SECONDARY HYPERPARATHYROIDISM

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Secondary hyperparathyroidism is defined as a derangement in calcium homeostasis, which leads to a compensatory increase in PTH secretion. It occurs primarily as a result of chronic kidney disease and is therefore sometimes referred to as renal Daniel Casanova , contemporary , University of Cantabria, Santander, Spain. intestinal malabsorption, vitamin D deficiency , liver disease or - chronic lithium usage. The pathogenesis of secondary hyperparathyroidism is related to renal dysfunction. Abnormalities in the r enal tubu - mild lar absorption of phosphate lead to hyper phosphataemia. This acts directly on the parathyroid cells and stimulates PTH secre - tion. More recent translational research has identified a novel - phosphaturia hor mone, fibroblast growth factor 23 (FGF23). This is progressively secreted from osteocytes to compensate for c hronic phosphate retention that in turn leads to a reduc - tion in 1,25-dihydroxyvitamin D, which by reducing the intes - tinal absorption of calcium also acts to increase secretion of PTH. Previous studies in patients with chronic renal disease - have shown that there is a reduction in the expression of the vitamin D rece ptor and the calcium-sensing receptor, with associated skeletal resistance to PTH. These factors interact - to form the complex pattern leading to progressive secondary hyperparathyroidism in the setting of chronic renal disease. one The pathological characteristics associated with secondary hyperparathyroidism include hyperplasia, asymmetrical glandular enlargement or nodularity . This di ff erentiation is important as, when the parathyroid gland becomes nodular, it loses expression of the vitamin D receptor and the calcium- sensing receptor gene. It has been proposed that nodular or parathyroid glands may be resistant to calcimimetics and therefore refractory to medical management. SECONDARY HYPERPARATHYROIDISM

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