Review Article

The role of albumin level in cardiovascular disease: a review of recent research advances

Goro Yoshioka[1], Atsushi Tanaka[1], Yuhei Goriki[2], Koichi Node[1]

[1]Department of Cardiovascular Medicine, Saga University, Saga, Japan; [2]Department of Cardiology, Ureshino Medical Hospital, Ureshino, Japan

Contributions: (I) Conception and design: G Yoshioka, A Tanaka; (II) Administrative support: G Yoshioka, A Tanaka; (III) Provision of study materials or patients: G Yoshioka, A Tanaka; (IV) Collection and assembly of data: G Yoshioka, A Tanaka; (V) Data analysis and interpretation: G Yoshioka, A Tanaka; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Goro Yoshioka; Atsushi Tanaka. Department of Cardiovascular Medicine, Saga University, 5-1-1 Nabeshima, Saga 849-8501, Japan. Email: s04211090s@gmail.com; tanakaa2@cc.saga-u.ac.jp.

Abstract: Serum albumin is a major component of serum protein that is widely used in clinical practice as an indicator of nutritional or inflammatory status. In clinical practice, serum albumin level is generally used to monitor the courses of several diseases, including hypovolemic state, burns, shock, surgical bleeding. Several studies have indicated that serum albumin level is associated with the prognoses of patients with cardiovascular disease (CVD). Low albumin level has been shown to be useful in predicting adverse events, especially in coronary artery disease. In addition, albumin level has been already reported to be useful for predicting cardiac events, such as cardiovascular death and hospitalization for heart failure (HF), in the acute and remote phases of acute coronary syndrome (ACS). Furthermore, the predictive value of albumin level for bleeding events has been reported, indicating the possibility of a comprehensive prediction of adverse events after ACS. However, since serum albumin levels fluctuate depending on a patient's general condition, the need for reassessment during the chronic phase of CVD has been discussed. In this review, we discuss the existing evidence on serum albumin level as a residual risk factor for coronary artery disease and outline future prospects for risk stratification using albumin level.

Keywords: Acute myocardial infarction (AMI); cardiovascular disease (CVD); heart failure (HF); bleeding; serum albumin

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Serum albumin

Serum albumin is an essential nutritional indicator, the major factor of oncotic pressure, and the modulator of body fluids (1). In clinical setting, serum albumin level is recognized as a simple and essential nutritional indicator and is generally used to evaluate the clinical courses of various diseases, including shock status, burns, bleeding event, trauma, hemorrhage, acute respiratory distress syndrome, terminal renal dysfunction, nutritional therapy, and post-resuscitation (2,3). Decrease in serum albumin level is common in critically ill patients and is mainly due to increased wasting, bleeding due to inflammation, and leakage of albumin from the gastrointestinal tract (1). Albumin level may also decrease as a result of redistribution from the intravascular space to the interstitial space due to increased capillary permeability, or as a reflection of poor nutritional status and liver function (4).

Regarding the action and pathophysiology of albumin, it is believed to have a significant relationship with cardiac dynamics and inflammation and has several characteristics in common with the aggravating factor of cardiovascular disease (CVD) (5). Thus, several studies have been conducted to clarify the relationship between serum albumin level and CVD.

In daily clinical setting, the serum albumin level under

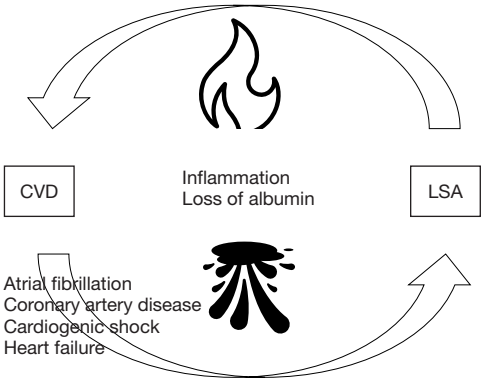

Figure 1 Relationship between cardiovascular disease and low serum albumin level. CVD, cardiovascular disease; LSA, low serum albumin.

3.5 g/dL is generally considered as hypoalbuminemia. However, there was no universal and clear definition of the low serum albumin (LSA) level to predict adverse events in cardiovascular medicine. Recent studies demonstrated an increased risk of cardiovascular events even at levels of 3.5–4.0 g/dL (6,7). In our previous study for patients admitted due to acute myocardial infarction (AMI), the serum albumin level under <3.8 g/dL was an independent predictor of adverse cardiac events at chronic phase of AMI (8). Therefore, we tentatively consider the serum albumin level under 3.8 g/dL as LSA of the risk factor in the area of cardiovascular medicine.

Previous reviews in 2010s have already summarized clinical evidence between serum albumin level and CVD (4). However, evidence including albumin level and bleeding event after CVD was not well taken in account. Furthermore, although serum albumin levels fluctuate depending on a patient's general condition, the need and significance for reassessment during the chronic phase of CVD has not been sufficiently discussed.

In this review, we discuss the existing studies and recent evidence on the association between serum albumin level, especially LSA, and CVD and present future prospects for risk assessment of CVD, including coronary artery disease.

Serum albumin and CVD

Reports on serum albumin and CVD are extensive. LSA level has been reported to be associated with CVD, such as heart failure (HF), atrial fibrillation, and coronary artery disease (*Figure 1*).

Development of CVD

Several studies have indicated that LSA is an independent predictor of the development of HF (9,10). A registry study of 2,907 patients (9) and a large observational study of 5,795 health checkup patients who were followed up for 9.6 years demonstrated that LSA is an independent risk factor for the development of chronic HF. This result was consistent after adjusting for various inflammatory markers and body mass index (BMI) (10).

Several reports have been published regarding the association between albumin level and arrhythmia (11,12). Albumin level has been shown to be an independent prognostic factor for the development of atrial fibrillation, the most common arrhythmia in real-world clinical settings. In an observational study of 8,870 patients (8-year observation period) without CVD, LSA was a predictor of the development of atrial fibrillation in women. This result was independent of other cardiac diseases, BMI, and inflammatory markers (12). An observational study of 830 patients with CVD who underwent coronary artery bypass surgery showed the usefulness of serum albumin level in the development of postoperative atrial fibrillation (13).

Prognostic factors for CVD

LSA is considered a predictive factor for the development and prognosis of CVD. In addition, LSA has been reported to be a useful predictor of HF. In a study of 352 patients with chronic HF, LSA at initial presentation was associated with elevated N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels and increased long-term mortality (14). LSA has also been shown to be an independent predictor of short- and long-term events in patients with acute HF. A study of 546 patients with non-ischemic acute HF showed that LSA at admission is an independent predictor of adverse cardiac events (15). In these reports, the adverse effects of LSA were attributed to malnutrition, decrease in hepatic synthesis, elevated vascular permeability, and/or renal dysfunction. Other reports have linked albumin level to adverse outcomes in a wide range of CVDs, including venous thromboembolism (16,17).

In a study of 204 patients with chronic coronary syndrome conducted over a 708-day observation period, the results showed that LSA predicted adverse cardiac events, including all-cause mortality, stroke, and myocardial infarction (MI) (18). Furthermore, a study of 1,316 patients with chronic coronary syndrome and chronic kidney

disease demonstrated that LSA at admission is a predictive factor for major adverse cardiac events (including all-cause mortality, stroke, and MI) in remote phase (19). These reports suggest that the primary cause of the negative effect of LSA is inflammation associated with severe atherogenesis.

Serum albumin and acute coronary syndrome (ACS)

Technological advances in primary reperfusion therapy, pharmacy, and physical therapy for ACS have reduced the incidence of short-term mortality in patients with ACS (20). The rate of in-hospital mortality among patients with ACS has improved as well, especially in the primary percutaneous coronary intervention (PCI) era (21). However, despite advances in multidisciplinary acute care for MI, the clinical need to manage the risks of HF and mortality, which are elevated in the remote phase of MI, remains unmet (22). Hence, risk stratification for the prevention of HF and mortality should be performed in the early phase of MI. Previous studies have shown that the following are important risk factors for HF and mortality in patients with MI: patient characteristic, electrocardiographic features, and factors of reperfusion therapy, ventricular function, and frailty status (23,24). Risk scores, such as the Global Registry of Acute Coronary Events (GRACE) score, have been established and are widely used (25,26). Albumin level is taken into account in some scores. Prognostic nutritional index is calculated using serum albumin and total lymphocyte count; geriatric nutritional risk index is calculated using serum albumin level and BMI; and controlling nutritional status score is calculated using the serum albumin level, total cholesterol, and total lymphocyte counts. These scores were also used as nutritional indicators and serve as significant prognostic factors in clinical outcomes in CVD (27-29). However, the clinical significance of albumin itself was not well identified in those existing models using multiple factors. Therefore, we examined the prognostic role of serum albumin level in CVD.

The prognostic impact of serum albumin level, a simple indicator, on cardiac events in the remote phase after ACS has been reported in several studies. A retrospective observational study of 7,192 patients with ACS showed that LSA (cutoff: 3.5 g/dL) at admission is an independent risk factor for in-hospital death and development of HF (6). There have also been several reports on hypoalbuminemia and the long-term prognosis of ACS (6,30). Our group conducted a single-center, retrospective observational study of 2,253 consecutive patients with AMI (8). In that study, the group with LSA (cutoff: 3.8 g/dL) in the acute phase of AMI showed a significantly increased frequency of adverse cardiac events (combined outcome of cardiovascular death and re-hospitalization for HF) in the chronic phase, indicating that LSA could be a prognostic factor for adverse cardiac events in patients with AMI. The results were consistent for each component of cardiovascular death and re-hospitalization for HF in the chronic phase of AMI, again demonstrating the clinical impact of risk stratification after AMI.

Regarding other adverse events, a single-center registry study demonstrated that LSA is useful for the risk stratification of bleeding events after PCI in patients with ACS, which is consistent with the results for the group without traditional bleeding risk factors (31). This finding was also reported in a multicenter registry study on AMI (32). In addition, a study of 1,724 consecutive patients with AMI undergoing PCI showed that LSA at admission is an independent predictor of chronic bleeding events. However, serum albumin levels are extremely variable during the acute phase of AMI owing to acute inflammation and circulatory dynamics (1). Some studies in which C-reactive protein/albumin ratio was used to account for the effects of inflammation showed its usefulness in predicting adverse cardiac events during hospitalization after coronary artery bypass surgery (33,34). Nevertheless, albumin levels may change with post-discharge medication use and change in nutritional intake status. Therefore, serum albumin levels measured in the acute phase of AMI alone may have limited predictive ability in the remote phase. The measurement of serum albumin levels during the chronic phase of AMI is expected to improve risk stratification. However, systematic reassessment in the chronic phase of AMI has not yet been fully established or implemented in real-world clinical settings. Hence, clinical evidence of the impact of such chronic transitions on post-MI prognosis is lacking. There are only a few studies on the association between chronic-phase serum albumin levels and long-term cardiovascular event. We conducted a single-center, retrospective, observational study of 1,424 consecutive patients with AMI to determine the changes in albumin levels from the acute phase of AMI to the chronic phase (one year later) in actual clinical practice (35). The results of the study suggest that LSA (cutoff: 3.8 g/dL) in the chronic phase of AMI, irrespective of the serum albumin level in the acute phase of AMI, is a predictive indicator for long-term outcomes

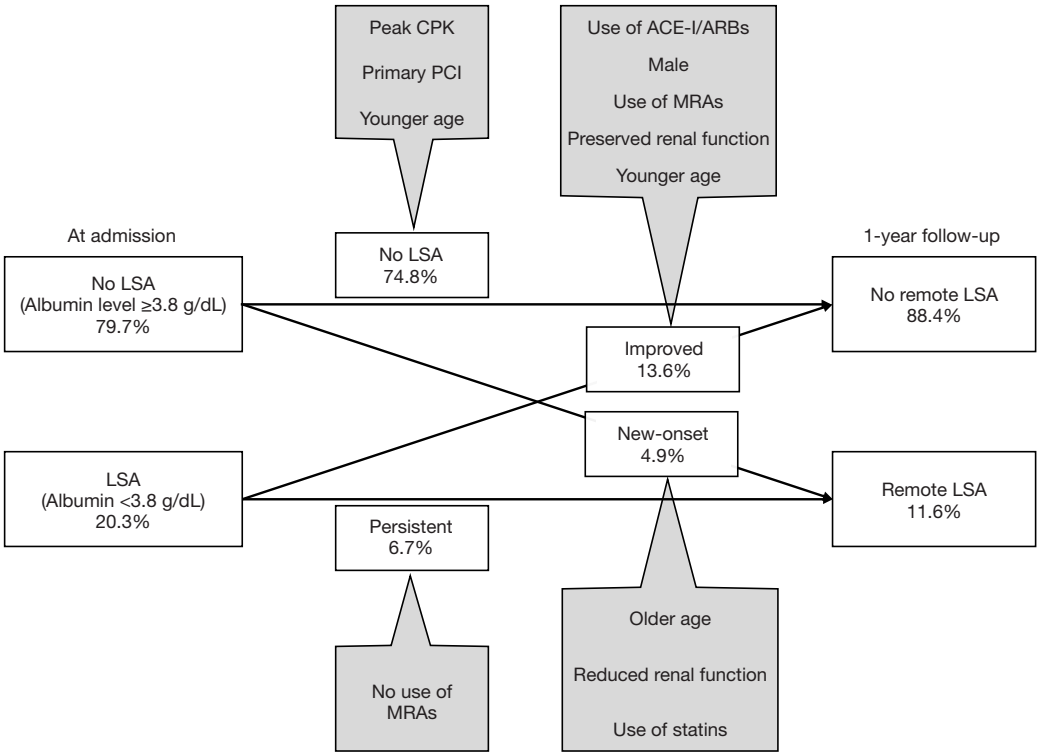

Figure 2 Risk factors for changes in albumin status. Adjusted for age, sex, body mass index, coronary risk factors (hypertension, dyslipidemia, diabetes mellitus, smoking, family history of cardiovascular disease), history of malignant tumor, onset-to-admission time, pre-thrombolysis in myocardial infarction grade, percutaneous coronary intervention, max creatine phosphokinase level, estimated glomerular filtration rate, length of hospital stay, use of statins at discharge, use of beta-blockers at discharge, use of angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers at discharge, use of mineralocorticoid receptor antagonists at discharge, left ventricular ejection fraction in the acute phase, cardiogenic shock, acute myocardial infarction due to lesions in left anterior descending artery, and acute myocardial infarction due to lesions in the left main trunk. CPK, creatine phosphokinase; PCI, percutaneous coronary intervention; ACE-I/ARB, angiotensin-converting enzyme inhibitor or angiotensin II receptor blocker; MRA, mineralocorticoid receptor antagonist; LSA, low serum albumin.

in AMI patients. In particular, our report demonstrated that use of statins and renin-angiotensin-aldosterone system inhibitors at discharge might positively associated with serum albumin levels (*Figure 2*). Thus, albumin level measured in the chronic phase can be a prognostic indicator, even in patients who survive the acute phase of AMI. However, careful monitoring is needed, especially for AMI patients with remote LSA.

Pathophysiological speculation of serum albumin for adverse events

Effect of serum albumin on HF after ACS

Malnutrition status, decrease in hepatic synthesis, elevated vascular permeability, and frailty have been suggested as adverse effects of LSA on cardiovascular events. LSA may induce increased peripheral edematous and pulmonary congestion, even at lower left ventricular end systolic pressures, acting as an aggravating factor for HF (4). Such changes are likely to contribute to new onset of HF, particularly in patients with reduced cardiac function after ACS. Serum albumin level is widely known as a marker of inflammation. However, previous reports have shown an association between LSA status and left ventricular remodeling through the activation of cardiac inflammation (36). Furthermore, pathophysiological evaluation has shown that excessive inflammation contributes to left ventricular remodeling associated with new development of HF (37). The acute phase of ACS causes inflammation and temporarily

decreases albumin levels (35). Inflammation and low albumin levels may have an additive negative impact on cardiovascular outcomes. Thus, further research on the association between inflammation and cardiac dysfunction is warranted.

Effect of serum albumin on bleeding events after ACS

Previous studies have indicated that the vulnerability of capillaries in systemic organs may be due to LSA (1). Protein metabolism plays an important role in tissue neogenesis and maintenance. Decreased albumin level is associated with tissue fragility, which consequently leads to fragility in vascular tissues and has a negative impact on bleeding events. Furthermore, LSA reflects malnutrition associated with vitamin C and K deficiencies. Vitamins C and K are essential for the coagulation system and play important roles in hemostasis during bleeding (38). Patients with LSA may simultaneously have vitamin deficiencies, which in turn may influence bleeding events after ACS. These multisystem factors may be responsible for increase in bleeding events after ACS in patients with LSA. However, the precise mechanism underlying high bleeding risk in patients with LSA is not well uncovered.

Limitation and future perspectives on the association between serum albumin and CVD

First, the detailed pathophysiological mechanisms underlying the serum albumin levels and adverse outcome are still uncertain. Future studies should be focused on albumin fluctuations from the acute to the chronic phase of CVD, because albumin fluctuations may help clarify the pathophysiology underlying the association between albumin level and CVD. Second, regarding the elevated albumin levels, there was little study examining the impact of non-LSA and elevated level of serum albumin levels in CVD. In an observational study for Korean, a higher serum albumin level was associated with an increased prevalence of metabolic syndrome (39). At the same time, an obesity paradox, in which overweight have a better prognosis compared with non-overweight/non-obese patients (40), is also well-known. Accordingly, there might be a possibility that an elevated level of serum albumin could play a protective role in cardiovascular medicine. In our recent study, among 289 AMI patients with LSA (<3.8 g/dL) at acute phase of AMI, 194 AMI patients (67.1%) showed improvement of albumin level (≥3.8 g/dL) at chronic phase. This improved subgroup demonstrated better outcomes in comparison with non-improved subgroup (35). However, it is still uncertain whether the higher the albumin levels, the better the prognosis in CVD. Third, evidence about the impact of interventions for increasing serum albumin levels on CVD is also uncovered. Albumin infusion therapy was significantly associated with a reduced risk of rebleeding and short-term mortality in patients with cirrhosis admitted for acute gastrointestinal bleeding (41). In contrast, recent retrospective observational study on the effect of albumin replacement in patients with HF did not show the significant clinical benefits of the therapy (42). Thus, the significance of interventions for LSA seems to remain controversial, which may partly result from differences in the methods of intervention and/or patient backgrounds, including disease status, among studies.

Serum albumin levels have been frequently used as a nutritional indicator. However, they could not reflect immediate changes in the nutritional and/or inflammatory statuses. Because prealbumin have shorter biologic half-life (2 days), compared with that of albumin (20 days), it would be sensitive to immediate changes in nutrition or inflammation (43). In a recent observational study, prealbumin was an independent factor to predict the development of HF after AMI (38). However, the clinical impact of prealbumin on cardiovascular events remains to be poorly understood. Association between prealbumin and serum albumin level, especially LSA, was also still unclear.

Conclusions

Serum albumin level has evolved as an indicator of nutrition and inflammation status, and its use in predicting the prognosis of CVD has recently been reported. Its usefulness in predicting bleeding events in coronary artery disease has also been reported, indicating its potential for comprehensive prediction of adverse events after ACS. Furthermore, evaluation of serum albumin levels in the chronic phase of ACS has shown the importance of risk stratification and post-ACS reassessment of LSA for the improvement of long-term prognosis. Further research on the pathophysiology of albumin level is needed to provide insight into the causal relationship between albumin levels and adverse events in CVD patients, which is currently unclear.

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Footnote

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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