

Prevention of Renal Osteodystrophy in Primary Care



Ibraim Rudhani, Naim Morina*, Marigonë Zubaku and Ahmet Avdullahu

Medicinal Faculty, University of Prishtina, Kosovo and University Clinical Center of Kosovo

Received: June 20, 2018; Published: July 03, 2018

*Corresponding author: Naim Morina, Specialist of Internal Medicine, Clinic for Nephrology and Hemodialysis UCCK Kosovo, Hospital Circle, Kosovo

Abstract

Introduction: Renal Osteodystrophy is a skeletal change most commonly caused by chronic renal resulting diseases. It is associated with hyperphosphatemia, hypocalcemia, increased secretion of parathyroid hormone, metabolic acidosis and decreased vitamin D activity, which results in skeletal changes.

Purpose of the Research: notification of pathologies of renal osteodystrophy and analysis of collected data.

Materials and Methods: The research was retrospective, conducted at the Hemodialysis Department of Nephrology Clinic, QKUK Prishtina. Part of this study was 89 patients selected in regular hemodialysis sessions. The age of patients ranges from 25 to 80 years, with an average of 57 years. The study was conducted through the collection of data from laboratory documentation of patients who suffered from terminal renal insufficiency.

Results: Out of 89 patients, 42 (47.19%) were women, 47 (52.81%) were males. The most vulnerable age group was 60-69 years old (47.19%), 44 patients with adult phosphorus, 4 patients with decreased phosphorus, 5 were with calcium, 11 with decreased calcium, 5 patients with phosphorus and adult calcium, 1 patient with phosphorus and decreased calcium and 14 patients with increased phosphorus and decreased calcium.

Conclusion: Renal osteodystrophy affects more about 60-69 years of age, while the most affected gender is female and has changes in calcium and phosphorus laboratory values. The most manifested manifestations of both sexes are accompanied by changes in bone remodeling.

Keywords: Renal Osteodystrophy; Chronic Renal Insufficiency

Introduction

Renal osteodystrophy refers to skeletal changes that result from chronic renal disease and are caused by disorders in calcium and phosphorus metabolism, abnormal vitamin D metabolism and increased thyroid gland activity. In early stages of renal insufficiency, intestinal calcium absorption is reduced because the kidneys are unable to convert vitamin D to its active form 1,25 dihydroxycalciferol [1-3]. Thus, with the breakdown of the active form of vitamin D, calcium metabolism is regulated, which is the most biologically active form in the absorption of calcium from the digestive tract. Damaged absorption of calcium occur due to impaired kidney function and the corresponding phosphatase retention due to lower serum calcium percentage. This hypocalcemia is associated with parathyroid gland compensator hyperactivity that increases phosphate excretion in urine, decreases calcium excretion in the urine, and regulates and promotes the release of calcium from the bone. The most frequent changes observed in association with compensatory hyperparathyroidism are those that affect and include the skeletal system. Bone changes are associated with alteration of bone remodeling, osteomalation,

osteocytic cystic fibrosis (osteotomy associated with fibrotic degeneration and cystic regions resulting from parathyroid gland hyperfunction) and osteosclerosis. Bone lesions usually appear on the fingers, the clavicle and the acromioclavicular articulation. Other lesions that can be observed are found in the skull, distal clavicle erosion and pubic symphysis margins, rib fractures, and femoral head necrosis [4-8]. In children as predominant lesion is osteomalacia which is associated with bone mitigation, leading to rib and pelvic deformities. Early renal osteodystrophic stages can be detected histologically or biochemically without the presence of distinct radiographic changes because radiographic data of bone disease appear only if 30% of the bone elements are lost [9,10].

Research

Purpose of the Work

The purpose of this paper is to inform about the most common pathologies that lead to the appearance of renal osteodystrophy. Especially the analysis of the data collected by gender, age in patients with renal osteodystrophy and research of calcium and

phosphorus values in these patients. Short description or brief description of the aforementioned pathologies (etiology, clinical features, diagnostics, treatment) [11-15].

Research Methods

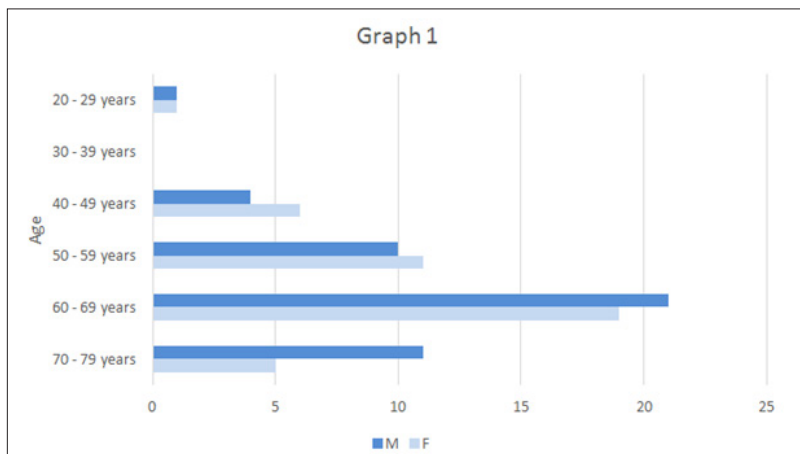
The research method is retrospective study. Part of this research was 89 patients selected at regular hemodialysis session. The age of patients ranges from 25 to 80 years, with an average of 57 years. The site of the research was the Clinic of Nephrology-Hemodialysis Department, QKUK Prishtina. The study was conducted between collecting data from laboratory records of patients who suffered from terminal renal failure and who were in regular Hemodialysis sessions from 02.01.2017 to 30 June 2017 (retrospective study) [15-18].

Procedure

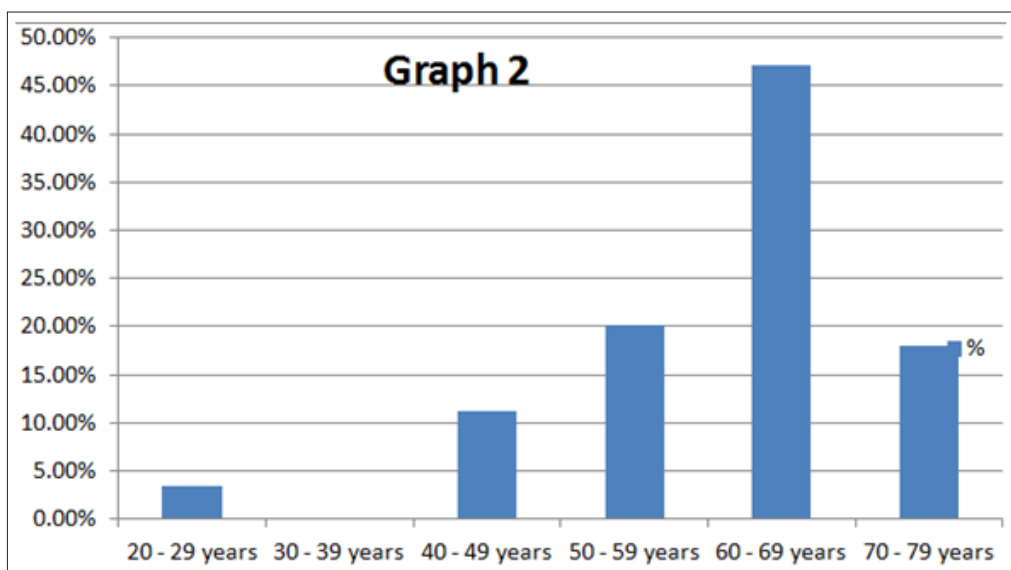
For this research, a permission from the director of the Nephrology Clinic was initially granted. The data collection was made from 02 January 2014 to 30 June 2017. The data is processed in Excel and are presented through tables and graphs [19-24].

Results

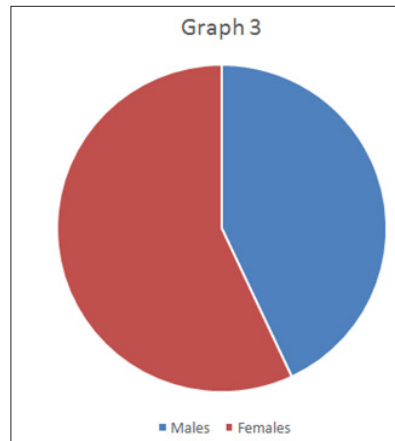
Presentation of the results begins with the data of the patients grouped according to age, gender, calcium and phosphorus values and the frequency of occurrence of diseases in the hemodialysis unit at QKUK (Table 1). In the diagram we see that the age most affected in the hemodialysis ward at UCCK from the total number of samples was 60-69 years old and the more attacked were the males (Graph 1) and (Table 2). From the diagram we find that the most frequent age in patients with renal osteodistropia in the hemodialysis ward at QKUK from the total number of samples is 60-69 years (47.19%) (Graph 2) and (Table 3). From the diagram we can see that the most affected gender of the patient with renal osteodystrophy in the dialysis ward at UCCK from the total number of samples was female by 57% (Graph 3) and (Table 4). From the diagram we can see that the phosphorus values have been the highest and the most stressed was the male gender, while the values of decreased calcium and increased phosphorus were the most affected by female gender (Graph 4).



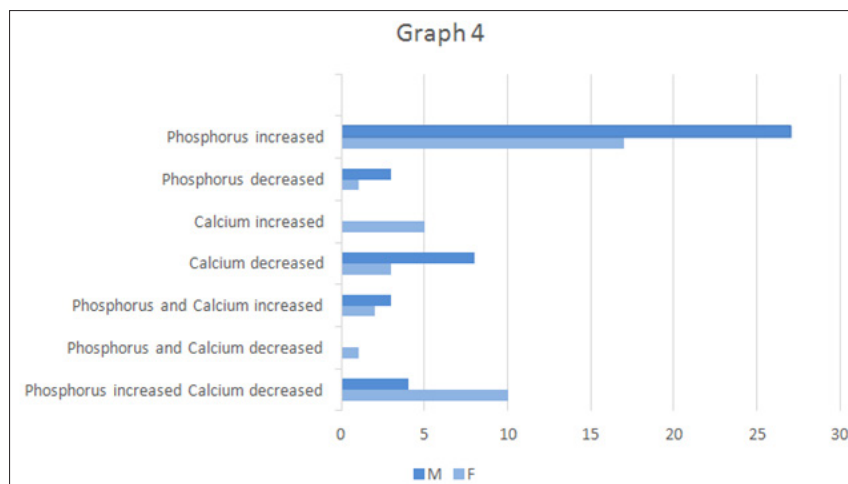
Graph 1: Graphic presentation of patients by age and gender.



Graph 2: Graphic presentation of patients with renal osteodistropia - terminal renal insufficiency.



Graph 3: Graphic representation of the number of patients with renal osteodistrophies by sex.



Graph 4: Graphic representation of phosphorus and calcium values by gender.

Table 1: Total number of patients by gender and age.

N = 89	n	%	n	%
Age	F		M	
20 - 29 years	1	2.38%	1	2.12%
30 - 39 years	0	0%	0	0%
40 - 49 years	6	14.28%	4	8.51%
50 - 59 years	11	26.19%	10	21.27%
60 - 69 years	19	45.23%	21	44.68%
70 - 79 years	5	11.90%	11	23.40%
Total	42	47.19%	47	52.81%

Table 2: Introduction of renal osteodystrophy - terminal renal insufficiency by age.

Age	n	%
20 - 29	3	3.37%
30 - 39	0	0%
40 - 49	10	11.23%
50 - 59	18	20.22%
60 - 69	42	47.19%
70 - 79	16	11.97%
Total	89	100%

Table 3: Presentation of patients with renal osteodistrophies by gender.

Gender	n	%
Male	38	43%
Female	51	57%
Total	89	100%

Table 4: Total number of patients based on the values of phosphorus and calcium by gender.

Gender	Phosphorus Increased	Phosphorus Decreased	Calcium Increased	Calcium Decreased	Phosphorus and Calcium Increased	Phosphorus and Calcium Decreased	Phosphorus Increased Calcium Decreased
F	17	1	5	3	2	1	10
M	27	3	0	8	3	0	4
Total	44	4	5	11	5	1	14

Discussion

During the period January-June 2017, 89 patients were screened for sample. In the total number of patients the age and sex frequency was investigated, the most affected age was 60-69 years, and the most common male gender. Over age-related research in patients with renal osteodystrophy, the most affected age was 60-69 years of age [25-27]. Based on the appearance of gender in renal osteodystoma patients, the most affected gender was female. If we compare with the state of Germany, then we see that the most common pathology of renal osteodystrophy is chronic renal failure. According to the statistics of 2016, we understand that from the diagnosed cases the most common age was 70-79 years old and the most affected gender was female [28-30]. <https://www.aerzteblatt.de/int/archive/article/174773>

Conclusion

The research carried out has shown that the pathology of renal osteodystrophy is terminal chronic insufficiency, with regard to age, the most affected age was around 60-69 years, the most commonly invasive gender among patients with renal osteodystrophy was female gender. The research also includes age-related incidence based on changes in phosphorus and calcium levels. As evidenced by statistics, care of early diagnosis of terminal renal disease, proper treatment and prevention, preclude the appearance of renal osteodystrophy.

References

- Vleming LJ, Brujlin JA, Van Es LA (1999) The pathogenesis of progressive renal failure. *Neth J Med* 54(3): 114-128.
- Shkoza A *Fiziologija Njeriut* pp. 681-683, 688.
- Gamulin S, Matko M, Kervavica S *Fispatologija* pp. 237.
- Gamulin S, Matko M, Kervavica S, *Fispatologija* pp. 246.
- Gamulin S, Matko M, Kervavica S, *Fispatologija* pp. 356.
- Almaden Y, Canalejo A, Hernandez A, Ballesteros E, Garcia Navarro S, et al. (1996) Direct effect of phosphorus on parathyroid hormone secretion from whole rat parathyroid glands in vivo. *Journal of Bone and Mineral Research* 11(7): 970-976.
- Brown AJ, Zhong M, Finch J, Ritter C, McCracken R, et al. (1996) Rat calcium-sensing receptor is regulated by vitamin D but not by calcium. *American Journal of Physiology* 270(3): 454-460.
- Lucas PA, Brown RC, Woodhead JS (1986) Acute responses of parathyroid hormone and 1,25 dihydroxyvitamin D3 to unilateral nephrectomy in healthy donors. *Nephrolog Dialysis Transplantation* 1(3): 199-203.
- Dunstan CR, Hills E, Norman AW, Bishop JE, Mayer E, et al. (1985) The pathogenesis of renal osteodystrophy: role of vitamin D, aluminium, parathyroid hormone, calcium and phosphorus. *The Quarterly Journal of Medicine* 55(217): 127-144.
- John MR, Goodman WG, Gao P, Cantor TL, Salusky IB, et al. Implications for PTH measurements in renal failure. *Journal of Clinic Endocrinology and Metabolism*.
- Lefebvre A, Vernejoul MCD, Gueris J, Goldfarb B, Graulet AM, et al. (1989) Optimal correction of acidosis changes progression of dialysis osteodystrophy. *Kidney International* 36(6): 112-1118.
- Massry SG (1987) Parathyroid hormone L a uremic toxin. *Advances in Experimental Medicine and Biology* p. 1-17.
- Massry SG, Garty J, Arieff AI, Coburn JW (1976) Skeletal resistance to the calcemic action of parathyroid hormone in uremia; role of 1,25(OH) D3 *Kidney International* 9(6): 467-474.
- Lucas PA, Woodhead JS, Brown RC (1988) Vitamin D3 metabolites in chronic renal failure and after renal transplantation. *Nephrology, Dialysis, Transplantation* 3(1): 70-76.
- Mitalk BH, Alpert M, Lo C, Delmonico F, Neer RM (1991) Parathyroid function in normocalcemic renal transplant recipients: evaluation by calcium infusion. *Journal of Clinical Endocrinology and Metabolism* 72(2): 350-355.
- Julian BA, Faugere MC, Malluche HH (1987) Oxalosis in bone causing a radiographic mimicry of renal osteodystrophy. *American Journal of Kidney Diseases* 9(5): 436-440.
- Llach T, Keshav G, Goldblat MV, Lindberg JS, Sadler R, et al. (1998) Suppression of parathyroid hormone secretion in hemodialysis patients by a novel vitamin D analogue. *American Journal of Kidney Diseases* 32(2): 48-54.
- Angelis M, Wong LL, Myers SA, Wong LM (1997) Calciphylaxis in patients on hemodialysis: A prevalence study 122(6): 1083-1090.
- Rostand SG, Thornley Brown D Soft tissue calcification in chronic renal failure. In the *Spectrum of Renal Osteodystrophy*.
- Luo G, Ducey P, McKee MD, Pinero GJ, Loyer E, et al. (1997) Spontaneous calcification of arteries and cartilage in mice lacking matrix GLA protein. *Nature* 386(6620): 78-81.
- Kurz P, Monier Faugere MC, Bognar B, Werner E, Roth P, et al. (1994) Evidence for abnormal calcium homeostasis in patients with adynamic bone disease. *Kidney International* 46(3): 855-861.
- Hutchinson AJ, Whitehouse RW, Boulton HF, Adams JE, Mawer EB, et al. (1993) Correlation of bone histology with parathyroid hormone,

- vitamin D3 and radiology in end-stage renal diseases. *Kidney International* 44(5): 1071-1077.
23. Nelson SR, Hawkins PN, Richardson S, Lavender JP, Sethi D, et al. (1991) Imaging of hemodialysis-associated amyloidosis using 123-I serum amyloid P component. *Lancet* 338(8763): 335-339.
24. Tominaga Y, Numano M, Tanaka Y, Uchida K, Takagi H (1997) Surgical treatment of renal hyperparathyroidism. *Seminars in Surgical Oncology* 13(2): 87-96.
25. Casas AT, Burke GJ, Sathyanarayana, Mansberger AR, Wei JR (1993) Prospective comparison of technetium-99m-sestamibi/iodine-123 radionuclide scan versus high-resolution ultrasonography for the preoperative localization of abnormal parathyroid glands in patients with previously unoperated primary hyperparathyroidism. *American Journal of Surgery* 166(4): 369-373.
26. Arnold A, Brown MF, Urena P, Gaz RD, Sarfati E, et al. (1995) Monoclonality of parathyroid tumors in chronic renal failure and in primary parathyroid hyperplasia. *Journal of Clinical Investigation* 95(5): 2047-2053.
27. Baker LR, Abrams L, Roe CJ, Faugere MC, Fanti P, et al. (1989) 1,25 (OH) D3 administration in moderate renal failure; a prospective double blind trial. *Kidney International* 35(2): 661-669.
28. Sheik MS, Maguire JA, Emmett M, Ana CAS, Schiller LR (1998) Reduction of dietary phosphorus absorption by phosphorus binders. A theoretical, *in vitro*, and *in vivo* study. *Journal of Clinical Investigation* 83(1): 66-73.
29. Klaus G, Mehls O, Ogata E, Ritz E (1991) Is intermittent oral calcitriol safe and effective in renal secondary hyperparathyroidism 337(8744): 800-801.
30. Bleyer AJ (1999) A comparison of the calcium free phosphate binder sevelamer hydrochloride with calcium acetate in the treatment of hyperphosphatemia in hemodialysis patient. *American Journal of Kidney International* 33(4): 694-701.



This work is licensed under Creative Commons Attribution 4.0 License

Submission Link: <https://biomedres.us/submit-manuscript.php>



Assets of Publishing with us

- Global archiving of articles
- Immediate, unrestricted online access
- Rigorous Peer Review Process
- Authors Retain Copyrights
- Unique DOI for all articles

<https://biomedres.us/>