Leptin is a Negative Acute-Phase Protein in Chronic Hemodialysis Patients

Masoumeh Mohkam

Department of Pediatric Nephrology and Pediatric Infections Research Center, Faculty of Medicine, Shahid Beheshti University of Medical Sciences, Tehran, Iran

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Leptin is a hormone secreted by adipocytes that regulates thermogenesis, appetite, and body fat. This hormone is a product of the ob gene and is a single-chain 16-kDa protein that is expressed almost exclusively in human adipose tissue. Furthermore, leptin has actions in the immune system, bone formation, and angiogenesis.[1,2] Previous data indicated that high serum leptin is a component of the metabolic syndrome and a risk factor for cardiovascular disease in the general population and patients with normal kidney function.[3,4] In patients with normal glomerular filtration rate (GFR), there is net renal uptake of 12% of circulating leptin, whereas in renal insufficiency there is no renal uptake of leptin. Plasma leptin is not cleared by hemodialysis with a modified cellulose membrane.[5] It seems that kidney failure is an important factor affecting blood leptin levels, especially in obese patients. Hyperleptinemia was closely related to fat mass in patients on chronic hemodialysis.[6] Carrero and coworkers demonstrated that patients with protein energy wasting and low plasma ghrelin had high leptin and high C-reactive protein levels, and higher mortality rate compared to the control group.[7] On the other hand, Nasri and Baradaran reported that in patients on hemodialysis, the association of leptin with C-reactive protein levels was inversed which could show the positive effects of leptin on nutrition and support the theory of protective effects of leptin in hemodialysis patients.[8]

Scholze and colleagues showed that low serum leptin is an independent predictor of mortality in stage 5 of CKD patients undergoing hemodialysis. They reported that baseline serum leptin was significantly lower in patients who died from cardiovascular disease or infections, but not in patients with cancer.[1] Experimental studies suggest that leptin deficiency reduces production of endothelial nitric oxide, which has protective effects and enhances sensitivity to endotoxin-induced lethality.[9,10] It supports Kalantar-Zadeh and colleagues' study results which showed that a low body fat percentage and fat loss over time are associated with higher mortality in patients undergoing maintenance hemodialysis.[11] Due to discrepancy and different conflicting results of prior studies evaluating the relationship between leptin and outcomes in patients undergoing hemodialysis, Bian and coworkers evaluated the association of leptin with mortality in patients on stable maintenance hemodialysis in a prospective study which published in this issue of the *Iranian Journal of Kidney Diseases*. They studied a total of 53 hemodialysis patients with no clinically active cardiovascular or infectious diseases. In this cohort study, the patients were on regular and acceptable hemodialysis. The authors evaluated predialytic serum leptin in their study group and found out that serum leptin concentration was significantly correlated with body mass index (Spearman r = 0.701; $P < .001$). In their 5-year follow-up, a total of 26 patients (49.1%) died; 15 (57.7%) of these 26 deaths were attributable to cardiovascular disease. Serum leptin concentrations at the study entry were lower among the 26 deceased patients (all-cause mortality) compared with those 27 patients who survived (median, 2.66 ng/mL [0.87 ng/mL to 6.31 ng/mL] versus 5.75 ng/mL [2.03 ng/mL to 29.85 ng/mL]; $P = .02$). The authors concluded that a low serum leptin concentration was associated with all-cause mortality, but not cardiovascular disease mortality in stable patients.[12]

It seems that the actions of leptin in stable chronic hemodialysis patient differ from that found in patients with normal kidney function and or general population.

CONFLICT OF INTEREST

None declared.

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Correspondence to:
Masoumeh Mohkam, MD
Pediatric Infections Reaserch Center, Mofid Children's Hospital, Shariati Ave, Tehran, Iran
Tel: +98 21 2222 7033
Fax: +98 21 2222 7033
E-mail: mohkam@pirc.ir