# Secondary Hyperparathyroidism in Association with Malnutrition – inflammation complex Syndrome in chronic hemodialysis

H NASRI A BARADARAN
*Nephrology Research Center, Shahrekord University of Medical Sciences, Hajar Medical, Educational & Therapeutic Centre, Iran*
*Correspondence to Dr. Hamid Nasri, Nephrologist, Assistant Professor E. mail: hamidnasri@yahoo.com*

**To found the association of high PTH levels with some indexes of malnutrition-inflammation complex syndrome (MICS), a study was carried out a group of maintenance hemodialysis patients(MHPs) consisting of nondiabetic and diabetic patients. Intact serum PTH (iPTH) and serum C-reactive protein(CRP), serum calcium (Ca), phosphorus (P), alkaline phosphatase (ALP), serum cholesterol (chol)and serum triglyceride (Tg) were measured .Body mass index (BMI) was also calculated . Total patients were 36(f=15 m=21), consisting of 25non-diabetic HD patients and 11 diabetic HD patients. The mean patient's age was 44(±17) years. The value of serum iPTH of total HD patients was 434±455 (median:309)pg/ml, the value of serum iPTH of diabetic and nondiabetic-dialysis patients were 201±277(median:41) and 537±483(median:340)pg/ml respectively. In this study we found a near significant positive correlation of serum iPTH with serum CRP, a significant inverse correlation of serum iPTH with BMI and a near significant positive correlation of serum ALP with Logarithm of CRP, a significant positive correlation of serum phosphorus with serum CRP and also a significant inverse correlation of serum phosphorus with BMI were found. A near significant inverse correlation of serum cholestrol with serum phosphorus as well as a near significant inverse correlation of s erum c holestrol w ith s erum C RP w ere e xisted t oo. W hen p atients w ith i PTH below than 200 pg/ml were deleted, the correlation of iPTH with CRP was positive ($r$ =0.42, $p$ =0.085) and when patients with iPTH more than 500pg/ml were deleted, this correlation was found to be negative($r$ = -0.42, $p$ =0.047), means that a low iPTH values is an index of malnutrition while higher values is associated with inflammation .Further attention needs to better control of hyperphosphatemia a nd m aintaining t he i PTH l evels 1 .5 t imes o f normal to avoid the sides effects of secondary hyperparathyroidism.**

**Keywords: End stage, renal failure, secondary hyperparathyrodisim, nutritional status.**

Among potential candidates for the high rate of hospitalization and mortality in maintenance dialysis patients, both protein-energy malnutrition (PEM) and inflammation continue to be at the top of the list. Epidemiological studies repeatedly and consistently have shown a strong association between clinical outcome and measures of both malnutrition[1,2] and inflammation in dialysis patients[3,4]. Moreover, many investigators have observed that these two conditions tend to occur concurrently and coexist in individuals with ESRD, and many factors that engender one of these conditions also lead to the other[233,5,6]. Therefore, the terms malnutrition-inflammation complex syndrome (MICS)[2,7], or malnutrition, inflammation, and atherosclerosis (MIA) syndrome[8] have been proposed to indicate the combination of these two conditions in these patients. The MICS increasingly has become the main focus of attention of outcome research concerning maintenance dialysis patients. Indeed Malnutrition is present to some extent in approximately 40% of chronic renal failure (CRF) patients on maintenance hemodialysis[9,10]. Several markers of malnutrition such as low body mass index and low serum albumin have been associated with high morbidity and mortality rates in this group of patients[11,12]. Malnutrition in these patients is considered to be due to anorexia with low food intake[13,14], to the loss of nutrients and catabolism during the dialysis procedure[15,16], intercurrent illnesses[17], metabolic acidosis[18], glucose intolerance[19], increased cytokine levels[20], and other hormonal derangements[21].

Among these last disturbances, high parathyroid hormone (PTH) levels, frequently observed in CRF patients, may be implicated i n t he nutritional abnormalities found i n t hese patients. In fact, it has been observed that patients with primary hyperparathyroidism may show evidence of weight loss, weakness and muscle atrophy and negative nitrogen balance[21,23]. In this regard few studies have analyzed the association of high PTH levels with body mass index as a marker of nutritional status and serum C-reactive protein as a marker of inflammation to better found the association of high PTH levels with malnutrition-inflammation complex syndrome (MICS). We therefore sought to study this adverse effect of secondary hyperparathyroidism in a group of maintenance hemodialysis patients (MHPs) consisting of nondiabetic and diabetic patients.

**Patients and methods:**

***Patients***: This cross-sectional study was conducted on patients with end-stage renal disease (ESRD), who were undergoing maintenance hemodialysis treatment with acetate basis dialysate and polysulfone membranes. According to the severity of secondary hyperparathyroidism, each patient being treated for secondary hyperparathyroidism was given oral active vitamin D3 (Rocaltrol), calcium carbonate, and Rena-Gel capsules at various doses. According to the severity of anemia ,patients were under IV iron therapy with Iron sucrose (venofer) at various doses after each dialysis

session, all patients were under treatments of 6mg folic acid daily, 500mg L-Carnitine daily , oral Vitamin B-complex tablet daily and also 2000U IV Eprex (recombinant human erythropoietin (rHuEPO) unique for each patient after each dialysis session routinely .Exclusion criteria were active or chronic infection.The study was done in the hemodialysis section of Hajar Medical Educational & Therapeutic Center of Shahrekord University of Medical Sciences in Shahrekord of Iran.

***Laboratory methods:*** After an overnight fast, blood samples were obtained. Intact serum PTH (iPTH) was measured by the radioimmunoassay (RIA) method using DSL-8000 of USA (normal range of values is 10-65 pg/ml). Also peripheral venous blood samples were collected for biochemical analysis including serum predialysis creatinine(Creat), post and predialysis blood urea nitrogen(BUN), albumin(Alb) as well as serum C-reactive protein(CRP), serum calcium (Ca), phosphorus (P) , alkaline phosphatase (ALP),serum cholesterol (chol)and serum triglyceride (Tg) were measured using standard kits. Body mass index (BMI) calculated using the standard formula (postdialyzed weight in kilograms/height in square meters ;$kg/m^2$) (25).For the efficacy of hemodialysis the urea reduction rate (URR) was calculated from pre- and post-blood urea nitrogen (BUN) data (26). Duration and the amount of sessions of hemodialysis treatment were calculated from the patients' records. The duration of each hemodialysis session was 4 hours.

***Statistical analysis:*** Results are expressed as the mean±SD and median values. Comparison between the groups was done using Student's t-test. Statistical correlations were assessed using partial correlation test. Statistical analysis was performed on total hemodialysis (HD), females, males, diabetics and non diabetic's populations separately. For some correlations the logarithm of some data were used too. All statistical analyses were performed using SPSS (version 11.5.00). Statistical significance was determined at a $p$-value <0.05.

**Results:**

Total patients were 36(f=15 m=21), consisting of 25(f=11 m=14) non-diabetic HD patients and 11(f=4 m=7) diabetic HD patients. Table 1, 2 and 3 show the Mean ±SD, Minimum and Maximum and median of age, duration and sessions of hemodialysis and also laboratory results of total hemodialysis (HD) patients. The value of serum iPTH of total HD patients was 434±455 (median:309) pg/ml, the value of serum iPTH of diabetic and nondiabetic-dialysis patients were 201±277 (median:41) and 537±483(median:340)pg/ml respectively. In total HD patients a near significant positive correlation of serum iPTH with serum CRP ($r$=0.33, $p$=0.081; figure1) (adjusted for Ca, P, URR, DM, age, duration and sessions of dialysis) was seen , a significant inverse correlation of serum iPTH with BMI ($r$=-0.46, $p$ =0.038; figure2) (adjusted for dialysis sessions) in male HD patients were seen. In total patients a near significant positive correlation of serum ALP with Logarithm of CRP ($r$= 0.32, $p$ =0.069; figure3) (adjusted for age, duration and sessions of dialysis) was found . In this group also a significant positive correlation of serum phosphorus with serum CRP ($r$=0.31, $p$=0.065; figure4) (adjusted for age duration & sessions of dialysis) and a significant inverse correlation of serum phosphorus with BMI ($r$=-0.31, $p$=0.042; figure5) (adjusted for dialysis sessions) were found too. Moreover in total HD patients a significant positive correlation of serum triglyceride with BMI ($r$ = 0.43, $p$ =0.012) and a near significant inverse correlation of serum cholesterol with serum phosphorus ( $r$ =-0.29, $p$ =0.093) as well as a near significant inverse correlation of serum cholesterol with serum CRP ($r$ =-0.29, $p$=0.091) (adjusted for age, duration and sessions of dialysis for three above correlations were demonstrated) was existed too. In total patients a significant inverse correlation of serum albumin with logarithm of CRP ($r$=-0.33, $p$ =0.038) ( adjusted for age, dialysis sessions and duration, serum Ca and P) was seen .In male HD patients there was a near significant positive correlation of serum albumin with BMI ($r$=0.999942, $p$=0.063; figure 6).

Table 1:Mean±SD, Minimum and maximum of age, duration, sessions & also laboratory results of total hemodialysis patients (n=36)

| | Minimum | Maximum | Mean±SD | Median |
|---|---|---|---|---|
| Age in years | 16 | 80 | 44±16.5 | 43 |
| DH* months | 2 | 156 | 30±36 | 17.5 |
| Dialysis dose sessions | 18 | 1584 | 285±396 | 144 |
| URR % | 39 | 75 | 53.5±9.8 | 57.5 |
| Ca mg/dl | 5 | 10 | 6.4±1.9 | 7.9 |
| P mg/dl | 3 | 10 | 6.4±1.9 | 6.2 |
| ALP IU/L | 175 | 5487 | 628±891 | 433 |
| Alb g/dl | 2.4 | 4.8 | 3.8±0.5 | 3.96 |
| CRP mg/l | 3 | 40 | 8.7±6.7 | 8 |
| iPTH pg/ml | 16 | 1980 | 434±455 | 309 |
| Chol mg/dl | 59 | 211 | 117±38 | 115 |
| TG mg/dl | 29 | 461 | 130±96 | 95 |
| BMI $kg/m^2$ | 16 | 34 | 22±4.4 | 21.5 |

***Duration of hemodialysis**

Table 2:Mean ±SD, Minimum and Maximum of age, duration , sessions and also laboratory results of non diabetic hemodialysis patients. (n=25)

| | Minimum | Maximum | Mean±SD | Median |
|---|---|---|---|---|
| Age in years | 16 | 80 | 44±16.5 | 43 |
| $DH^+$ months | 2 | 156 | 40±40.8 | 22 |
| Dialysis dose sessions | 36 | 1584 | 370±452 | 156 |
| URR % | 60 | 76 | 61±7.5 | 60 |
| Ca mg/dl | 6 | 15 | 7.8±0.75 | 8 |
| P mg/dl | 4 | 10 | 6.6±1.8 | 6.5 |
| ALP IU/L | 190 | 5478 | 760±1044 | 478 |
| Alb g/dl | 2.4 | 4.7 | 3.8±0.5 | 4 |
| CRP mg/l | 2 | 20 | 7.4±3.8 | 6 |
| iPTH pg/ml | 22 | 1980 | 537±483 | 340 |
| Chol mg/dl | 59 | 171 | 110±33 | 120 |
| TG mg/dl | 61 | 461 | 129±85 | 99 |
| BMI $kg/m^2$ | 16 | 33 | 21±4.6 | 19 |

***Duration of hemodialysis**

Table 3: Mean ±SD, Minimum and Maximum of age, duration , sessions and also laboratory results of diabetic hemodialysis patients. (n=25)

| | Minimum | Maximum | Mean±SD | Median |
|---|---|---|---|---|
| Age in years | 27 | 75 | 53±15.8 | 55 |
| $DH^+$ months | 6 | 24 | 14.5±6 | 12 |
| Dialysis dose sessions | 54 | 216 | 123±54 | 108 |
| URR % | 39 | 75 | 53.5±9.85 | 54 |
| Ca mg/dl | 5 | 10 | 7.4±1.3 | 7.5 |
| P mg/dl | 3 | 10 | 5.9±2 | 6 |
| ALP IU/L | 175 | 584 | 327±148 | 295 |
| Alb g/dl | 3 | 4.8 | 3.8±0.5 | 3.9 |
| CRP mg/l | 4 | 40 | 12±10 | 10 |
| iPTH pg/ml | 16 | 840 | 201±277 | 41 |
| Chol mg/dl | 60 | 211 | 133±49 | 111 |
| TG mg/dl | 29 | 456 | 130±120 | 88 |
| BMI $kg/m^2$ | 20 | 34 | 23.3±4 | 23 |

***Duration of hemodialysis**

**Figure 1**: Near significant positive correlation of serum iPTH with serum CRP.

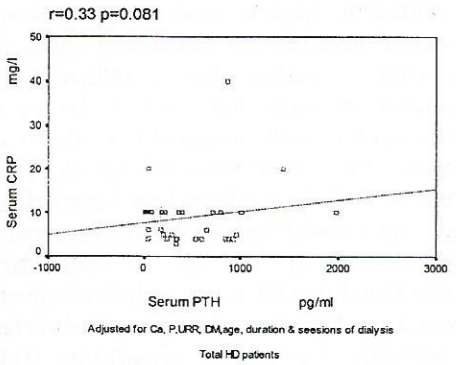



**Figure 2**: Significant inverse correlation of serum iPTH with BMI

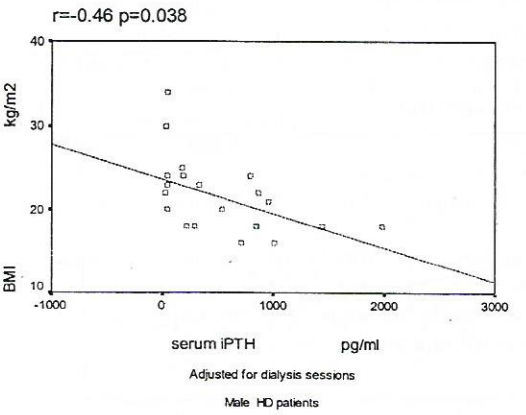



**Figure3:** Near significant positive correlation of serum ALP with Logarithm of CRP.

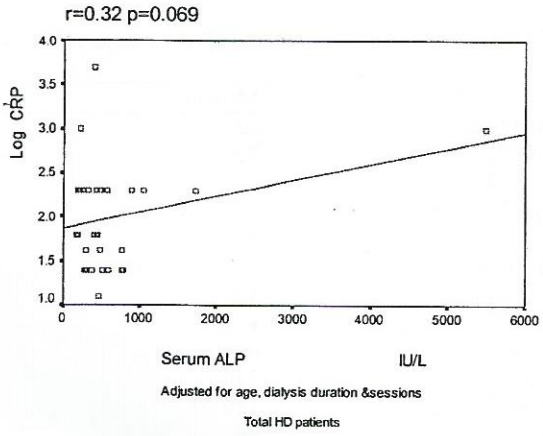



**Figure 4**: Significant positive correlation of serum phosphorus with serum CRP.

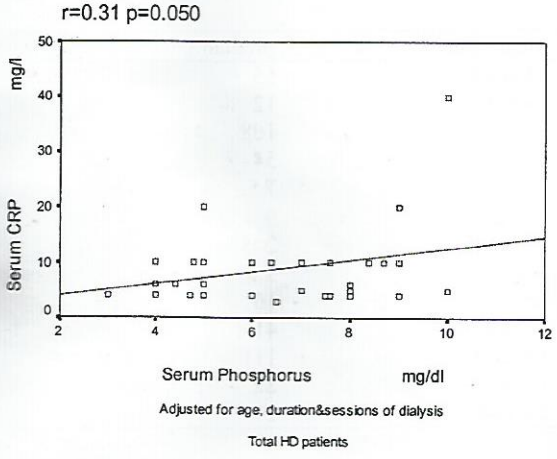



**Figure5:** Significant inverse correlation of serum phosphorus with BMI

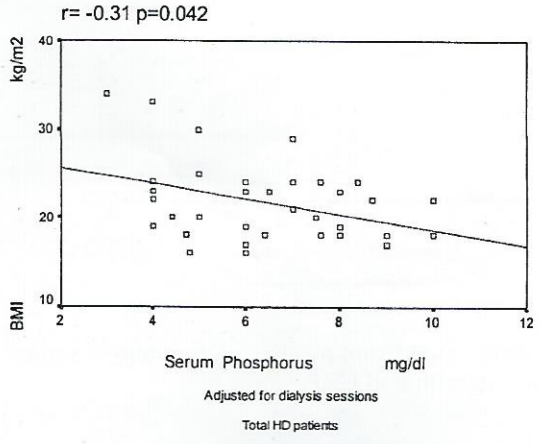
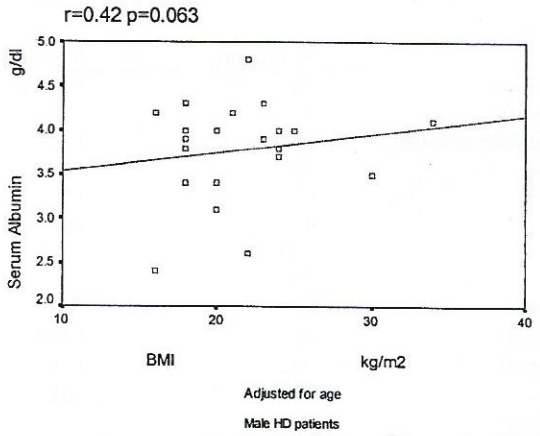



**Figure 6**: Near significant positive correlation of serum albumin with BMI




**Discussion:**

In this study we found a near significant positive correlation of serum iPTH with serum CRP, a significant inverse correlation of serum iPTH BMI and a near significant positive correlation of serum ALP with Logarithm of CRP a significant positive correlation of serum phosphorus with serum CRP and also a significant inverse correlation of serum phosphorus with BMI were found. A near significant inverse correlation of serum cholesterol with serum phosphorus as well as a near significant inverse correlation of serum cholesterol with serum CRP were existed too. We also found a significant inverse correlation of serum albumin with logarithm of CRP .Moreover a near significant positive correlation of serum albumin with BMI were shown too. Serum albumin ,cholesterol and also BMI are indexes of nutritional status in HD patients while serum CRP could show the inflammation status. PTH has long been considered a uremic toxin, with many deleterious cellular and metabolic effects[27]. It increases bone turnover[28] and induces neuropathy[29], myopathy, cardiac hypertrophy, hyperlipidemia, carbohydrate intolerance, and immune dysfunction[30,31]. Although specific studies are lacking, such conditions could influence the nutritional status of uremic patients with secondary hyperparathyroidism. Garber[32] demonstrated *in vitro* that high PTH levels enhanced muscle proteolysis and increased the release of alanine and glutamine. This effect, however, was observed only in normal rats. In a study conducted by Yasunaga et al on Thirty-four patients under dialysis therapy received a parathyroidectomy (PTx) for secondary hyperparathyroidism found that PTx had beneficial effects on humoral immunological markers. They concluded that this effect are probably due to the remarkable PTH reduction and partly improved nutritional state after PTx[33]. The nutritional and biochemical parameters of 15 chronic hemodialysis (HD) patients with severe secondary hyperparathyroidism who had undergone total parathyroidectomy (PTX), with a forearm implant, were retrospectively studied by Khajehdehi et al. at 1, 3, 6, and

12 months pre- and post-PTX. They found that in 53% of the patients, the weight gain was more than 5% above the baseline[34]. Avram et al[35] studied prospectively the relationship between the enrollment serum intact PTH and all-cause mortality in 345 hemodialysis and 277 peritoneal dialysis patients for 14 years and found that lower than expected levels of PTH in uremic patients are associated with increased mortality. Moreover, Guh et al[36] recently reported similar findings that low levels of serum PTH at entry and lower time-dependent PTH levels predict mortality in hemodialysis patients. Avram et al[35] hypothesized that inadequate protein intake, phosphorus intake or both result in impaired development of the expected secondary hyperparathyroidism and in the excess mortality risk inherent with malnutrition. However, to date epidemiologic studies have shown a positive association between a high serum phosphorus and poor outcome among ESRD patients [37]. In maintenance hemodialysis (MHD) patients, associations between demographic, clinical and laboratory values and mortality, including cardiovascular death, are significantly different and, in some cases, in the opposite direction of those derived from the general population. This phenomenon, termed 'reverse epidemiology[2,7]. Hence, the association between serum PTH and nutritional status may be bidirectional. Similar reverse epidemiologic observations have been made for serum creatinine and parathyroid hormone (PTH). These studies show that, in MHD patients, the relation between the measure and outcome is counterintuitive. The cause of the unanticipated relation between lower serum PTH and increased mortality might be explained by the malnutrition-inflammation syndrome. Low intakes of calcium, phosphorus, and protein and low serum phosphorus, which may all be associated with malnutrition or an inflammatory state (or both), may account for this relation[40,41]. Reduced intakes of these substances might lead to lower serum PTH concentrations and, directly or as a result of associated diseases, might induce higher mortality. While a 1.5 time of PTH level is necessary for bone activity in dialysis patients , the values more than this amount have deleterious effects as mentioned. While serum CRP is a marker of inflammation[38], we showed its positive correlations with serum iPTH as well as its negative correlation with BMI. Whilest the BMI is a marker of nutritional status[39]. More over positive associations of high serum phosphorus and alkaline phosphtase as markers of uncontrolled secondary hyperparathyroidism in MHPs with CRP and also negative correlation of high seum phosphorus with BMI further support the association of poorly controlled SHPTH with MICS in dialysis patients. In this regard when we deleted patients with iPTH below than 200 pg/ml we found that the correlation of iPTH with CRP was positive ($r$ =0.42, $p$ =0.085) and when we deleted patients with iPTH more than 500pg/ml we found that this correlation was negative ($r$ = -0.42, $p$ =0.047), means that a low iPTH is an index of malnutrition while higher values is associated with inflammation .Thus further attention needs to control of hyperphosphatemia and maintaining the iPTH levels 1.5 times of normal to avoid the sides effects of secondary hyperparathyroidism.

**References:**


1. Fung F, Sherrard DJ, Gillen DL, et al. Increased risk for cardiovascular mortality among malnourished end-stage renal disease patients. Am J Kidney Dis 2002;40:307-314.
2. Kalantar-Zadeh K, Kopple JD. Relative contributions of nutrition and inflammation to clinical outcome in dialysis patients. Am J Kidney Dis 2001;38:1343-1350.
3. Qureshi AR, Alvestrand A, Divino-Filho JC, et al. Inflammation, malnutrition, and cardiac disease as predictors of mortality in hemodialysis patients. J Am Soc Nephrol 2002;13:S28-S36.
4. Zimmermann J, Herrlinger S, Pruy A, Metzger T, Wanner C. Inflammation enhances cardiovascular risk and mortality in hemodialysis patients. Kidney Int 1999;55:648-658.
5. Stenvinkel P, Heimburger O, Paultre F, et al. Strong association between malnutrition, inflammation, and atherosclerosis in chronic renal failure. Kidney Int 1999;55:1899-1911.
6. Bergstrom J. Inflammation, malnutrition, cardiovascular disease and mortality in end-stage renal disease. Pol Arch Med Wewn 2000;104:641-643.
7. Kalantar-Zadeh K, Kopple JD, Block G, Humphreys MH. A malnutrition-inflammation score is correlated with morbidity and mortality in maintenance hemodialysis patients. Am J Kidney Dis 2001;38:1251-1263.
8. Pecoits-Filho R, Lindholm B, Stenvinkel P. The malnutrition, inflammation, and atherosclerosis (MIA) syndrome—The heart of the matter. Nephrol Dial Transplant 2002;17:S28-S31.
9. Bergström J & Lindholm B (1993). Nutrition and adequacy of dialysis: How do hemodialysis and CAPD compare? Kidney International, 43 (Suppl 40): 39-50.
10. Cuppari L, Medeiros FAM, Pappini HF, Cendorolo Neto M, Canziani MEF, Martini L, Ajzen H & Draibe SA (1994). Effectiveness of oral energy-protein supplementation in severely malnourished hemodialysis patients. Journal of Renal Nutrition, 4 (Suppl 3): 127-135.
11. Degoulet P, Reach L, Aimé F, Rioux P, Jacobs C & Legrain M (1980). Risk factors in chronic hemodialysis. Proceedings of the European Dialysis and Transplant Association, 17: 149-154.
12. Lowrie EG & Lew NL (1990). Death risk in hemodialysis patients: the predictive values of commonly measured variables and an evaluation of death rate differences between facilities. American Journal of Kidney Diseases, 15 (Suppl 5): 458-482.
13. Acchiardo SR, Moore LW & Latour PA (1983). Malnutrition as the main factor in morbidity and mortality of hemodialysis patients. Kidney International, 24 (Suppl 16): 199-203.
14. Cuppari L, Draibe AS, Anção MS, Sigulem D, Sustovic DR, Ajzen H & Ramos OL (1989). Avaliação nutricional de pacientes renais crônicos em programa de hemodiálise. Estudo multicêntrico. Revista da Associação Médica Brasileira, 35 (Suppl 1): 9-14.

15. Shoenfeld P, Henry RR, Laird NM & Roxe DM (1983). Assessment of nutritional status of the national cooperative dialysis study population. Kidney International, 23 (Suppl 13): 80-88.
16. Kopple JD, Swendseid ME, Shinaberger JH & Umezawa CY (1973). The free and bound amino acids removed by hemodialysis. Transactions of the American Society for Artificial Internal Organs, 19: 309-313.
17. Gutierrez A, Alvestrand A, Wahren J & Bergstrom J (1990). Effect of in vivo contact between blood and dialysis membranes on protein catabolism in humans. Kidney International, 38: 487-494.
18. Bergström J (1995). Why are dialysis patients malnourished? American Journal of Kidney Diseases, 26 (Suppl 1): 229-241.
19. Mitch WE, Medina R, Grieber S, May RC, England BK, Price SR, Bailey JL & Goldberg AL (1994). Metabolic acidosis stimulates muscle protein degradation by activating the adenosine triphosphate-dependent pathway involving ubiquitin and proteasomes. Journal of Clinical Investigation, 93: 2127-2133.
20. Akmal M, Massry SG, Goldstein DA, Fanti P, Weisz A & DeFronzo R A ( 1985). R ole of p arathyroid h ormone i n t he glucose intolerance of chronic renal failure. Journal of Clinical Investigation, 75: 1037-1044.
21. Luger A, Kovarik J & Stummvoll HK (1987). Blood-membrane interaction in hemodialysis leads to increased cytokine production. Kidney International, 32: 84-88.
22. Frame B, Heinze Jr EG, Block MA & Manson GA (1968). Myopathy in primary hyperparathyroidism. Annals of Internal Medicine, 68: 1022-1027.
23. Patten BM, Bilezikian JP, Mallete LE, Prince A, Engel WK & Aurbach G H ( 1974). N euromuscular d isease i n p rimary hyperparathyroidism. Annals of Internal Medicine, 80: 182-193.
24. Kopple JD, Cianciaruso B & Massry SG (1980). Does parathyroid hormone cause protein wasting? Contributions to Nephrology, 20: 138-148.
25. http://www.halls.md/body-mass-index/av.htm.
26. Boag JT. Basic Truths in Optimal Hemodialysis, Dialysis & Transplantation, Volume 23, Number 11, November 1994 Page 636.
27. Feinfeld DA (1992). The role of parathyroid hormone as a uremic toxin: current concepts. Seminars in Dialysis, 5 (Suppl 1): 48-53.
28. Kronemberg HM (1993). Parathyroid hormone: mechanism of action. In: Favus M J (Editor), P rimer on the Metabolic Bone Diseases and Disorders of Mineral Metabolism. Lippincott-Raven, Philadelphia, 58-60.
29. Goldstein DA, Chui LA & Massry SG (1978). Effect of parathyroid hormone and uremia on peripheral nerve calcium and motor nerve conduction velocity. Journal of Clinical Investigation, 62: 88-93.
30. Rezende L.T.T, Cuppari L, Carvalho A.B, Canziani M.E.F, Manfredi S.R, Cendoroglo M. Nutritional status of hemodialysis p atients w ith s econdary h yperparathyroidism. Braz J Med Biol Res, November 2000, Volume 33(11) 1305-1311.
31. Nasri H, Baradaran A, Naderi AS. Close association between parathyroid hormone and left ventricular function and structure in end-stage renal failure patients under maintenance hemodialysis.Acta Med Austriaca. 2004 Aug;31(3):67-72.
32. Garber AJ (1983). Effect of parathyroid hormone on skeletal muscle protein and amino acid metabolism in the rat. Journal of Clinical Investigation, 71: 1806-1821.
33. Yasunaga C. Nakamato M. Matsuo K. Nishihara G. Yoshida T.Goya T. Effects of a parathyroidectomy on the immune system and nutritional condition in chronic dialysis patients with secondary hyperparathyroidism. Am J Surg. 1999 Oct;178(4):332-6.
34. Khajehdehi p, Ali M, Al-Gebory F, Henry G, Bastani B. The effects of parathyroidectomy on nutritional and biochemical status of hemodialysis patients with severe secondary hyperparathyroidism. J Ren Nutr. 1999 Oct;9(4):186-91.
35. Avram MM, Mittman N, Myint MM, Fein P: Importance of low serum intact parathyroid hormone as a predictor of mortality in hemodialysis and peritoneal dialysis patients: 14 years of prospective observation. Am J Kidney Dis 38: 1351-1357, 2001.
36. Guh JY, Chen HC, Chuang HY, et al: Risk factors and risk for mortality of mild hypoparathyroidism in hemodialysis patients. Am J Kidney Dis 39: 1245-1254, 2002.
37. Levin NW, Hoenich NA: Consequences of hyperphosphatemia and elevated levels of the calcium-phosphorus product in dialysis patients. Curr Opin Nephrol Hypertens 10: 563-568, 2001.
38. Bárány P.Inflammation, serum C-reactive protein, and erythropoietin resistance. Nephrol Dial Transplant (2001) 16: 224-227.
39. Kalantar-Zadeh K .Causes a nd c onsequences o f t he r everse epidemiology of body mass index in dialysis patients. J Ren Nutr. 2005 Jan;15(1):142-7.
40. Mehrotra R, Kopple JD. Causes of protein-energy malnutrition in chronic renal failure. In: Kopple JD, Massry SG, eds. Nutritional management of renal disease. 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2004:167–82.
41. Kalantar-Zadeh K, Kopple JD. Nutritional management of patients undergoing maintenance hemodialysis. In: Kopple JD, Massry SG, eds. Nutritional management of renal disease. 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2004:433–66.