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Effect of Thyroidectomy Afflicted with Cancer on IGF-1 and PTH in the Women of Dhi Qar Governorate

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ABSTRACT

The study includes knowledge of the change in concentrations IGF-1 and PTH levels for patients with thyroid cancer after thyroidectomy. The study included 100 samples, divided into 50-control group and 50 patients; with 50patients having their thyroid glands removed in a period ranging from one day to a full month, and the study included all patients who had their total thyroid gland removed in Thi-Qar Governorate. The study found a drop in IGF-1 and PTH. The study's findings revealed a low in totals after the operation as compared to the control group.

1. Introduction

The endocrine system is a compilation of glands and tissues responsible for producing and releasing hormones. These hormones are vital for controlling and coordinating essential physiological processes such as growth, metabolism, sexual function, reproduction, sleep, and mood [1, 2]. The complexity of the endocrine system is noteworthy [1, 2]. It is comprised of specialized glands like the thyroid, parathyroid, and adrenal glands, as well as tissues including adipose tissue, bone, and other organs that also have secondary endocrine functions. These organs secrete a diverse array of hormones [1-4]. Some theories propose that the wide variety of bacteria in the human body's microbial biome might act as a "virtual endocrine organ," releasing chemical signals that impact human physiology [1-4].

This intricate system of glands and tissues generates hormones that are crucial for regulating essential bodily functions. One significant hormone, IGF-1, is secreted by various tissues, and its behavior may be influenced by its site of origin. Acting as both an endocrine hormone, initially produced by the liver and then transported to other organs, and a paracrine hormone, with multiple tissues including cartilaginous cells releasing it, IGF-1's potential role as an autocrine oncogene has also been suggested [1-4]. Recent reviews delve into how IGF-1 affects growth and metabolism in different tissues [1-4]. The debate over whether IGF-1, influenced by pituitary GH, is the "true growth hormone" is further explored below.

Notably, IGF-II is composed of 67 amino acids, while IGF-I is a basic single-chain peptide consisting of 70 amino acids. Interestingly, half of the amino acids in these peptides overlap with those found in insulin [1, 2, 5-8]. Moreover, they share a structural component resembling the C-

peptide in proinsulin. Elevated insulin levels trigger increased thymidine absorption and cell division in various normal cells [1, 2, 5-8]. Another vital hormone, PTH, containing 84 amino acids, plays a crucial role in regulating blood calcium and phosphate levels. PTH's short half-life is approximately 5 minutes [1]. Its secretion is governed by serum-ionized calcium, which interacts with a sensitive calcium-sensing receptor (CaSR) on the surface of parathyroid gland cells. Activation of this G-protein coupled receptor inhibits PTH release and reduces serum calcium levels [1, 7]. PTH levels exhibit an inverse sigmoidal correlation with serum ionized calcium levels [1, 7].

The relationship between PTH, also known as parathormone or parathion, and bone has been a subject of complex and extensive research [8-11]. Despite the challenges, this research has yielded valuable insights with significant translational implications. The first FDA-approved osteoanabolic drug for severe osteoporosis, Teriparatide, also referred to as PTH (1-34), emerged as a result of this ongoing investigation [8-11]. The association between PTH and the skeletal system, initially observed by anatomical pathologists Virchow and Erdheim in the late nineteenth century, was initially tenuous and observational. However, it has since become more clearly defined [8-11].

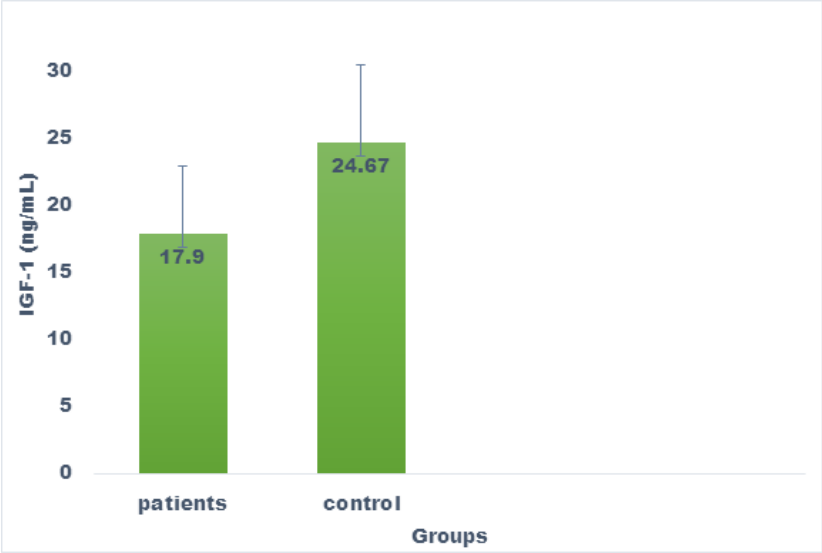
2. Patients and methods: This research was carried out at Nasiriyah Teaching Hospital in Thi-Qar, The Endocrine Glands Center in Thi Qar Governorate, and the Biochemistry Laboratory between 1/8/2022 and 1/8/2023. The study included 100 samples, divided into 50 control group and 50 patients, with 50patients having their thyroid glands removed in a period ranging from one day to a full month, and patients with euthyroid goiter aged (45-65). Using the ELISA instrument, medical tests on the patients' blood were done, including (IGF-1 and PTH).

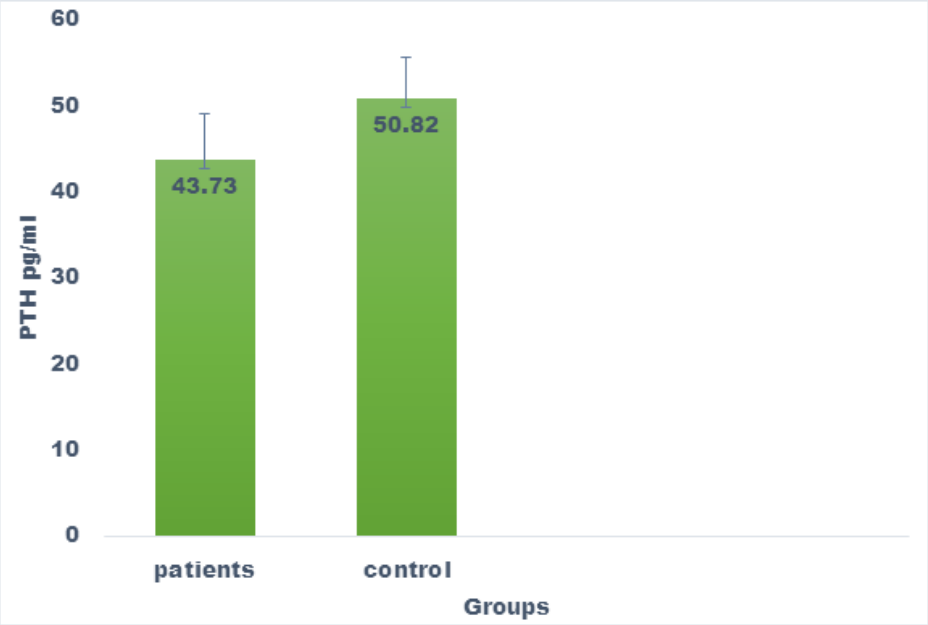
3. Results

The results showed a clear decrease in the patient group compared to the control group. The study showed that the process of total thyroidectomy for patients with thyroid cancer leads to a significant decrease in IGF-1 and PTH .

Table .1. Results of the parameters involved in our study

Parameters Groups	(IGF)-1 ng/ml	PTH pg/ml
patients	17.90 ± 5.01[b]	43.73 ±5.36[c]
Control	24.67 ± 5.78[a]	50.82 ± 4.89[a]
L.S.D	8.21	8.11





4. Discussion

Limited understanding exists regarding the mechanisms by which thyroid hormones impact bone health. Thyroid hormones can influence bone cells either by elevating the production of growth hormone (GH) and insulin-like growth factor-1 (IGF-1), or by directly regulating specific target genes through nuclear receptors. Thyroid hormone receptors (TRs) have been identified in human and rodent osteoblast-like cells, cell lines, and more recently, osteoclasts generated in vitro from an osteoclastoma [11-15]. A multitude of endocrine signals, including thyroid hormone, insulin-like growth factor I, glucocorticoids, and growth hormones, orchestrate the intricate process of longitudinal bone development. mThe growth of a child's skeleton is reliant on insulin-like growth factor-1 (IGF-1). Both bone mass and serum IGF-1 levels in individuals experience an increase from birth and reach their peak during puberty. Deviations from normal IGF-1 levels, either too low or too high, can lead to skeletal abnormalities such as acromegaly, dwarfism, and gigantism [15-17]. Deleting IGF1 conditionally from hepatocytes in the skeletal system has moderate effects on radial bone growth and no impact on linear bone growth or trabecular bone volume. Our study illustrates an association between hypothyroidism and reduced serum IGF-I levels. Furthermore, individuals with growth hormone (GH) deficiency exhibit lower IGF-I levels due to decreased muscle mass. Additionally, a diminished osteoblastic response to parathyroid hormone (PTH) is also a plausible outcome. Serum IGF-1 levels and osteoblasts' PTH-dependent synthesis of IGF-1 are elevated in acromegaly produced by a pituitary adenoma, which also results in enhanced bone remodeling [17-19]. The most common side effect of a complete thyroidectomy is hypoparathyroidism. The removal of the parathyroid gland and damage to its blood supply, which can result in temporary or chronic hypoparathyroidism, are just two of the causes of hypocalcemia after thyroid surgery [20, 21]. A common postoperative consequence following complete thyroidectomy is hypoparathyroidism or a lack of parathyroid hormone (PTH). If PTH deficient patients are not appropriately treated with activated vitamin D (1, 25-dihydroxycholecalciferol or calcitriol), they will experience significant hypocalcemia. On the long-term results of these patients, there hasn't been much research done. Parathyroid hormone (PTH) deficiency, often known as hypoparathyroidism, is one of a few postoperative problems linked to total thyroidectomy or complete thyroidectomy [22, 23]. While a lot has been written on the prevalence of hypocalcemia in people who have had thyroid surgery, less is known regarding the prevalence of PTH deficit and the long-term prognosis of these patients. There have not been any research done on the time course for serum PTH levels to return to normal levels or the time [22-25]

References

1. Galligan, T. M., Temkin, A. M., & Hale, M. D. (2023). Endocrine system. In Environmental Contaminants and Endocrine Health (pp. 3-23). Academic Press.
2. Ortega, M. A., Fraile-Martinez, O., García-Montero, C., Haro, S., Álvarez-Mon, M. Á., Leon-Oliva, D., ... & Álvarez-Mon, M. (2023). A comprehensive look at the psychoneuroimmunoendocrinology of spinal cord injury and its progression: mechanisms and clinical opportunities. Military Medical Research, 10(1), 1-40.
3. Keith, S. A. (2023). Steroid hormone regulation of innate immunity in Drosophila melanogaster. Plos Genetics, 19(6), e1010782.
4. Hymer, W. C., & Kraemer, W. J. (2023). Resistance exercise stress: theoretical mechanisms for growth hormone processing and release from the anterior pituitary somatotroph. European Journal of Applied Physiology, 1-12.
5. Trivellin, G., Daly, A. F., Hernández-Ramírez, L. C., Araldi, E., Tatsi, C., Dale, R. K., ... & Stratakis, C. A. (2023). Germline loss-of-function PAM variants are enriched in subjects with pituitary hypersecretion. Frontiers in Endocrinology, 14, 1166076.
6. Peppa, M., & Mavroeidi, I. (2023). The Role of the Environment in Endocrine Cancers. Environmental Endocrinology and Endocrine Disruptors: Endocrine and Endocrine-targeted Actions and Related Human Diseases, 457.
7. Mancusi, R., & Monje, M. (2023). The neuroscience of cancer. Nature, 618(7965), 467-479.
8. Kicińska, A. M., Maksym, R. B., Zabielska-Kaczorowska, M. A., Stachowska, A., & Babińska, A. (2023). Immunological and Metabolic Causes of Infertility in Polycystic Ovary Syndrome. Biomedicines, 11(6), 1567.
9. Quinteros, C. A., Cevallos, G. Y. H., & Paredes, K. A. B. (2023). Utility of serum parathyroid hormone measurement as a predictor of postthyroidectomy hypocalcemia: A single-center observational study. Oncología (Ecuador), 33(1), 91.
10. Bosman, A., Koek, W. N. H., Campos-Obando, N., van der Eerden, B. C., Ikram, M. A., Uitterlinden, A. G., ... & Zillikens, M. C. (2023). Sexual dimorphisms in serum calcium and phosphate concentrations in the Rotterdam Study. Scientific Reports, 13(1), 8310.
11. Sheetal, K., Sooria, N., & Nikisha, G. N. (2023). Impact of Clinico Pathological and Surgical Related Risk Factor in Post-Operative Hypoparathyroidism in Total Thyroidectomy Patients. Indian Journal of Otolaryngology and Head & Neck Surgery, 1-8.
12. Klug, S. T. (2023). Pituitary-Targeted Knockout of Glucocorticoid Receptors Suppresses Growth Hormone Expression During Mouse Embryonic Development (Doctoral dissertation, University of Maryland, College Park).
13. Cappola, A. R., Auchus, R. J., El-Hajj Fuleihan, G., Handelsman, D. J., Kalyani, R. R., McClung, M., ... & Verbalis, J. G. (2023). Hormones and Aging: An Endocrine Society Scientific Statement. The Journal of Clinical Endocrinology & Metabolism, dgad225.
14. Bogart, J. A. (2023). Transcriptomic Analysis of a Variant of Growth Hormone with Therapeutic Potential (Doctoral dissertation, Ohio University).
15. Wang, L. T., Chen, L. R., & Chen, K. H. (2023). Hormone-Related and Drug-Induced Osteoporosis: A Cellular and Molecular Overview. International Journal of Molecular Sciences, 24(6), 5814.
16. Styne, D. M. (2023). Pediatric endocrinology: A clinical handbook. Springer Nature.

17. Gubbi, S., Wurth, R., Hannah-Shmouni, F., & Koch, C. A. (2023). Environmental Endocrinology: Basic Concepts. In Environmental Endocrinology and Endocrine Disruptors: Endocrine and Endocrine-targeted Actions and Related Human Diseases (pp. 1-43). Cham: Springer International Publishing.
18. Yakar, S., & Isaksson, O. (2016). Regulation of skeletal growth and mineral acquisition by the GH/IGF-1 axis: Lessons from mouse models. Growth Hormone & IGF Research, 28, 26-42.
19. Pass, C. (2011). The role of suppressor of cytokine signalling-2 in endochondral bone growth (Doctoral dissertation, University of Glasgow).
20. Casey, C., & Hopkins, D. (2023). The role of preoperative vitamin D and calcium in preventing post-thyroidectomy hypocalcaemia: a systematic review. European Archives of Oto-Rhino-Laryngology, 280(4), 1555-1563.
21. Sittitrai, P., Ruenmarkkaew, D., Klibngern, H., Ariyanon, T., Hanprasertpong, N., Boonyaprapa, S., & Sreesawat, M. (2023). Perioperative versus postoperative calcium and vitamin D supplementation to prevent symptomatic hypocalcemia after total thyroidectomy: a randomized placebo controlled trial. International Journal of Surgery, 109(1), 13-20.
22. Aldhafar, A., Alghareeb, F., & Alammar, Z. (2023). Clinical Study of Post Thyroidectomy Hypocalcemia. Journal of Endocrine Surgery, 23(2), 40-46.
23. Choe, Y., Lee, Y. J., Shin, C. H., Chung, E. J., & Lee, Y. A. (2023). Risk factors of postoperative hypoparathyroidism after total thyroidectomy in pediatric patients with thyroid cancer. Annals of Pediatric Endocrinology & Metabolism, 28(1), 26.
24. Tu, C. L., Chang, W., Sosa, J. A., & Koh, J. (2023). Digital spatial profiling of human parathyroid tumors reveals cellular and molecular alterations linked to vitamin D deficiency. PNAS nexus, 2(3), pgad073.
25. Singhal, A. A. (2023). Atlas of Sonography of Parathyroid. Springer Nature.