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Case Report

Prolactinoma Associated with L-Dopa–Resistant Hyperprolactinemia in a Child with Tetrahydropterin (BH4) Deficiency

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Abstract
Tetrahydrobiopterin (BH4) deficiency causes hyperphenylalaninemia and impaired synthesis of serotonin and dopamine. Individuals with BH4 deficiency require personalized neurotransmitter replacement therapy to optimize treatment outcomes. An effective way to measure the success of this treatment is through serum prolactin levels; hyperprolactinemia indicates inadequate treatment and requires adjustments.Here, we report a 11-year- old girl with a diagnosis of BH4 deficiency who developed growth failure and persistent hyperprolactinemia despite L-dopa supplementation. Laboratory evaluation for growth failure revealed normal biochemical and thyroid hormone profiles, with decreased serum Insulin-like Growth Factor 1 (IGF-1) and Insulin-like Growth Factor Binding Protein 3(IGFBP-3), delayed bone age, and markedly elevated prolactin. Accordingly, growth hormone stimulation tests were conducted, revealing suboptimal responses and confirming growth hormone deficiency. In light of growth hormone deficiency and persistent hyperprolactinemia, cerebral magnetic resonance imaging was performed, demonstrating a solid pituitary lesion highly suggestive of prolactinoma. These findings suggest that in patients with BH4 deficiency, hyperprolactinemia resistant to L-dopa therapy may indicate the development of prolactinoma. Therefore, in resistant cases, regular laboratory follow-up and cranial imaging are clinically important.
Keywords: Tetrahydrobiopterin deficiency, prolactinoma, L-dopa, hyperphenylalaninemia

What is already known on this topic?
Recent clinical data suggest that in patients with BH_4 deficiency, chronic dopamine depletion leads to a loss of inhibitory control over pituitary lactotrophs. This "disinhibition" often results in L-DOPA-resistant hyperprolactinemia, as the intermittent nature of L-DOPA therapy fails to provide the continuous dopaminergic tone required to suppress prolactin secretion. Consequently, long-term stimulation can trigger lactotroph hyperplasia and the development of structural pituitary adenomas (prolactinomas).

What this study adds?
This report highlights several important clinical implications. First, if hyperprolactinemia persists despite optimal treatment with L-DOPA/BH_4, imaging studies, such as pituitary MRI, should be performed without delay to rule out pituitary adenomas. Furthermore, dopamine agonists such as cabergoline may be more effective in managing prolactin levels or treating symptomatic hyperprolactinemia and adenomas in this patient population due to their superior receptor affinities and longer duration of action.

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Introduction:
Biopterin metabolism disorders are rare but critical neurometabolic conditions that lead to hyperphenylalaninemia and require careful management. These disorders occur due to defects in the synthesis or recycling of Tetrahydrobiopterin (BH_4), an essential cofactor for the enzymes phenylalanine hydroxylase and tyrosine and tryptophan hydroxylase. The clinical manifestations extend beyond hyperphenylalaninemia and can involve severe neurological complications. These complications are characterized by deficiencies in vital neurotransmitters such as dopamine, serotonin, and adrenaline (1,2).
The main objective in managing these cases is to restore neurotransmitter balance in the central nervous system while maintaining controlled levels of blood phenylalanine (Phe). During monitoring, it is essential to assess blood Phe and tyrosine levels, as well as cerebrospinal fluid (CSF) metabolites such as 5-Hydroxyindoleacetic acid (5-HIAA) and homovanilic acid (HVA), which indicate neurotransmitter status in the brain. These measurements are considered the "gold standard." However, due to the difficulties associated with frequent lumbar punctures, the use of peripheral biomarkers has become increasingly important in clinical practice (3).
In this context, serum prolactin levels serve as a valuable tool for clinicians, acting as an indirect indicator of central dopaminergic activity. Dopamine in the hypothalamus primarily inhibits prolactin release; therefore, a deficiency of dopamine, such as that caused by BH4 deficiency, typically leads to hyperprolactinemia. Monitoring prolactin levels provides a non-invasive, practical, and cost-effective method for adjusting L-Dopa treatment dosages. However, since prolactin levels can be influenced by factors such as stress and sleep, results should always be evaluated in a multidisciplinary manner, considering the clinical picture and other metabolic data (3,4).
However, there are emerging reports of cases in which patients with BH_4 deficiency develop persistently elevated prolactin despite optimal L-DOPA therapy, suggestive of L-DOPA-resistant hyperprolactinemia. In some of these patients, imaging has revealed pituitary adenomas (prolactinomas), suggesting that chronic dopamine deficiency may lead not only to functional hyperprolactinemia but also to structural changes in the pituitary (5).
We report here a child with BH_4 deficiency who developed a prolactinoma despite compliance with L-DOPA therapy, highlighting challenges in diagnosis and management, and raising important questions about monitoring prolactin levels, imaging, and the role of dopamine agonists in this context.

Case Report

A 7-year-old girl with BH4 deficiency presented with pubic hair development. The patient was born at term (2150 g) to non-consanguineous parents. Following delivery, the neonate was admitted to the neonatal intensive care unit (NICU) due to a meconium plug and underwent surgery. Investigations for cystic fibrosis were performed; however, the results were within normal limits. Due to prolonged hospitalizations, the initial newborn screening (NBS) was delayed; subsequently, a heel prick sample was obtained at one month of age, and two months later the patient was referred to a pediatric metabolic center due to elevated Phe levels. Physical examination at the metabolic center at the age of 3 months revealed axial hypotonia (lack of head control), limb hypertonia, and absence of social smiling. Laboratory investigations showed a Phe level of 1630 µmol/L (N: below 120 µmol/L) and a tyrosine (Tyr) level of 60.2 µmol/L (N:30-120 µmol/L). BH4 loading test was performed with 20 mg sapropterin per kg, where the patient showed a 97% response, with normalization of Phe levels within 4 hours (1302 to 80 µmol/L), suggesting an inherited disorder of BH4 synthesis. BH4 deficiencies were investigated; revealing normal dihydropteridine reductase activity in dried blood spots (3.2 mU/mg (N: 1.8–3.8), and increased neopterin (22.21 mmol/mol creatinine (N: 1.2–15.5), and undetectable biopterin (0 mmol/mol creatinine [N: 0.75–7.3]) in urine, suggesting the diagnosis of 6-pyruvoyl-tetrahydropterin synthase (PTPS) deficiency. The diagnosis was confirmed by molecular genetic analysis, which identified of a homozygous mutation in the PTPS gene: c.243_243+1dup, p.(Glu82Glyfs*24). According to ACMG guidelines, this variant, consisting of frameshift and splice-site mutations, meets the Pathogenic Very Strong (PVS1) criterion because it completely disrupts the protein's structure (loss of function). Treatment was subsequently modified to include levodopa/carbidopa and oxitriptan in addition to sapropterin.

At presentation, the patient weighed 18 kg (-1.3 SD) and measured 111.1 cm in height (-2.1 SD), with a sitting height-to-height ratio of 0.53, and no dysmorphic features observed. Pubertal examination revealed Tanner stage 2 pubic hair, no breast development, and no clitoromegaly. She had delays in climbing stairs, running, and communication, was receiving special education services. She did not have swallowing dysfunction.

Laboratory investigations showed normal 17-hydroxyprogesterone, total testosterone, androstenedione, and thyroid hormone levels, while dehydroepiandrosterone sulfate (DHEAS) was elevated at 184.2 µg/dl (N:<40 µg/dl). Bone age assessment by the Greulich–Pyle method was consistent with 7 years and 10 months. On two separate days, early morning PRL levels measured before L-Dopa/carbidopa treatment were found to be high, at 50 ng/ml and 54 ng/ml, (N:<25 ng/ml), respectively. Based on these clinical and laboratory findings, the patient was diagnosed with idiopathic premature adrenarche.

Given the patient's history of congenital intrauterine growth restriction, congenital metabolic disease, and mild short stature, close follow-up was planned, with particular attention to annual growth velocity. The increase in serum PRL levels was discussed with the pediatric metabolism team, and the carbidopa/L-DOPA (25mg/250 mg) dose was increased to L-DOPA 10 mg/kg/day. The new treatment regimen has effectively normalized prolactin levels, measuring 2 ng/ml before morning L-Dopa/carbidopa treatment. One-year follow-up revealed a normal growth velocity of 5.2 cm/year, leading to the conclusion that the patient's short stature was consistent with idiopathic short stature. During follow-up, her annual growth rate was initially within the normal range; however, by 11 years of age, her height had increased by only 3.5 cm over the preceding year, indicating reduced growth velocity. At this visit, her weight was 37.2 kg (-0.27SD), height 127.7 cm(-2.6 SD) and sitting height/height ratio 0.53 (within the normal range). Systemic examination was unremarkable, and pubertal evaluation showed breast development at Tanner stage 2 and pubic hair at Tanner stage 2. Laboratory evaluation, including complete blood count and biochemical parameters were normal, thyroid hormones were normal, and serum IGF-1 and IGFBP-3 were at the lower end of normal (153 ng/ml and, 3.9 µg/ml respectively). Serum PRL was markedly elevated at 700 ng/ml. Since the patient was being followed at another center for metabolic BH4 deficiency, PRL values for the 7-11 age range were unavailable. Bone age assessment was consistent with 10 age. Growth hormone (GH) stimulation tests were performed using clonidine and L-Dopa. Peak GH responses were insufficient in both tests (6.23 ng/ml and 5.13 ng/ml, respectively). A 1 mcg Adrenocorticotropic hormone (ACTH) stimulation test revealed a normal cortisol response of 22.4 mcg/dl. Hypothalamo–pituitary Magnetic Resonance Imaging (MRI) demonstrated a 15 × 10 mm pituitary macroadenoma displacing the pituitary stalk without compressing the optic chiasm (Figure 1). Because the clear cause of hyperprolactinemia is established, macroprolactinemia has not been studied.The case was reviewed at the neuroradiology and neurosurgery council. Since there were no compressive symptoms, surgical intervention was deferred, and a 6-month observation plan was initiated. GH therapy could not be initiated due to lack of parental consent. Because of persistently elevated prolactin levels, the patient was started on long-acting carbidopa/levodopa therapy 4 times per day (15mg/kg/day). Even after three months of long-acting carbidopa/levodopa treatment, very high PRL levels of 800 ng/dl were still detected in the 24-hour measurement profile. Blood samples were collected two hours after treatment and prior to treatment (Figure 2). Therefore, cabergoline was started at 0.5 mg once weekly. One month after initiating cabergoline, serum PRL normalized, and after 6 months of treatment, follow-up MRI showed shrinkage of the pituitary lesion to 12 × 6 mm. After one year of cabergoline therapy, the lesion further decreased to 6 × 3.4 mm (Figure 3).

At her most recent clinical evaluation, the patient was 13 years old and presented with a height of 137.2 cm (-3.87 SDS). The presence of a macroadenoma and the family's refusal of GH treatment further worsened her short stature.Although a significant improvement in the annual growth rate to 5.2 cm/year was observed in the first year of treatment, the patient had completed puberty and had begun menarche by the second year of follow-up. Height growth had slowed considerably, with an annual growth rate of 2.8 cm/year. Bone age was consistent with 14 years, and the MRI scan showed the pituitary adenoma had reduced in size to 3x3.8 mm. Current biochemical markers indicated stable somatotropic and lactotropic axes: IGF-1: 294 ng/ml (N: 143–693), IGFBP-3: 5.9 µg/ml (N: 2.7–8.9), PRL: 14.8 ng/ml (N: 3.3–26.7). The patient is currently maintained on cabergoline at a dose of 0.125 mg/week.

Discussion

BH_4 deficiencies lead to impaired synthesis of catecholamines, particularly dopamine, which in turn reduces the tonic inhibitory control that dopamine usually exerts on lactotroph prolactin secretion. This may results in hyperprolactinemia under standard treatment regimens that include BH_4 supplementation, L-DOPA, and dietary phenylalanine restriction (2).

Treatment guidelines recommend using a combination of L-DOPA and a decarboxylase inhibitor, such as Carbidopa, for central dopamine replacement in BH4 deficiency. However, some case series have reported instances where patients were "resistant" to treatment, as PRL levels were not fully suppressed despite being on standard doses. This phenomenon is generally attributed to receptor desensitization or pharmacokinetic limitations that affect L-DOPA's ability to cross the blood-brain barrier (3,6).

High PRL levels are known to stimulate DHEA-S synthesis from the adrenal gland (7). Hyperprolactinemia, particularly seen in patients with L-DOPA-resistant BH4 deficiency, may be a cause of early adrenarche in our patient, although this cannot be definitively confirmed. Since puberty progresses physiologically during follow-up, even when PRL levels return to normal, a decrease in DHEA-S levels to prepubertal levels is not expected.

PRL secretion is regulated by a complex interplay of neurotransmitters, neurohormones, neuropeptides, metabolic substances, and systemic hormonal signals. Among physiological stimuli, estrogen, stress, and nutrient absorption are key factors promoting PRL release. Substances such as estrogens, noradrenaline, serotonin, opioids, and galanin stimulate PRL secretion by inhibiting the activity of the tuberoinfundibular dopaminergic system. Conversely, neurotransmitters that enhance dopaminergic activity reduce PRL secretion (8). In mild hyperphenylalaninemia with BH4 deficiency, the synthesis of neurotransmitters such as dopamine, noradrenaline, serotonin, and melatonin is impaired, as BH4 is a critical cofactor for the enzymes involved in their production. Since CSF neurotransmitter measurements are invasive, technically challenging, and provide only short-term data, serum PRL levels are often used as a surrogate marker to evaluate treatment efficacy in patients with BH4 deficiency (9). However, the most specific and critical treatment-monitoring measurement is the level of neurotransmitter metabolites in CSF (3).

There is evidence linking metabolic disorders that affect biogenic amine synthesis to pituitary enlargement and the development of prolactinomas. This may occur due to chronic dopamine deficiency, which leads to hyperplasia and hypertrophy of lactotroph cells, potentially resulting in multifocal prolactinomas, as observed in murine models with BH4 metabolism disorders (10). A particularly recent and illustrative example is the case of a patient with DHPR deficiency reported by Diaz-Moreno et al., in whom hyperprolactinemia persisted with amenorrhea despite appropriate BH_4 and L-DOPA therapy. Pituitary MRI revealed a microprolactinoma, and treatment with cabergoline normalized the prolactin levels and resolved symptoms (5). This suggests that dopamine precursor therapy alone may sometimes be insufficient to suppress PRL secretion or prevent lactotroph proliferation completely, particularly in cases where pharmacological dopamine action is inadequate, as in our case.

In a recent multicenter study, hyperprolactinemia was identified in 34 of 320 patients with inherited biogenic amine metabolism disorders. Among these, 26 patients exhibited PRL levels resistant to L-DOPA therapy. Pituitary MRI was performed in 21 patients, revealing normal findings in 10 and pathological findings in 11. Of those with abnormal MRIs, 6 had pituitary hyperplasia and heterogeneity, and 5 had adenomas. Notably, no macroadenomas were detected. Additionally, patients with pituitary hypertrophy or heterogeneity had significantly higher mean serum PRL levels compared to those with routine imaging (11). This case is noteworthy for highlighting a rare macroadenoma, offering essential insights that will enhance the existing literature on this medical condition.

The rise in PRL during puberty, reported in PTPS deficient patients, suggests that increasing hormonal milieu and possibly increased demands on dopamine production make the inhibition of prolactin secretion more challenging in adolescence. Hsu et al. (12) found that in 12 patients with PTPS deficiency, five had highly elevated PRL or symptoms related to hyperprolactinemia despite being under standard replacement therapy. However, none had MRI evidence of a tumor in that study. The patient presented here, in contrast, was just in early adolescence.

The development of a prolactinoma in the setting of BH_4 deficiency may thus represent a continuum from functional hyperprolactinemia (due to dopamine deficiency) to structural change in the pituitary (adenoma formation) if suppression of PRL is inadequate over time. The exact mechanism for adenoma development remains speculative, but it likely involves prolonged lactotroph activation in a low-dopamine environment (13). It has also been reported that hyperprolactinemia can be better controlled if the same L-DOPA dose is given more frequently (14). Although L-DOPA treatment was increased to 4 doses per day in our patient, high PRL levels could not be controlled.

It was hypothesized that prolonged and inadequate suppression of PRL secretion due to inadequate L-DOPA replacement may have contributed to the development of a pituitary macroadenoma in our patient. Despite receiving an appropriate dose of long-acting L-DOPA, the patient's PRL levels remained significantly elevated, necessitating initiation of treatment with the dopamine agonist cabergoline. PRL levels began to decline during the second week of treatment and returned to normal within the first month. A follow-up MRI at six months revealed a 30% reduction in tumor size, and one year later, the adenoma had shrunk by approximately 50%. The patient's annual growth rate also increased by 5.2 cm per year.

Dopamine agonists are the first-line treatment for both micro- and macroprolactinomas due to their effectiveness in suppressing PRL secretion and reducing tumor size. These drugs—including bromocriptine, cabergoline, pergolide, and quinagolide—act via G protein-coupled D2 dopamine receptor agonism. Bromocriptine also inhibits pituitary cell mitosis and induces apoptosis and perivascular fibrosis, contributing to tumor shrinkage. However, it requires twice-daily administration and is associated with notable side effects, including nausea, vomiting, orthostatic hypotension, and mental clouding (15). Cabergoline, on the other hand, offers better tolerability and requires less frequent dosing, while maintaining equivalent efficacy (16). In our case, treatment was initiated with cabergoline at a dose of 0.5 mg once weekly to support treatment adherence. After one month, PRL levels normalized, and the tumor size was reduced by half within one year. Subsequently, the dose was tapered to 0.125 mg per week.

Ergot-derived dopamine agonists exhibit potent agonist activity, particularly at serotonin 5-HT2B receptors, triggering fibroblast proliferation. This activation can lead to serious complications, most commonly heart valve fibrosis (restrictive valvulopathy), pleuropulmonary fibrosis, and retroperitoneal fibrosis. Although the risk is much lower in the pediatric population and at low doses (e.g., 0.125 mg cabergolin weekly) compared to adult Parkinson's patients, it should be noted that periodic monitoring of these "silent" fibrotic processes with echocardiography is necessary with cumulative dose increases (17).

Conclusion

In conclusion, effective management of BH_4 deficiency must address not only the biochemical correction of phenylalanine levels and the supply of neurotransmitter precursors but also include careful monitoring of endocrinological health for PRL abnormalities. Early detection of prolactinoma is crucial, as it can help prevent complications associated with chronic hyperprolactinemia, especially during adolescence. Future research should focus on establishing criteria for when imaging is necessary, determining the optimal timing and dosage of dopamine agonists for BH_4 deficiency, and evaluating the long-term outcomes of treated versus untreated prolactinoma in this population.

Ethics

Informed Consent: Written informed consent for publication of clinical details and images were obtained from parents.

Author Contributions: Concept- Fatma Özgüç Çömlek, Filiz Tütüncüler Kökenli
Design- Fatma Özgüç Çömlek, Filiz Tütüncüler Kökenliş, Hümeyra Yaşar Köstek Supervision- Fatma Özgüç Çömlek Materials, Fatma Özgüç Çömlek, Hümeyra Yaşar Köstek, Emine dilek data Collection and /or Processing- Fatma Özgüç Çömlek, Hümeyra Yaşar Köstek, Emine Dilek, Şebnem Kılıç Analysis and /or interpretation- Fatma Özgüç Çömlek, Literature Review Fatma Özgüç Çömlek; Hümeyra Yaşar Ksötek, Emine dilek Writer- Fatma Özgüç Çömlek,Şebnem Kılıç ;Critical Review- Fatma Özgüç Çömlek, Filiz Tütüncüler Kökenli

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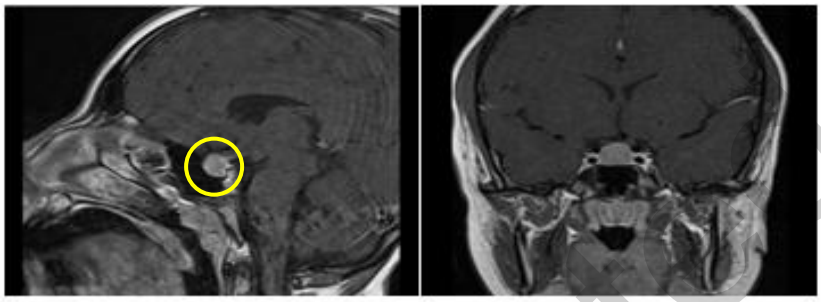

Figure 1: Coronal (right) and sagittal (left) T1-weighted MRI showing a 15 × 10 mm pituitary macroadenoma displacing the pituitary stalk without optic chiasm compression

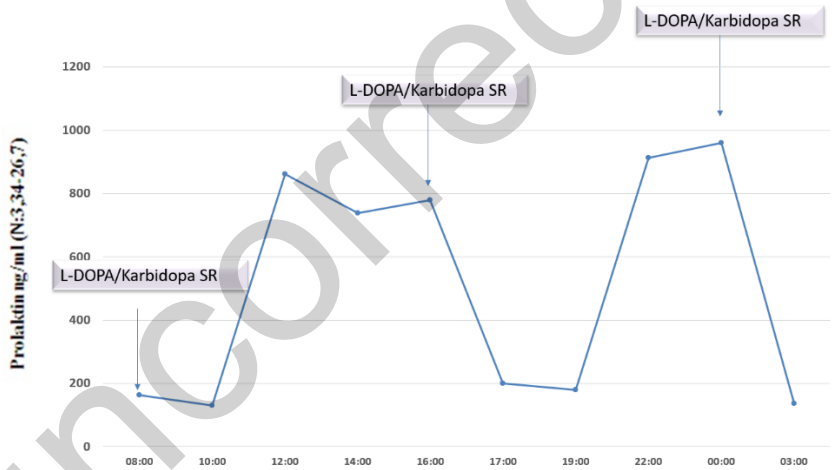

Figure 2: Serum prolactin levels in the patient following during long-acting L-Dopa/carbidopa therapy, showing persistently elevated values.

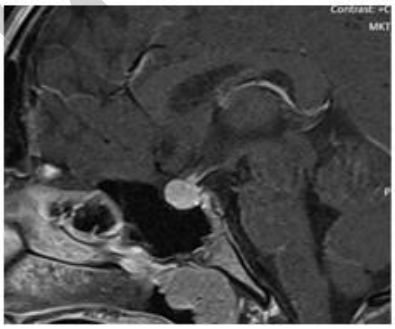

Figure 3: Sagittal T1-weighted MRI showing; image of a 6 X 3.4 mm mass in the pituitary gland at the first year of cabergoline treatment