Oxidized albumin as a mediator of kidney disease

Figueroa, S.M.
Araos, P.
Reyes, J.
Gravez, B.
Barrera-Chimal, J.
Amador, C.A.

Abstract
Renal diseases are a global health concern, and nearly 24% of kidney disease patients are overweight or obese. Particularly, increased body mass index has been correlated with oxidative stress and urinary albumin excretion in kidney disease patients, also contributing to increased cardiovascular risk. Albumin is the main plasma protein and is able to partially cross the glomerular filtration barrier, being reabsorbed mainly by the proximal tubule through different mechanisms. However, it has been demonstrated that albumin suffers different posttranslational modifications, including oxidation, which appears to be tightly linked to kidney damage progression and is increased in obese patients. Plasma-oxidized albumin levels correlate with a decrease in estimated glomerular filtration rate and an increase in blood urea nitrogen in patients with chronic kidney disease. Moreover, oxidized albumin in kidney disease patients is independently correlated with higher plasma levels of transforming growth factor beta (TGF-β1), tumor necrosis factor (TNF-α), and interleukin (IL)-1β and IL-6. In addition, oxidized albumin exerts a direct effect on neutrophils by augmenting the levels of neutrophil gelatinase-associated lipocalin, a well-accepted biomarker for renal damage in patients and in different experimental settings. Moreover, it has been suggested that albumin oxidation occurs at early stages of chronic kidney disease, accelerating the patient requirements for dialytic treatment during disease progression. In this review, we summarize the evidence supporting the role of overweight-and obesity-induced oxidative stress as a critical factor for the progression of renal disease and cardiovascular morbimortality through albumin oxidation.

Author keywords
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