v. 2, n. 11, 2022

Journal of Agricultural Sciences Research

SECONDARY RENAL HYPERPARATHYROIDISM IN DOGS

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INTRODUCTION

It is known that the kidneys play a fundamental role in the maintenance of homeostasis. Any damage to their functions causes serious damage to health2. A great example can be seen in chronic kidney disease (CKD), in which there is continuous loss of functional nephrons, leading to deterioration of renal function. CKD is characterized by its polysystemic involvement, including metabolic alterations that corroborate the progression of the disease itself4,5. Renal secondary hyperparathyroidism (SRPH) is an example5.

The objective of the present study is to elucidate the pathophysiology, diagnosis and treatment of RHPT, as well as to clarify its interference in calcium and phosphorus metabolism, deterioration of bone and renal tissue and consequent rise of CKD.

MATERIAL AND METHODS

To prepare this summary, national and international articles and journals extracted from virtual databases such as Scholar Google and Scielo were used, mainly using the keywords: secondary renal hyperparathyroidism, chronic kidney disease, hyperphosphatemia and parathyroid hormone. Copies of books related to the topic were also used.

RELITERATURE VIEW

From stage III of CKD onwards, hyperphosphatemia is frequently observed, which occurs due to a lower renal elimination of phosphorus.1,3,4,5,7

Hyperphosphatemia favors ionic hypocalcemia, mainly due to three factors: formation of serum complexes between calcium and phosphorus, explained by the law of mass equation; decreased production of calcitriol in the kidneys by inhibiting enzyme 1α-hydroxylase (a key enzyme in the process of calcitriol formation) and by stimulating the production of fibroblast growth factor 23, a hormone produced by osteoblasts and osteocytes, which also inhibits the enzyme1α-hydroxylase and consequently inhibits the production of calcitriol.5

It is known that calcitriol, in addition to stimulating the intestinal absorption of calcium, regulates the production of parathyroid hormone by the parathyroid glands and also favors the action of parathyroid hormone in the bones. Thus, the low concentration of calcitriol is one of the factors responsible for ionic hypocalcemia. 4.5

The parathyroid glands are primarily responsible for regulating the serum concentration of calcium and phosphorus through the action of parathyroid hormone (PTH). PTH acts with the aim of increasing the serum calcium concentration, through bone resorption and calcium reabsorption in the distal convoluted tubules, and aims to reduce the serum phosphorus concentration by reducing the renal reabsorption. The main stimulus for its secretion is hypocalcemia, and for its inhibition, hypercalcemia.2

With the onset of hypocalcemia, there is excessive release of PTH and consequent hyperplasia of the parathyroid glands, which in turn will have a less satisfactory response to the inhibitory effects of ionic calcium and serum calcitriol.5

Excess serum PTH triggers serious damage to the body, including uremic syndrome, inhibition of erythropoiesis, calcification of soft tissues, such as the renal tissue and arteries, aggravating CKD and predisposing atherosclerosis and still seems to favor the development of cardiomyopathies.

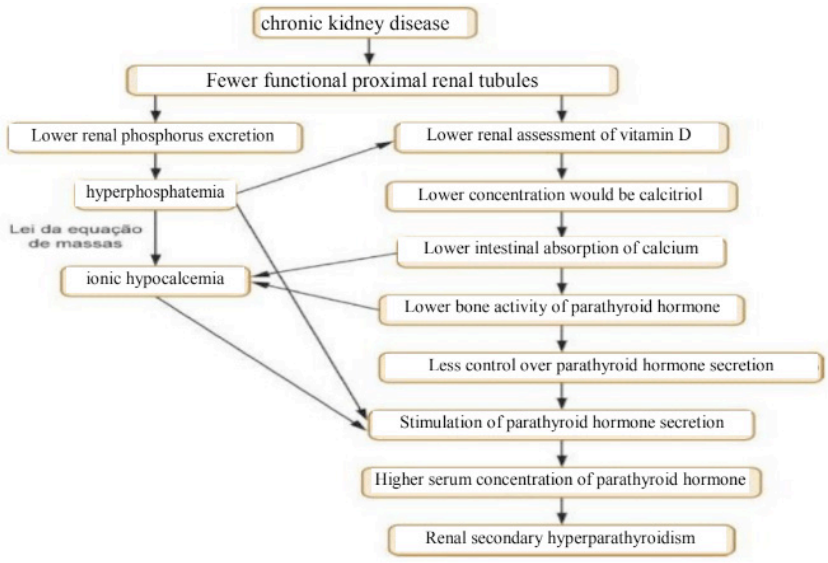

Figure 1: Schematic representation of the pathophysiology of HPTSR.5

The definitive diagnosis of HPTSR is performed through the measurement of serum PTH together with the assessment of calcemia6. Some authors also report the existence of a correlation between the serum PTH concentration and the serum phosphorus concentration5. An increase in the volume of the parathyroid glands can be observed on ultrasound1. Radiography is also an important diagnostic tool, as it allows the identification of osteodystrophies4.

The therapeutic approach to HPTSR is mainly aimed at controlling hyperphosphatemia. The use of restricted diets with low phosphorus concentration is one of the main tools used. Commercial feed formulas that meet this standard are already widely marketed. When dietary restriction is not sufficient to correct hyperphosphatemia, the use of intestinal phosphorus binders is an excellent option. Aluminum hydroxide and calcium carbonate, indicated only for patients with hypocalcemia, are the most commonly used. In dogs with moderate or severe azotemia, administration of calcitriol is indicated. It is important to point out that theRocaltrol®, a commercial formula available for human use, has a concentration much higher than the doses recommended for dogs. 1,4,5,7

FINAL CONSIDERATIONS

The hyperphosphataemia caused by CKD is the main cause of RHPT. From then on, a serum imbalance of calcium and phosphorus is generated, responsible for triggering a series of compensatory mechanisms that could be harmful to the body. There are losses that will culminate, in most cases, in kidney damage, aggravating the CKD condition, generating a cyclical scenario.

Therefore, the importance of prevention, early diagnosis and assertive treatment for effective control of CKD is evident.

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