P value= 0.006). Serum albumin can significantly predict the mortality at cut-off 3.44 with 76.2% sensitivity, 57% specificity, 32% PPV, 90% NPV and 61% accuracy (P value= 0.007). **Table 4**

Table 4: Mortality, serum albumin, serum albumin mortality crosstabulation using the 3.34 cut-off and the 3.44 cut-off to improve sensitivity

Group mortality crosstabulation					P-value
			Mortality		
			Yes	No	
Groups	Hypoalbuminemia	% within Group	16(32.0%)	34(68.0%)	0.007*
		% within mortality	16(76.2%)	34(43.0%)	
	Normal	% within Group	5(10.0%)	45(90.0%)	
		% within mortality	5(23.8%)	45(57.0%)	
Serum albumin (g/dL)			3.34(0.38)	3.53(0.42)	0.062
Serum albumin mortality crosstabulation using the 3.34 cut-off					
Groups	Hypoalbuminemia	% within Serum albumin	15(33.3%)	30(66.7%)	0.006*
		% within mortality	15(71.4%)	30(38.0%)	
	Normal	% within Serum albumin	6(10.9%)	49(89.1%)	
		% within mortality	6(28.6%)	49(62.0%)	
Serum albumin mortality crosstabulation using the 3.44 cut-off to improve sensitivity					
Groups	Hypoalbuminemia	% within Serum albumin	16(32.0%)	34(68.0%)	0.007*
		% within mortality	16(76.2%)	34(43.0%)	
	Normal	% within Serum albumin	5(10.0%)	45(90.0%)	
		% within mortality	5(23.8%)	45(57.0%)	

Data is presented as frequency (%), *significant as p value ≤0.05.

Age, sex, smoking, DM, HTN, dyslipidemia, history of CAD, LAD culprit coronary artery, LVEF, total cholesterol, LDL cholesterol, HDL cholesterol, hemoglobin, leukocytes, neutrophils, platelets, aspirin, clopidogrel, statin and b-blocker were insignificantly different between died and alive patients. **Table 4**

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Table 4: Factors associated with mortality,

Group* Mortality (n=21)			p-value
Age (years)		63.1±7.2	0.115
Gender	Male	15(27.3%)	0.089
	Female	6(13.3%)	
Smoking		7(22.6%)	0.795
DM		8(25.0%)	0.501
HTN		10(18.9%)	0.578
Dyslipidemia		12(21.1%)	0.988
History of CAD		7(28.0%)	0.321
LAD culprit coronary artery		11(28.2%)	0.157
LVEF		10.8±52.5	0.653
Total cholesterol (mg/dL)		20.3±188.1	0.848
LDL cholesterol (mg/dL)		35.9±122.5	0.530
HDL cholesterol (mg/dL)		6.6±32.7	0.130
Hemoglobin (g/dL)		1.6±12.8	0.592
Leukocytes (103 /mL)		2.6±9.8	0.953
Neutrophils (103 /mL)		2.4±6.3	0.718
Platelets (103 /mL)		74±240	0.794
Aspirin		21(22.1%)	0.581
Clopidogrel		20 (23.0%)	0.290
Statin		21(22.8%)	0.198
b-blocker		18(19.4%)	0.159

Data is presented as mean ± SD, Frequency (%), DM: Diabetes mellitus, HTN: Hypertension, CAD: Coronary artery disease, LAD: Left anterior descending artery, LVEF: Left ventricular ejection fraction, LDL: Low-density lipoprotein, HDL: High-density lipoprotein.

Creatinine, lymphocytes, CRP and baseline troponin I were insignificantly different between died and alive patients. Peak troponin I was significantly lower in died patients than alive patients (P value=0.041). **Table 5**

Table 5: Selected biomarkers for cardiovascular and inflamatory evaluation

	Group *Mortality			
	Median	Minimum	Maximum	p-value
Creatinine (mg/dL)	0.8	0.1	2.9	0.314
Lymphocytes (103 /mL)	1.5	0.2	3.9	0.195
CRP (mg/L)	1.8	0.1	9.0	0.956
Baseline troponin I (ng/mL)	1.5	0.1	9.0	0.174
Peak troponin I (ng/mL)	5.7	0.4	15.7	0.041*

Data is presented as numbers, *significant as p value ≤0.05, CRP: C-reactive protein.

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Serum albumin can significantly predict mortality at cut-off 3.34 with 71.4% sensitivity, 62% specificity, 33.3% PPV, 89.1% NPV and 64% accuracy (P value= 0.006). Serum albumin can significantly predict the mortality at cut-off 3.44 with 76.2% sensitivity, 57% specificity, 32% PPV, 90% NPV and 61% accuracy (P value= 0.007). **Figure 1**

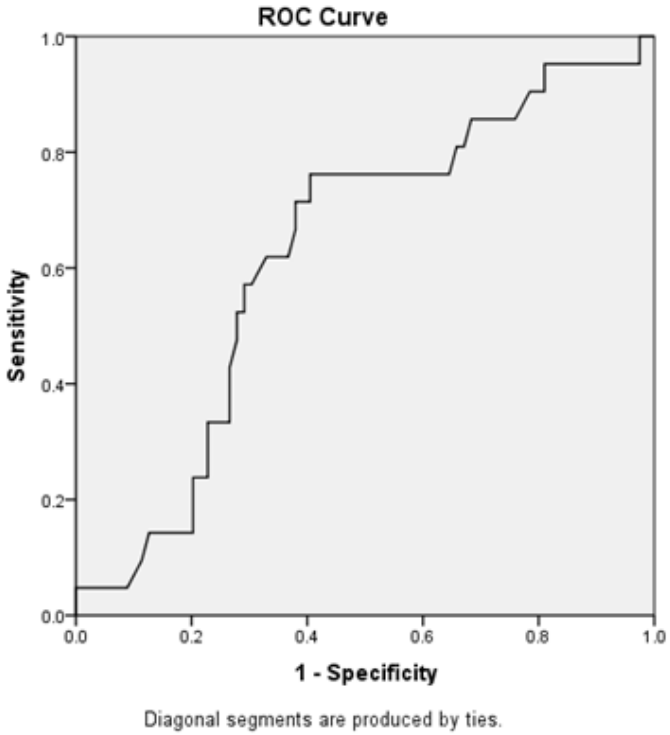

Figure 1: ROC Curve

Discussion

The ACS encompasses a range of clinical presentations, including unstable angina, NSTEMI and STEMI, all of which share a common pathophysiological mechanism of myocardial ischemia [9,10]. The prognosis for patients with ACS is influenced by a variety of factors, including the severity of the myocardial injury, the extent of CAD, comorbid conditions, and biomarkers that reflect the degree of tissue damage and inflammation. One such biomarker that has gained increasing attention in recent years is serum albumin [11, 12].
In the current study, creatinine was significantly higher in the hypoalbuminemia group than in the normal group. HDL cholesterol, hemoglobin and peak troponin I were significantly lower in the hypoalbuminemia group than in the normal group.

Along with our findings, Pan et al. [13] reported that participants with lower serum albumin levels had lower haemoglobin levels.

Besides, Watanabe et al. [14] found that creatinine was significantly higher in the hypoalbuminemia group than in the normal group.

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In the present study, mortality was significantly higher in the hypoalbuminemia group than normal.

It is known that albumin has antioxidant and anti-inflammatory effects [15, 16]. In case of hypoalbuminemia, oxidative stress and inflammation may further decrease the functionality of the heart and thus increase the mortality rates [17]. Also, low serum albumin may be associated with an increased risk for endothelial damage and for thrombotic events associated with platelet activation and aggregation [18,19].

Supporting our findings, Dhanju et al. [20] elucidated that the patients with low serum albumin levels had been linked with worse outcomes and mortality in patients of ACS.
Alongside our findings, Huang et al. [21] reported that CAD patients with hypoalbuminemia had higher mortality than those without hypoalbuminemia.

Our findings revealed that serum albumin can significantly predict mortality at cut-off 3.34 with 71.4% sensitivity, 62% specificity, 33.3% PPV, 89.1% NPV and 64% accuracy. Serum albumin can significantly predict the mortality at cut-off 3.44 with 76.2% sensitivity, 57% specificity, 32% PPV, 90% NPV and 61% accuracy.

The prognostic importance of hypoalbuminemia in patients with ACS may be due to the fact that hypoalbuminemia is associated with other comorbidities such as inflammation, malnutrition, and cachexia [22,23]. Another reason for the association of albumin with poor prognosis in patients with UAP/NSTEMI may be the association between low albumin levels and the development of HF [24].

Validating our findings, Yoshioka et al. [25] reported that serum albumin level was associated with the prognosis of patients with cardiovascular disease. Low albumin level has been shown to be useful in predicting adverse events, especially in CAD. In addition, albumin level has been already reported to be useful for predicting cardiac events, such as cardiovascular death and hospitalization for HF, in the acute and remote phases of ACS.
Besides, Zhu et al. [26] found that low serum albumin level was a powerful predictor of all-cause mortality in ACS patients.

Regarding the prediction long term of mortality. In consistence , Çinar et al. [27] concluded that admission serum albumin level was a strong and independent predictor of all-cause long-term mortality in patients with ACS.

Alongside our findings, Huang et al. [21] found that hypoalbuminemia were independently and jointly associated with long-term mortality among CAD patients.
In the same line, Polat et al. [3] reported that the cutoff value of 3.10 g/dL for serum albumin predicted mortality with a sensitivity of 74% and specificity of 67%.

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In the present study, age and sex were insignificantly different between died and alive patients. Smoking, DM, HTN, dyslipidemia, history of CAD and LAD culprit coronary artery were insignificantly different between died and alive patients. LVEF, total cholesterol, LDL cholesterol, HDL cholesterol, hemoglobin, leukocytes, neutrophils, and platelets were insignificantly different between died and alive patients. Creatinine, lymphocytes, CRP and baseline troponin I were insignificantly different between died and alive patients. Peak troponin I was significantly lower in died patients than alive patients.

Supporting our findings, Polat et al. [3] reported that there was no significant genders difference in short-term survival. DM prevalence did not show a consistent significant difference between those who died and survived. However, the mean age was higher in the mortality group. HTN and prior CAD were significantly more frequent in the mortality group. The mean LVEF was substantially lower in those who died. There was no significant correlation between albumin level and peak or baseline troponin values in ACS.
In this context, Sujino et al. [28] demonstrated a small but significant effect of higher initial troponin on in-hospital death in elderly STEMI.

Limitations included single center study that may result in different findings than elsewhere. Small sample size that may produce insignificant results. We didn't assess other inflammatory biomarkers such as CRP, and we didn't combine albumin with other predictors (as BNP) to improve risk stratification accuracy. We didn't stratify by ACS type (STEMI/NSTEMI) or demographics to identify high-risk subgroups.

Conclusions:

Serum albumin at cut off 3.34 is a promising predictor of short-term mortality in ACS patients. These support the potential utility of serum albumin as a simple, cost-effective biomarker for identifying patients at higher risk of mortality following an ACS event. Furthermore, the study found that patients with hypoalbuminemia had significantly higher levels of creatinine and lower levels of HDL cholesterol, hemoglobin, and peak troponin I compared to the patients with normal serum albumin.

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