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Lithium-induced parathyroid disease; an updated mini-review

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Abstract

Lithium is a medication commonly used to treat bipolar disorder and other psychiatric conditions. However, long-term use of lithium can lead to parathyroid dysfunction, known as lithium-induced parathyroid disease. The exact mechanism underlying lithium-induced parathyroid disease is not fully understood. Still, it is believed that lithium interferes with the normal function of the parathyroid gland by inhibiting the synthesis and secretion of parathyroid hormone (PTH) and impairing the sensitivity of the parathyroid gland to calcium. The management of lithium-induced parathyroid disease depends on the type and severity of parathyroid dysfunction. In patients with hypoparathyroidism, treatment involves the replacement of calcium and vitamin D supplements. In severe cases, intravenous calcium may be necessary. In patients with hyperparathyroidism, treatment consists in discontinuing lithium therapy and monitoring serum calcium and PTH levels. In situations where hyperparathyroidism persists, or complications like kidney stones or osteoporosis arise, surgery may become necessary.

Keywords: Lithium, Parathyroid disease, Lithium toxicity, Parathormone, Hypoparathyroidism, Bipolar disorder, Hyperparathyroidism, Parathyroid hormone

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Introduction

Lithium is a commonly used medication for treating bipolar disorder and other psychiatric conditions. However, its use has several adverse effects, including parathyroid dysfunction (1). Lithium-induced parathyroid disease is a well-known complication of long-term lithium therapy. This review paper aims to summarize the current knowledge regarding the pathophysiology, clinical presentation, and management of lithium-induced parathyroid disease (2,3).

Search strategy

For this review, we conducted an extensive search across various scientific databases, including PubMed, Web of Science, EBSCO, Scopus, Google Scholar, Directory of Open Access Journals (DOAJ), and Embase, using relevant keywords such as “lithium,” “parathyroid disease,” “lithium toxicity,” “parathormone,” “hypoparathyroidism,” “bipolar disorder,” “hyperparathyroidism,” and “parathyroid hormone.

Pathophysiology of lithium-induced parathyroid disease

The exact mechanism underlying lithium-induced parathyroid disease is not fully understood. However,

lithium is believed to interfere with the parathyroid gland's normal function by inhibiting the synthesis and secretion of parathyroid hormone (PTH; parathormone) (3-5). Lithium also impairs the sensitivity of the parathyroid gland to calcium, leading to a decrease in parathormone secretion in response to low serum calcium levels. These effects result in hypoparathyroidism or hyperparathyroidism, depending on the duration and dose of lithium therapy (2,6).

Clinical presentation of lithium-induced parathyroid disease

Lithium-induced parathyroid disease can present as either hypoparathyroidism or hyperparathyroidism. Hypoparathyroidism is characterized by low serum calcium levels, high serum phosphate levels, and low or undetectable PTH levels (2,3). Patients may experience symptoms such as muscle cramps, seizures, and tetany. High serum calcium levels, low serum phosphate levels, and high PTH levels characterize hyperparathyroidism. Patients may experience symptoms such as bone pain, fractures, renal stones, and fatigue (7,8).

Hyperparathyroidism is the most common manifestation of parathyroid disease induced by lithium toxicity. It occurs due to the stimulation of parathyroid

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■ Implication for health policy/practice/research/medical education

Lithium is a commonly used medication for treating bipolar disorder and other psychiatric conditions. However, long-term use of lithium can lead to various adverse effects, including parathyroid dysfunction. Parathyroid disease induced by lithium toxicity is a well-known complication that can result in hyperparathyroidism, hypoparathyroidism, or both. Early recognition and appropriate management of lithium-induced parathyroid disease are essential to prevent complications and improve patient outcomes. Patients taking lithium should be monitored regularly for signs and symptoms of lithium-induced parathyroid disease, including serum calcium and parathormone levels. Healthcare providers should also consider alternative medications for patients at high risk of developing lithium-induced parathyroid disease.

gland growth and function by lithium. This results in increased secretion of PTH, leading to elevated serum calcium levels and decreased serum phosphate levels. Hyperparathyroidism can cause symptoms such as bone pain, fractures, kidney stones, and gastrointestinal disturbances (3,5).

Hypoparathyroidism is less common but can also occur due to lithium toxicity. It results from the destruction or atrophy of parathyroid glands caused by long-term exposure to lithium. Hypoparathyroidism leads to low serum calcium levels and high serum phosphate levels, which can cause muscle cramps, seizures, and other neurological symptoms (9,10).

Management of lithium-induced parathyroid disease

The management of lithium-induced parathyroid disease depends on the type and severity of parathyroid dysfunction. In patients with hypoparathyroidism, treatment involves the replacement of calcium and vitamin D supplements. In severe cases, intravenous calcium may be necessary (2,11). Treatment for hyperparathyroidism patients involves discontinuing lithium therapy and monitoring serum calcium and parathormone levels. Surgery may be required when hyperparathyroidism persists, or complications such as renal stones or osteoporosis arise (3,12).

Conclusion

Lithium-induced parathyroid disease is a well-known complication of long-term lithium therapy. The pathophysiology of lithium-induced parathyroid disease involves inhibiting parathormone synthesis and secretion by lithium. Lithium-induced parathyroid disease can present as either hypoparathyroidism or hyperparathyroidism,

with different clinical manifestations and management strategies. Early recognition and appropriate management of lithium-induced parathyroid disease are essential to prevent complications and improve patient outcomes.

Conflicts of interest

The author declares that she has no competing interests.

Ethical issues

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