Journal of Clinical & Experimental Pathology

Hyland, J Clin Exp Pathol 2024, 14:1

Short Communication Open Access

Emerging the Role of Parathyroid Glands in Long-Term Dialysis Patients

Lauren Hyland*

Department of Anatomy, Rostock University Medical Center, Rostock, Germany

***Corresponding author:** Lauren Hyland, Department of Anatomy, Rostock University Medical Center, Rostock, Germany, E-mail: lauhyl@RUMC.de

Received: 04-Jan-2024, Manuscript No. JCEP-24-128438; **Editor assigned:** 08-Jan-2024, PreQc No. JCEP-24-128438 (PQ); **Reviewed:** 22-Jan-2024, QC No. JCEP-24-128438; **Revised:** 29-Jan-2024, Manuscript No. JCEP-24-128438 (R); **Published:** 05-Feb-2024, DOI: 10.4172/2161-0681.24.14.482

Citation: Hyland L (2024) Emerging the Role of Parathyroid Glands in Long-Term Dialysis Patients. J Clin Exp Pathol. 14:482.

Description

The parathyroid glands are small and barely noticeable, but they are essential to the body's ability to maintain calcium homeostasis. The production of Parathyroid Hormone (PTH), which controls blood calcium levels, is attributed to these four tiny glands, which are usually found on the posterior surface of the thyroid gland. A variety of clinical symptoms can arise from disorders affecting the parathyroid glands, which can cause substantial disruptions in the calcium balance. The parathyroid glands are typically four in number, although variations in their location and number can occur. They are named based on their anatomical position relative to the thyroid gland: Two superior (or upper) parathyroid glands and two inferior (or lower) parathyroid glands. These glands are responsible for secreting Parathyroid Hormone (PTH), a key regulator of calcium and phosphate metabolism. PTH acts primarily on bone, kidney, and intestine to regulate calcium levels in the blood. It stimulates the release of calcium from bone, enhances calcium reabsorption in the kidneys, and promotes the production of activated vitamin D, which facilitates calcium absorption in the intestine. Through these actions, PTH helps maintain serum calcium levels within a narrow physiological range, crucial for various physiological processes, including neuromuscular function, bone health, and cardiac function [1-3].

Primary Hyperparathyroidism disorder occurs due to excessive secretion of PTH from one or more parathyroid glands, typically caused by a benign tumor or hyperplasia of the glands. Primary hyperparathyroidism leads to elevated serum calcium levels (hypercalcemia) and low serum phosphate levels (hypophosphatemia). Patients may present with symptoms such as fatigue, weakness, bone pain, kidney stones, and gastrointestinal disturbances. Long-term untreated hypercalcemia can result in osteoporosis, kidney damage, and cardiovascular complications. Secondary hyperparathyroidism develops as a compensatory response to chronically low serum calcium levels, often seen in conditions such as Chronic Kidney Disease (CKD) or vitamin D deficiency. In this condition, the parathyroid glands become hyperplastic and overproduce PTH in an attempt to maintain normal serum calcium levels. Secondary hyperparathyroidism can lead to bone demineralization, renal osteodystrophy, and cardiovascular complications. Tertiary hyperparathyroidism typically occurs as a complication of long-standing secondary hyperparathyroidism, where the parathyroid glands become autonomously overactive; disregarding the regulatory mechanisms that initially caused their hyperplasia. This condition often develops in patients with advanced CKD who undergo renal transplantation [4,5]. Tertiary hyperparathyroidism is characterized by persistently elevated PTH levels and hypercalcemia, despite successful kidney transplantation. Hypoparathyroidism results from inadequate secretion of PTH, leading to decreased serum calcium levels (hypocalcemia) and increased serum phosphate levels (hyperphosphatemia). This condition can be congenital or acquired, with causes including surgical removal of the parathyroid glands, autoimmune destruction, or genetic disorders such as DiGeorge syndrome. Hypoparathyroidism manifests with symptoms such as muscle cramps, paresthesia, seizures, and cardiac arrhythmias due to neuromuscular irritability and impaired calcium signaling. Diagnosing parathyroid disorders involves a combination of clinical assessment, biochemical tests, and imaging studies [6,7].

Measurement of serum calcium, phosphate, and PTH levels is essential for diagnosing parathyroid disorders. In primary hyperparathyroidism, serum calcium is elevated, while PTH levels are typically elevated or inappropriately normal. Secondary hyperparathyroidism is characterized by elevated PTH levels with either normal or low serum calcium levels. Imaging modalities such as ultrasound, technetium-99 m sestamibi scintigraphy, and more recently, Positron Emission Tomography (PET) scans with radiolabeled tracers, can aid in localizing abnormal parathyroid glands. These imaging techniques help identify the presence of adenomas, hyperplastic glands, or ectopic parathyroid tissue. Dual-Energy X-Ray Absorptiometry (DXA) scans are useful for assessing bone mineral density in patients with primary hyperparathyroidism or hypoparathyroidism, aiding in the diagnosis of osteoporosis or osteopenia. The management of parathyroid disorders depends on the underlying etiology and clinical manifestations. Surgical removal (parathyroidectomy) of the abnormal parathyroid glands is the definitive treatment for primary hyperparathyroidism, particularly in symptomatic patients or those with complications such as nephrolithiasis or osteoporosis. Minimally invasive techniques such as focused parathyroidectomy or intraoperative parathyroid hormone monitoring can help localize and remove the diseased glands with minimal morbidity [8-10].

Pharmacological interventions may include phosphate binders, vitamin D analogs, and calcimimetic agents to control PTH secretion and maintain serum calcium and phosphate levels within the target range. Treatment often involves surgical intervention to remove hyperplastic parathyroid glands, particularly in patients with persistent hypercalcemia and biochemical evidence of autonomous PTH secretion [11].

Conclusion

Management focuses on correcting hypocalcemia and minimizing symptoms while avoiding hypercalcemia and hypercalciuria. This typically involves oral calcium supplementation and active vitamin D analogs to maintain serum calcium levels within the low-normal range and promote intestinal calcium absorption. Close monitoring of serum calcium, phosphate, and urinary calcium excretion is essential to prevent

complications such as renal calcifications and nephrolithiasis. A thorough understanding of parathyroid anatomy, physiology, and pathology is crucial for accurate diagnosis and optimal management of these disorders, ultimately improving patient outcomes and quality of life.

References

1. Freitas JE, Freitas AE (1994) Thyroid and parathyroid imaging. Semin Nucl Med 24:234-245.
2. Palestro CJ, Tomas MB, Tronco GG (2005) Radionuclide imaging of the parathyroid glands. Semin Nucl Med 35:266-276.
3. Snyder SH, Bredt DS (1991) Nitric oxide as a neuronal messenger. Trends Pharmacol Sci 12:125-128.
4. Ekblad E, Alm P, Sundler F (1994) Distribution, origin and projections of nitric oxide synthase-containing neurons in gut and pancreas. Neuroscience 63:233-248.
5. Kendall CH, Roberts PA, Pringle JH, Iauder I (1991) The expression of parathyroid hormone messenger RNA in normal and abnormal parathyroid tissue. J Pathol 165:111-118.
6. Leong AS-Y, Milios J (1993) An assessment of the efficacy of the microwave-antigen retrieval procedure on a range of tissue antigens. Appl Immunohistochem 1:267-274.
7. Medrano C, Hazelrigg SR, Landreneau RJ, Boley TM, Shawgo T, et al. (2000) Thoracoscopic resection of ectopic parathyroid glands. Ann Thorac Surg 69:221-223.
8. Wells SA, Debenedetti MK, Doherty GM (2002) Recurrent or persistent hyperparathyroidism. J Bone Miner Res 17:158-162.
9. Kang YS, Rosen K, Clark OH, Higgins CB (1993) Localization of abnormal parathyroid glands of the mediastinum with MR imaging. Radiology 189:137-141.
10. Pruhs ZM, Starling JR, Mack E, Chen H (2005) Changing trends for surgery in elderly patients with hyperparathyroidism at a single institution. J Surg Res 127:58-62.
11. Ott MC, Malthaner RA, Reid R (2001) Intraoperative radioguided thoracoscopic removal of ectopic parathyroid adenoma. Ann Thorac Surg 72:1758-1760.