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Chronic Kidney Disease and Brown Tumour: A Review of an Old Disease from a New Angle

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Introduction

A number of risk factors, including age, sex, drugs that impact calcium metabolism, and vitamin D insufficiency, can affect the development of brown tumors, skeletal lesions that are frequently encountered in people with chronic kidney disease. Secondary hyperparathyroidism is the main culprit, which also activates osteoclasts and causes calcium and phosphorus levels to be out of equilibrium. They might also develop as a result of additional causes like RAAS hyperactivity and persistent inflammation. Brown tumors went from reactive lesions to potentially neoplastic lesions when KRAS mutations were discovered to be involved recently. Pharmacologic therapies such bisphosphonates, calcimimetics, vitamin D supplementation, and denosumab can aid in managing these disorders by lowering hyperparathyroidism, restoring calcium levels, and preventing the development of OFC. Due to their rarity, brown tumors' appearance and treatment are not well understood. But when you take into account how they affect CKD patients' quality of life. The overactive osteoclasts that cause osteoeitis fibrosa cystica and Brown Tumors are two related but different forms of bone diseases that are most frequently linked to chronic renal disease. These illnesses are little understood despite the potential ramifications they may have due to their uncommon occurrence and variable clinical appearance. OFC and Brown Tumors are canonically brought on by secondary hyperparathyroidism in CKD. Recent research revealed that a number of variables, including chronic inflammation and hyperactivity of the renin-angiotensin-aldosterone pathway, may also contribute to the development of these disorders by activating osteoclasts [1].

Description

Renal osteodystrophy affects more than 70% of individuals on dialysis. Osteitis fibrosis cystic, commonly known as Brown Tumor and von Recklinghausen's disease of the bone, is a group of related but different bone diseases connected to chronic renal disease and hyperparathyroidism. Osteoclast activation and production are overactive in OFC, which results in cyst development and bone resorption. The first case of OFC was in the collection of Hunter, who lived from 1718 to 1783; Engel first described it in 1864, followed by von Recklinghausen in 1891. The presence of brown pigment within the lesion distinguishes brown tumors from less aggressive tumour-forming lesions of the OFC. There have been numerous prior instances of Brown Tumours being discovered in patients with chronic kidney disease. Around the world, 8% to 16% of people suffer from CKD, a prevalent ailment that is characterized by a progressive decrease of renal function for longer than 3 months. For individuals with advanced CKD, hemodialysis is a popular form of therapy. These people are more likely to develop brown tumors and OFC.

Brown tumor formation is also significantly influenced by primary hyperparathyroidism. It is a disorder where the excess parathyroid hormone produced by the parathyroid glands or parathyroid adenoma causes an imbalance in the metabolism of calcium and phosphorus. This may lead to increased osteoclast activity and consequent bone tissue breakdown, resulting in the emergence of Brown Tumors and OFC [2].

It is unclear how exactly OFC and Brown Tumours are developed as a result of chronic renal illness and hyperparathyroidism. PTH production, along with other elements like cytokines and growth factors, may contribute to the pathophysiology of various disorders, as was previously mentioned. The downstream signaling of PTH and calcium homeostasis was mediated by a variety of mediators, including receptor activator of NF-B ligand and macrophage colony-stimulating factor. Although the exact prevalence of OFC and Brown Tumours is unknown, patients with advanced CKD who are receiving hemodialysis are more likely to have these conditions. According to one report, 32% of patients with end-stage renal illness had OFC. The prevalence of Brown Tumours, in comparison, is only about 2% among patients with secondary hyperparathyroidism.Although the exact prevalence of OFC and Brown Tumours is unknown, patients with advanced CKD who are receiving hemodialysis are more likely to have these conditions. With an incidence of about 2% among patients with secondary hyperparathyroidism, brown tumors are rather uncommon. Age, gender, comorbidities, and specific drugs are only a few of the risk factors linked to an elevated likelihood of having these illnesses.

Another important risk factor is gender. Males are more frequently affected than females, with a male-to-female ratio of 1:3 in people under 30. Although the precise causes of this gender disparity are not fully understood, hormonal and genetic variables have been postulated to be relevant. Patients with OFC and Brown Tumors may experience a lower quality of life due to the current comorbidities. Secondary hyperparathyroidism in CKD is commonly accompanied by spontaneous tendon ruptures, itching from calcium deposits in the skin, ocular calcification, and calcification of the joints. The chance of having these illnesses can also be increased by specific drugs. In individuals with CKD, using calcium supplements, getting enough vitamin D, and using glucocorticoids have all been linked to an increased risk of OFC and Brown Tumours [3].

The pathophysiology of secondary HPT in CKD involves a number of variables. The development of secondary hyperparathyroidism is influenced by phosphate retention, hyperphosphatemia, low serum calcium ions, deficiencies, high levels of PTH, intestinal calcium malabsorption, and a decrease in vitamin D receptors and calcium-sensing receptors in the parathyroid glands. Additionally, parathyroid hyperplasia is frequently seen. Based on these findings about the pathophysiology, treatment for secondary HPT in the context of CKD and ESRD include regulating blood phosphate concentrations, giving out calcium and vitamin D analogs, and giving out calcimimetics. In hyperparathyroidism, increased PTH synthesis causes an imbalance in the metabolism of calcium and phosphorus. Osteoclasts, cells that are in charge of dissolving and removing bone tissue, may become active as a result of this. PTH receptors are not functionally expressed by osteoclasts. As a result, non-cell-autonomous mechanisms are responsible for the increases in osteoclast activity and number caused by PTH. M-CSF and RANKL are the two primary cytokines that support osteoclast development and activity. Additionally, M-CSF binds to its receptor on the surface of osteoclast precursor cells after being secreted. A number of intracellular signaling processes are sparked by the binding of M-CSF to c-Fms and help osteoclast precursor cells survive, proliferate, and differentiate into adult osteoclasts [4,5].

Conclusion

Bone lesions like OFC and Brown Tumours are common in CKP patients. Age, sex, prescription drugs that interfere with calcium metabolism, and a vitamin D deficiency are risk factors for acquiring these two disorders. The primary factor

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causing an imbalance in the levels of calcium and phosphorus, which may activate osteoclasts, is secondary hyperparathyroidism. The development of OFC and Brown Tumours in CKD, however, may potentially be influenced by additional mechanisms, such as RAAS hyperactivity and chronic inflammation. Given those principles, pharmaceutical therapies such bisphosphonate, calcimimetics, vitamin D supplements, and denosumab may be used to reduce hyperparathyroidism, raise the calcium level, and stop the development of OFC. Tumours contribute to a dearth of knowledge regarding the symptoms and therapies for this illness. Additionally, little is known about the pathophysiology of numerous pathways connected to Brown Tumours in CKD patients. However, nephrologists and other medical professionals who interact with dialysis patients should be aware of a variety of choices for diagnosis and therapy given the impact this condition has on the life-quality of CKD patients. Additionally, more study is required to compare the safety and efficacy of various treatment choices in populations of CKD patients.

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Conflict of Interest

There are no conflicts of interest by author.

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