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CASE REPORT

Iatrogenic Hypoparathyroidism: Basal Ganglia Calcification, Bilateral Cataract, and Medullary Nephrocalcinosis

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Abstract

Hypoparathyroidism is a rare endocrine disorder characterised by low levels of serum calcium and parathyroid hormone. This report highlights iatrogenic hypoparathyroidism in a juvenile Graves' disease patient post near-total thyroidectomy, leading to low serum calcium and parathyroid hormone levels. Uncommon complications emerged, including asymptomatic basal ganglia calcification, medullary nephrocalcinosis, and bilateral cataracts. The case emphasises the need to recognize and manage the complexities linked to hypoparathyroidism resulting from surgical interventions in autoimmune thyroid disorders.

Keywords

Hypoparathyroidism, Juvenile Graves' disease, Basal ganglia calcification, Medullary nephrocalcinosis

Introduction

Primary hypoparathyroidism, characterised by hypocalcemia due to deficient parathyroid hormone (PTH) production, can result from inadvertent parathyroid gland destruction during thyroid surgery (75% of cases) [1]. Radiotherapy to the head and neck, infiltrative diseases (Wilson's disease, hemochromatosis, metastatic cancer), autoimmune disorders (autoimmune polyglandular syndrome type 1), or genetic abnormalities (isolated mutations or part of complex syndromes) [2-4]. PTH deficiency leads to low calcium, high phosphate levels, and an elevated serum calcium-phosphorus product, contributing to ectopic soft tissue calcifications. Intracranial calcifications, though rare, are typically found in the lentiform nuclei (putamen and globus pallidus) and caudate nuclei of the basal ganglia. Kidneys are more frequently affected, presenting as nephrolithiasis or nephrocalcinosis, while less common manifestations include eyes (cataract), skin, vasculature, and other organ systems [4,5]. Clinical symptoms correlate with the rate of hypocalcemia development, with acute hypocalcemia causing tetany and chronic hypocalcemia being either asymptomatic or presenting with mild symptoms. In some cases, it can lead to serious neurological and cardiovascular complications, making it a medical emergency [5].

Case Presentation

A 13-year-old girl, initially diagnosed with juvenile Graves' disease, underwent a protracted antithyroid drug (ATD) therapy without achieving remission. Subsequently, a near-total thyroidectomy was performed. Post-thyroidectomy, the patient presented with paresthesia and positive sign of latent tetany, indicative of hypocalcemia (serum calcium 1.54 mmol/L), high normal phosphate (1.45 mmol/L) and low serum parathyroid hormone (PTH) levels (0.085 pmol/L). Intravenous calcium infusion was initiated, and the patient was discharged with oral calcium and calcitriol replacement. After an initial period of regular follow-ups, the patient discontinued visits for several years. She now reported with reduced vision in both eyes. Ophthalmological examination revealed bilateral posterior subcapsular cataracts with visual acuity of

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6/36 in both eyes, subsequently patient underwent successful cataract surgeries and vision restoration.

Further investigations revealed echogenic renal pyramids on ultrasonography, suggestive of nephrocalcinosis (Figure 1), despite normal renal function tests, including creatinine levels (0.8). Non-contrast computed tomography (NCCT) of the head revealed asymptomatic bilateral basal ganglia calcifications (Figure 2).

Discussion

Primary hypoparathyroidism is characterised by abnormally low levels of parathyroid hormone (PTH), a crucial regulator of calcium balance. The clinical manifestations depend on the severity and pace of hypocalcemia development. Acute hypocalcemia can result in severe symptoms such as neuromuscular irritability, cognitive impairment, laryngospasm, stridor, airway obstruction, prolonged QT interval, cardiac arrhythmias, and in rare cases, depressed systolic function and heart failure. Neuromuscular symptoms include circumoral numbness, paresthesia, hyperreflexia, muscle cramping, tetany, and, in extreme cases, seizures, manifested by Trousseau and Chvostek signs [6]. Chronic hypocalcemia may present with neuropsychiatric symptoms like fatigue, hyperirritability, anxiety, and depression. Prolonged hypocalcemia with hyperphosphatemia, often seen in PTH deficiency or resistance, may lead to ectopic calcium-phosphorus complex deposition in various

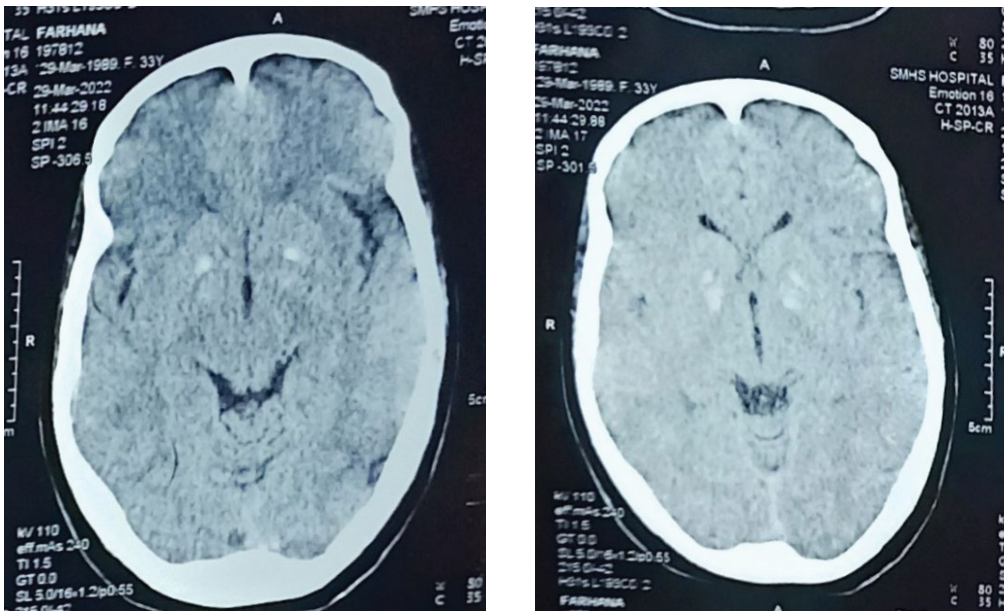

Figure 1: NCCT brain showing clacifications in the basal ganglia region bilaterally.

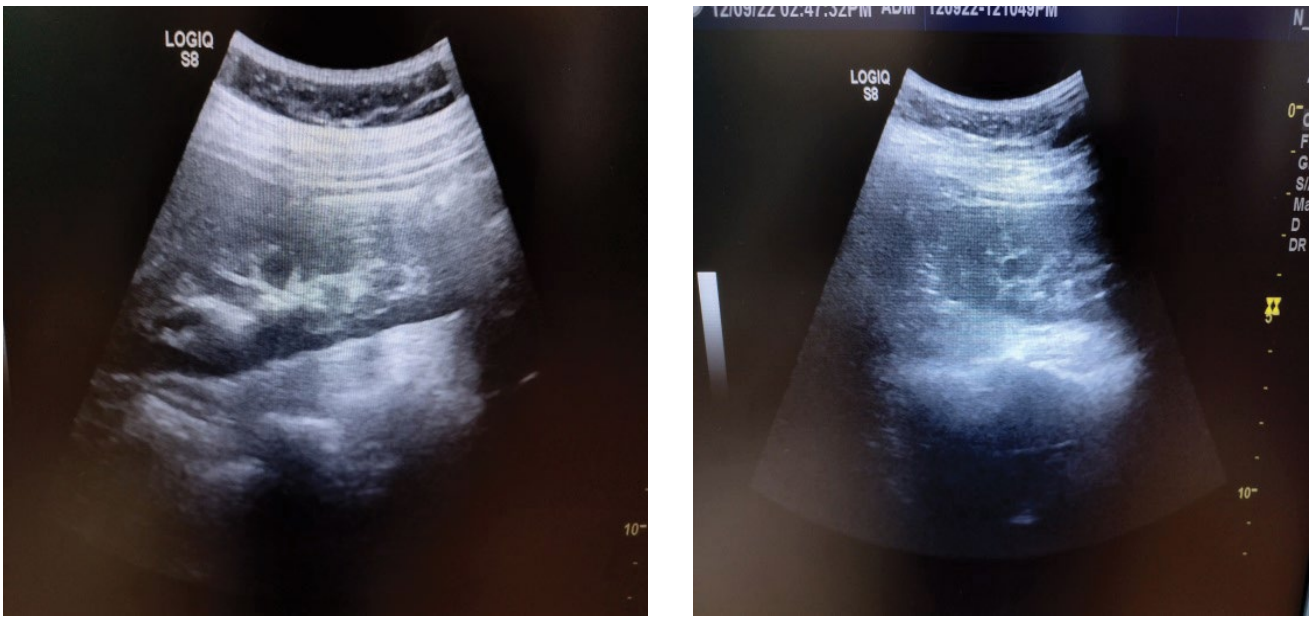

Figure 2: USG of kidneys showing echogenic renal pyramids suggestive of nephrocalcinosis.

tissues, potentially asymptomatic or causing secondary symptoms. Remarkably low ionized calcium levels can render patients clinically asymptomatic or present with initially overlooked symptoms.

Intracranial calcifications are seen physiologically in 0.3-1.5% of individuals [7], hypoparathyroidism and pseudohypoparathyroidism are among common causes of basal ganglia calcification [8]. Patients with basal ganglia calcifications can be asymptomatic (as in our case) or can have varied clinical manifestations [8,9]. Movement disorders, chorea, or parkinsonism seen in 20 to 30% of patients with basal ganglia calcification [10]. In addition to basal ganglia calcification, in rare cases, some patients may develop calcification of subcortical white matter, cerebral hemispheres including the frontal and parietal, and, more generally, extensive intracranial calcification [11]. Calcification of basal ganglia can be also caused by other factors, such as carbon monoxide poisoning, Fahr's disease, encephalitis, idiopathic calcification of the basal ganglia, tuberculous, Cocayne syndrome, cerebellar vascular disease, brain parasites, neurofibromatosis, but decreased PTH level and hypocalcemia rules out these causes [12].

Chronic hypoparathyroidism managed with conventional therapy of oral calcium and active vitamin D supplementation has adverse renal outcomes of nephrolithiasis, nephrocalcinosis, and renal insufficiency, generally one-third of the patients have these renal complications [13]. With chronic use of calcium without the renal calcium sparing effect of PTH, patients may become hypercalciuric. A retrospective cohort study found that patients with postsurgical hypoparathyroidism (n = 668) had an almost fivefold increased risk of nephrolithiasis and renal insufficiency compared with age- and gender-matched controls (n = 2064) [14]. Meola, et al. reported that 30% of the hypoparathyroid population had nephrolithiasis detected by renal ultrasound and that most were asymptomatic [15]. A long-term Massachusetts-based registry study of 120 patients with hypoparathyroidism treated with conventional therapy further supported these findings, nearly one-third of patients with hypoparathyroidism who underwent renal imaging were found to have either nephrolithiasis or nephrocalcinosis [5]. Pathophysiology of nephrolithiasis and nephrocalcinosis is related but physical expression and manifestation are different. Nephrolithiasis is characterised by solid (mostly calcium) stones that appear in the kidney and manifests as an acute, painful, and often recurring condition that may require hospitalisation whereas nephrocalcinosis consists of excess deposits of calcium salts within the renal tubules, tubular epithelium, or interstitium, develops slowly without symptoms [16,17]. In our case the patient had asymptomatic grade1 nephrocalcinosis without nephrolithiasis. Long standing untreated nephrocalcinosis and nephrolithiasis can lead to renal insufficiency.

Ophthalmic involvements in hypoparathyroidism are well established and include papilledema and early cataract. Hypocalcemic cataracts are bilateral, punctate, iridescent opacities in the anterior and posterior cortex lying beneath the lens capsule which are usually separated from the lens capsule by a zone of clear lens [18]. The opacities may remain stable or mature into complete cortical cataracts [19]. The proposed mechanism of cataract formation in hypoparathyroidism is membrane damage due to chronically low calcium level in the aqueous humour [20].

On review of the literature, we found various isolated case reports of hypoparathyroidism with cataract or basal ganglia calcification, but to the authors knowledge there is no reported case of hypoparathyroidism complicated with basal ganglia calcification, bilateral cataract and nephrocalcinosis in same patient, as seen in our case.

Conclusion

Basal ganglia calcifications, bilateral cataracts, and nephrocalcinosis are important long-term complications of hypoparathyroidism, which can be asymptomatic initially but later on can progress to severe neuropsychiatric manifestations, reduction in visual acuity, and renal failure respectively. Soit is important to look out for these complications in patients of hypoparathyroidism.

Learning Points

- Iatrogenic hypoparathyroidism as a potential complication following surgical interventions, particularly near-total thyroidectomy in autoimmune thyroid disorders such as juvenile Graves' disease.
- Hypoparathyroidism can affect multiple organ systems beyond the endocrine system. In this case, the manifestations included renal involvement (nephrocalcinosis), ophthalmic complications (bilateral cataracts), and neurological findings (basal ganglia calcification).
- The case emphasises the importance of long-term follow-up and monitoring in patients with hypoparathyroidism. Complications such as renal issues, vision impairment, and neurological manifestations may not be immediately apparent, underscoring the need for ongoing care to detect and manage these complications in a timely manner.

Disclosure Statement

This case report "Iatrogenic Hypoparathyroidism: Basal Ganglia Calcification, Bilateral Cataract, and Medullary Nephrocalcinosis" is presented without conflicts of interest. Patient consent has been secured, and efforts made to anonymize identity. Findings are

based on a single case, and the report does not endorse specific treatments. The authors encourage further research on this topic. Information provided is accurate to the best of authors knowledge.

Contributors

M.E.A., S.M.P., M.H.B and M.A.B. were involved in the diagnosis and management of this patient and manuscript submission.

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Disclosures

None.

Informed Patient Consent for Publication

Signed informed consent obtained directly from the patient.

Data Availability Statement

Original data generated and analyzed during this study are included in this published article.

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