Elevated serum parathyroid hormone is a heart risk factor in hemodialysis patients

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Cardiovascular disease is a major cause of mortality and disability throughout the world. The prevalence of cardiovascular disease is increasing rapidly (1). Patients with end-stage kidney failure generally have cardiovascular diseases too and cardiovascular disease is the most common cause of death in patients on hemodialysis (1,2). Cardiovascular disease accounts for most of the morbidity in this group of patients (1-3). Hemodialysis patients are also expose to atherosclerosis and consequent ischemic cardiac disease, on the other hand, myocardial dysfunction and overt cardiac insufficiency also are highly prevalent too (1-3). Secondary hyperparathyroidism is common in patients on hemodialysis and is defined by excessive serum parathyroid hormone level, parathyroid gland hyperplasia and an imbalance in calcium and phosphorus metabolism (1-4). In fact, parathormone is a main uremic toxin, and may be responsible for long-term outcomes that include severe vascular calcifications, kidney osteodystrophy, immune dysfunction, alterations in heart structure and function, and anemia (1-5). These adverse consequences may contribute to an increased risk of cardiovascular mortality and morbidity among end-stage renal failure patients (2-5). It was recognized that hyperphosphatemia is related to cardiovascular disease and, hence, increased mortality and morbidity. Despite dramatic improvement in our understanding of the pathogenesis, pathophysiology and sequels of secondary hyperparathyroidism, research is still required to better understand the role of parathormone excess and hyperphosphatemia in hemodialysis patients (3-6). To found the association of parathyroid hormone excess due to secondary hyperparathyroidism with coronary artery disease, an investigation was planned on a group of stable hemodialysis patients. The presence of cardiac chest pain was confirmed through the complaint of heart burn or epigastric pain, retrosternal discomfort and chest compression ,which was established by symmetrical depressed T wave at that time on a 12-lead ECG by means of a 12-channel and also improving the pain after taking sub-lingual trinitroglycerine pearls.

A sample of 36 stable hemodialysis patients was tested. Around 21% of patients had chest pain. Median of intact PTH of patients was 309 pg/ml. In our study, a significant difference of hemodialysis duration, hemodialysis amount and also serum phosphorus between patients with and without cardiac chest pain was detected. There was also a significant difference of intact PTH between male hemodialysis patients with and without cardiac chest pain. This study supported the importance of better control of secondary hyperparathyroidism as a responsible factor to promote the coronary heart disease in hemodialysis patients (7). Recently Lishmanov et al. studied, 196 patients with stages 3 and 4 chronic renal failure, during median follow-up of 27.2 months, 48 patients had cardiovascular events, while 148 patients did not. Intact parathyroid hormone was elevated for patients who had cardiovascular events compared with those without. They concluded that, intact parathyroid hormone level in patients with stages 3 and 4 chronic kidney disease is associated with increased incidence of cardiovascular events independent of calcium-phosphorous level. Indeed
intact parathyroid hormone levels were detected to be an important and an independent risk factor for left ventricular hypertrophy (8). Parathyroid hormone acts on cardiomyocytes by binding to the PTH/PTHrP receptor, which stimulates a rise in the intracellular levels of calcium. Increased calcium levels activate protein kinase C and mediate hypertrophic and metabolic processes (1-4,9-11). Various published investigations detected that intact parathyroid hormone contributes to heart fibroblast activation and the fibrosis of cardiac myocytes, which is a precondition of diastolic dysfunction (9-11). Furthermore, higher levels of calcium due to high parathyroid hormone levels have been found to induce arrhythmia (1-7). Hence, high parathyroid hormone levels are accompanied with diastolic dysfunction and left ventricular hypertrophy in patients on hemodialysis (8-12). The reduction of parathyroid hormone levels to normal ranges may have beneficial effect to reduce left ventricular hypertrophy and improve heart function. Thus, among hemodialysis population, higher parathyroid hormone concentrations were associated with higher all-cause mortality risk, mostly explained by fatal cardiovascular events (8-12). We suggest to adequate control of hyperparathyroidism to reduce the risk of developing left ventricular hypertrophy.

Author's contribution
HN is the single author of the manuscript.

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