Acta Scientific Gastrointestinal Disorders (ISSN: 2582-1091)

Volume 6 Issue 3 March 2022

Research Article

Features of Calcium Homeostasis Among Patients with Malabsorption Syndrome on the Background of Chronic Kidney Disease

Zub LO, Shkarutyak AY and Buzdugan IO*

Department of Inter Medicine, Bukovinian State Medical University, Ukraine

***Corresponding Author:** Buzdugan IO, Department of Inter medicine, Bukovinian State Medical University, Ukraine.

Received: April 25, 2022

Published: February 15, 2022

Abstract

Today, data on the role of factors of non-microbial etiology that form the combined pathology of the kidneys and gastrointestinal tract, remain scarce. Calcium homeostasis, which according to the medical literature plays an important role in the progression of renal pathology, especially when it is impaired among patients with malabsorption syndrome due to chronic kidney disease (CKD).

The Aim: The research was to study the processes of calcium homeostasis among patients with malabsorption syndrome on the background of chronic kidney disease.

Materials and Methods: 99 patients with malabsorption syndrome (MAS) on the background of CKD were examined. Patients were divided into groups: Group I (25 people) - Stages 1 and 2 of CKD without MAS; Group II (26) - Stages 1 and 2 of CKD with MAS; Group III (23)- Stage 3 of CKD without MAS; Group IV (25) - Stage 3 of CKD with MAS.

According to the morphological study of in vivo biopsies of the small intestinal mucosa, mild and moderate morphological changes were observed among all patients. The level of calcium and phosphorus in the blood, as well as calcium in the urine were detected. At the same time, the level of calcium-regulating hormones in the blood - parathyroid hormone and calcitonin - was studied.

The Results of the Research: Pathological changes in calcium metabolism were observed among patients with malabsorption syndrome. The severity of these disorders was more evident among patients with Stage 3of CKD. Calcium levels in daily urine were reduced in groups III and IV. No changes were detected in phosphorus metabolism. Changes in parathyroid hormone and osteocalcin are caused primarily by combined renal pathology with impaired renal calcium absorption.

Conclusion: Patients with malabsorption syndrome on the background of CKD revealed deeper violations of calcium homeostasis, which can lead to rapid progression of this combined pathology involvinga bone tissue in the pathological process.

Keywords: Malabsorption Syndrome; Chronic Kidney Disease; Calcium

Introduction

Nowadays, the great importance of metabolic disorders and chronic intestinal diseases in the formation of combined pathology of the kidneys and digestive organs has been proven [1-6]. At the same timethere are no data on the role of factors of non-microbial etiology that form the combined pathology of the kidneys and digestive organs.

Malabsorption syndrome (MAS) combines all types of pathology caused by indigestion or absorption. Among the huge range of diseases with impaired intestinal absorption syndrome, the most common in therapeutic practice are lactase deficiency, exudative enteropathy, food allergy, Crohn's disease, nonspecific ulcerative colitis, helminthic invasion, chronic pancreatitis [7,8]. So far, data on the role of factors of non-microbial etiology, that form a combined

pathology of the kidneys and gastrointestinal tract are small. There is no well-developed program for early diagnosis, prevention of development and progression of this pathology [9-11].

Calcium homeostasis, as proven in medical literature, plays crucial role in the progression of renal pathology [17-19], especially when it is impaired among patients with malabsorption syndrome [12-16]on the background of chronic kidney disease (CKD).

The aim of this research was to study the processes of calcium homeostasis among patients with malabsorption syndrome on the background of chronic kidney disease.

Materials and Methods

99 patients with malabsorption syndrome on the background of CKD were examined. There were 88 women and 11 men aged 52.5 ± 8.5 years. In most of the examined patients the cause of malabsorption syndrome was chronic pancreatitis [2]. 2 patients had nonspecific ulcerative colitis. 1 patient had Crohn's disease. The reason for the CKD development among the studied patients was chronic pyelonephritis and dysmetabolic nephropathy. Patients were divided into groups: Group I (25 people) - Stages 1 and 2 of CKD without MAS; Group II (26) - Stages 1 and 2 of CKD with MAS; Group III (23) - Stage 3 of CKD without MAS; Group IV (25) - Stage 3 of CKD with MAS. Also, 20 healthy individuals of the appropriate age were examined. Blood calcium and phosphorus levels,renal excretion in the urine were detected using standard kits.At the same time, the level of calcium-regulating hormones in the blood - parathyroid hormone and calcitonin - was examined by enzyme-linked immunosorbent assay using standard kits, which allowed to assess the hormonal effect (secondary hyperparathyroidism) on phosphorus-calcium metabolism.

According to the morphological study of *in vivo* biopsies of the small intestinal mucosa, mild and moderate morphological changes were observed among all patients.

Crypt deepening decreased small intestinal villus height, (without atrophy), the change in the length of villi and crypt depth correlation, the increase in the number of lymphohistiocytic and plasma cells in the plate, change in enterocytes were typical morphological signs of the moderate severity of the process.

The level of non-collagenous osteocalcin protein, which is a marker of osteoporosis and plays an important role in the formation of the organic matrix of bone tissue was determined to evaluate the activity of osteoporosis and bone loss.

Enzyme-linked immunosorbent assay using standard kit was applied to study the level of osteocalcin in the serum. All patients were also examined according to the clinical protocol of nephrological patients.

The obtained material was processed statistically using Student's t-tests. All indicators are presented as averages with their standard deviation (M ± m). Deviation at $p < 0.05$ were considered credible. The research was conducted in accordance with the ethical principles of the Declaration of Helsinki revised in 2008.

The Results of the Research

The study showed that all patients with MAS on the background of CKD had severe calcium disorders. Hypocalcemia was observed in the group of patients with Stage 1 and 2 of CKD with MAS ($p < 0.05$). In Group I, the level of calcium probably did not differ from the norm ($p > 0.05$). Patients with Stage 3 of CKD without MAS had low calcium levels ($p < 0.05$), which is probably related to impaired calcium reabsorption, due to significant renal impairment. This indicator was significantly reduced ($p < 0.05$) among patients with Stage 3 of CKD with MAS, which is explained both by disturbance of reabsorption processes, and disturbance of absorption. Changes in parathormone and osteocalcin levels were found among patients from Group IV ($p < 0.05$). Some symptoms of osteoporosis were observed among these patients during an x-ray examination. Calcium levels in daily urine were reduced among patients from Groups III and IV ($p < 0.05$), which indicates a violation of filtration processes in Stage 3 of CKD. The level of calcitonin among all patients probably did not change ($p > 0.05$). Indicators of inorganic phosphorus also did not change in all groups of patients ($p > 0.05$) (Table 1).

It should also be noted that patients with morphologically severe changes in the intestinal mucosa had lower calcium levels (Table 2).

Indexes	Patient group				
	Healthy (20 people)	Group I (25 people)	Group II (26 people)	Group III (23 people)	Group IV (25 people)
Blood calcium (mmol/l)	2,20 ± 0,6	2,25 ± 0,05	1,85 ± 0,02*	1,82 ± 0,01*	1,80 ± 0,03*
Urine calcium(mmol/day)	4,25 ± 2,34	3,95 ± 1,37	4,01 ± 1,98	1,04 ± 0,9*	0,97 ± 0,88*
Phosphorus (mmol/l)	0,81 ± 0,99	0,81 ± 0,04	0,82 ± 0,33	0,88 ± 0,21	0,93 ± 0,11
Osteocalcin (ng/l)	25,4 ± 61,98	72,23 ± 4,28	73,03 ± 1,11	78,87 ± 1,16	98,23 ± 0,14*
Parathyroid hormone (pg/ml)	9,85 ± 66,94	45,38 ± 10,11	59,52 ± 9,23	51,99 ± 8,65	91,56 ± 9,11*
Calcitonin (pg/ml)	7,22 ± 11,91	8,68 ± 2,12	8,02 ± 2,34	7,99 ± 3,02	8,71 ± 2,86

Table 1: Characteristics of calcium, phosphorus and osteometabolism hormones among patients with MAS on the background of chronic kidney disease.

Notes: * - probability in comparison with the group of healthy people.

Patient group	Severity of morphological lesion complexity	
	Mild	Moderate
Calcium (mmol/l)	2,22 ± 0,05	2,16 ± 0,04
Phosphorus (mmol/l)	1,45 ± 0,05	1,20 ± 0,04

Table 2: Comparative characteristics of calcium and phosphate indicators depending on the severity of morphological lesions.

Conclusion

Therefore, the results of the studies showed that patients with MAS had pathological changes in calcium metabolism. The severity of these disorders was higher among patients with Stage 3 of CKD. No changes in phosphorus metabolism were found in any of the patient groups. Changes in parathyroid hormone and osteocalcin with some manifestations of osteoporosis, which was confirmed radiologically, in our opinion, are primarily due to combined renal pathology with impaired renal calcium absorption.

Thus, deeper violations of calcium homeostasis, which can cause rapid progression of this combined pathology, were found among patients with malabsorption syndrome on the background of CKD.

Bibliography

1. Dubrovskaya MI., *et al.* "Malabsorption Syndrome. Ambulance Clinical Events". *Voprosy Sovremennoi Pediatric* 3 (2015): 402-407.

2. Nagornaya NV and Limarenko ÌP. "Pancreatic exocrine function and methods of its evaluation". *Zdorov'e Rebenka* (2012): 118-121.

3. Dudka RV. "Digestive status in patients with complicated forms of chronic pancreatitis". *Medical Perspectives* 1 (2004): 57-60.

4. Misselwitz B., *et al.* "Lactose malabsorption and intolerance: pathogenesis, diagnosis and treatment". *United European Gastroenterology Journal* 1 (2013): 151-159.

5. Lacy BE., *et al.* "Bowel Disorders". *Gastroenterology* 150 (2016): 1393-1407.

6. Neiko EM. "Editor". *Internal Medicine. Maldigestion and Malabsorption Syndrome* (2009).

7. Schiller LR., *et al.* "APDW/WCOG Shanghaiworking party report: chronicdiarrhea-definition, classification, diagnosis". *Journal of Gastroenterology and Hepatology* 29 (2014): 6-25.

8. Schiller LR., *et al.* "Chronic Diarrhea: Diagnosis and Management". *Clinical Gastroenterology and Hepatology* 15 (2017): 182-193.

9. Klyaritskaya IL and Rabotjagova YS. "New risk factors developing chronic pancreatitis". *Crimean Therapeutic Journal* 2 (2012): 1-7.

10. Anokhina GA., *et al.* "Chronic pancreatitis, comorbid with diseases of the small intestine. Which enzyme preparation to choose?" *Health of Ukraine of the 21st Century* 22 (2021): 36-37.

11. Babinets LS and Nazarchuk NV. "Pancreas, chronic pancreatitis and trophological insufficiency: etiological, pathogenetic and clinical aspects". *Health of Ukraine. Gastroenterology. Hepatology. Coloproctology* (2015): 5.

12. Klimov LYA., *et al.* "Hormonal-metabolic patterns disorders of bone tissue mineralization in children with celiac disease". *Medical Advice* 1 (2017): 149-154.

13. Milehina SA and Klimkina TN. "The condition of the phoshpor-calcium exchange at children with caries". *Pacific Medical Journal* 3 (2014): 59-62.

14. Viun TI and Pasiyeshvili ILM. "Diagnostic role of biochemical markers of bone metabolism and FDPS gene in the evaluation of secondary osteoporosis in patients with chronic pancreatitis and hypertensive disease". *Science and Healthcare* 4 (2018): 3-17.

15. Golovach IYU. "Disorders of Bone Mineral Density and Secondary Osteoporosis in Pathology of Hepatobiliary System and Gastrointestinal Tract: at the Crossing of Problems". *Pain. Joints. Spine* 3 (2012): 49-53.

16. Pasiyeshvili LM. "Role of chronic pancreatitis in impairment of bone metabolism and development of osteoporosis". *Current Issues of Modern Medicine: Bulletin of the Ukrainian Dental Academy* 4 (2016): 167-170.

17. Ivanov DD. "Mineral Metabolism in Chronic Kidney Disease". *Kidneys* 2 (2012): 58-65.

18. Ivanov DD. "Lectures on Nephrology". Donetsk (2010).

19. Jama S and Spiegel DM. "Bone-Intestinal-Vascular-Renal Axis: Modeling and Managing Calcium and Phosphorus Disturbances in Chronic Kidney Disease". *CME/CE* (2012).