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Journal of Parathyroid Disease

Journal of Parathyroid Disease 2014,2(1),53–54

News and Views

Association of serum leptin and bone metabolism indices in peritoneal dialysis patients

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Leptin is a 16000 Dalton's protein that is produced primarily in fat cells which regulates the amount of fat stored in the body (1). Large fat cells produce greater amount of leptin than small cells, so serum leptin concentration is correlated with body fat mass in adults. Secretion of leptin by adipocytes may rapidly decrease during starvation (2). In addition, leptin secretion is stimulated by insulin and glucocorticoids. Sense of satiety occurs when the amount of fat storage reaches a certain level. Leptin circulates in blood as both free and bound forms to leptin binding protein. Leptin has higher levels in blood at midnight than early morning, probably due to suppression of appetite throughout the night. Leptin circulates through the circulation, and activates its receptors in the hypothalamus, and then energy expenditure may be increased by the signal to the brain, and leptin receptors on peripheral tissues (3). Furthermore, leptin could act as the opposite of ghrelin, called "hunger hormone". Site of ghrelin and leptin receptors are the same, thus these cells may receive both hunger and satiety signals (1,2). Neuropeptide Y (NPY) is potent stimulators for food intake, thus leptin could decrease the amount of NPY as well as food intake, increase energy expenditure and eventually cause weight loss. In addition, leptin can suppress secretion of alpha-melanocyte-stimulating hormone, and could block the feelings of hunger.

The kidneys have important role in the metabolism of leptin, because leptin is a small protein with molecular weight of 16000, which is freely filtered by the glomerulus; however, a very small amount of leptin appears in the urine. Kidneys could absorb almost all of uptake of filtered leptin and may result in its degenerating via tubular cell degeneration. Other organs such as muscle, lung and liver may also play an important role in the leptin metabolism. Serum level of leptin is higher than normal in almost all of chronic kidney disease (CKD) patients, but not all of them. Although definite cause of increased serum leptin level in CKD is not well defined. However, various factors may be involved such as; loss of significant number of functioning nephrons, chronic inflammation, dialysis membrane and hyperinsulinemia.

Implication for health policy/practice/research/medical education

The role of leptin in the bone metabolism in chronic kidney disease or end-stage renal disease patients especially in patients under peritoneal dialysis is not exactly clear. Further studies are needed in this regard.

Leptin could also cause adverse outcome in CKD patients, for example anorexia and weight loss due to protein energy wasting, frequently observed in dialysis patients, may be related to high serum leptin. Moreover, presence of leptin receptors in some peripheral organs such as kidney may raise this possibility. Thus, it can be concluded that leptin may probably involve in pathways other than weight and energy metabolism control.

On the other hand, serum leptin has correlation with bone density in male population (4). Leptin may also be a dual effect on bone metabolism, due to local stimulation of osteoblast and inhibitory effect on hypothalamus (5). In addition, leptin could decrease bone metabolism in chronic kidney diseases patients due to bone resistance. For instance, in the study on 75 continuous ambulatory peritoneal dialysis (CAPD) patients in 2007, we found higher level of serum leptin in female compare to male patients (6). There was significant correlation between serum leptin level and duration of renal failure. There was also a reverse association between serum leptin level and serum parathormone. However, we did not find any correlation of serum leptin level and other indices of bone metabolism such as serum calcium, phosphorus and alkaline phosphatase (6). Ahmadi *et al.* (7) in study on 72 hemodialysis patients showed that serum leptin had positive correlation with calcium but not with bone mineral density (BMD). Małyszko in the study on 25 hemodialysis and 23 CAPD patients found no association of serum leptin and bone mineral density or bone turn

Received: 12 January 2014, Accepted: 24 February 2014, ePublished: 1 March 2014

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over markers (8). Additionally, in the study of Zoccali *et al.* (9) on 161 hemodialysis patients, they concluded a reverse correlation of serum leptin and serum PTH and skeletal alkaline phosphatase in the male patients. There are also some other studies on the effect of leptin on bone metabolism in normal population. For example, in 296 healthy post-menopausal women, serum leptin has correlation with bone mineral densitometry (10). Likewise, Sato in 221 healthy men showed a reverse correlation of bone mineral densitometry and serum leptin level (11).

In conclusion, the role of leptin in the bone metabolism in chronic kidney disease or end-stage renal disease patients especially in patients under CAPD is not exactly clear. Further studies are needed in this regard.

Authors' contributions

All authors contributed to the manuscript equally.

Conflict of interests

The authors declared no competing interests.

Ethical considerations

Ethical issues (including plagiarism, misconduct, data fabrication, falsification, double publication or submission, redundancy) have been completely observed by the authors.

Funding/Support

None.

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Please cite this paper as: Momeni A, Amiri M. Association of serum leptin and bone metabolism indices in peritoneal dialysis patients. J Parathyr Dis 2014; 2(1): 53-54.