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Homozygous Leptin Receptor Mutation Presenting with Severe Obesity, Hypopituitarism, and Type 2 Diabetes: A Case Report

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Abstract:

Background: Leptin receptor (LEPR) deficiency is a rare autosomal recessive disorder causing severe early-onset obesity, hyperphagia, and multiple endocrine abnormalities. Hypopituitarism is a significant but underrecognized complication of this genetic condition.

Case Presentation: We report a 14-year-old girl with homozygous LEPR mutation (c.3132delC; p.Asn1045Thrfs*2) presenting with severe obesity (BMI 62.7 kg/m²), new-onset type 2 diabetes (HbA1c 6.5%), and multiple pituitary hormone deficiencies including hypogonadotropic hypogonadism (undetectable LH/FSH), likely growth hormone deficiency, and central hypothyroidism. Despite advanced bone age indicating she was at near final height, the patient remained prepubertal with significant short stature (7th percentile for height) well below her mid-parental target height of 165 cm (75th percentile). Management included planned therapy with setmelanotide, an MC4R agonist specifically approved for LEPR deficiency, along with pituitary hormone replacement and management of diabetes and dyslipidemia.

Conclusions: This case highlights the complex endocrine manifestations of LEPR deficiency beyond obesity, emphasizing the importance of systematic evaluation for hypopituitarism in affected patients. The availability of targeted therapies like setmelanotide represents a paradigm shift in management, offering hope for improved outcomes in this challenging condition. Early recognition and comprehensive endocrine assessment are crucial for optimal patient care and long-term prognosis.

Introduction:

Monogenic obesity represents a distinct subset of pediatric obesity, accounting for less than 5.8% of cases but carrying profound implications for affected individuals and their families [1]. Unlike polygenic obesity, which results from complex interactions between multiple genetic variants and environmental factors,

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monogenic obesity follows Mendelian inheritance patterns and typically presents with severe early-onset obesity before age 2 years, accompanied by severe hyperphagia and resistance to conventional weight management strategies [2].

The leptin-melanocortin pathway serves as a critical regulatory system for energy homeostasis and appetite control, with leptin functioning as the primary adipose-derived hormone signaling satiety to the hypothalamus (See Figure 1)[3]. This pathway begins when leptin, produced by adipose tissue, binds to leptin receptors in the hypothalamus, triggering a cascade that ultimately activates melanocortin-4 receptors (MC4R) to generate satiety signals.

Leptin receptor (LEPR) deficiency, caused by biallelic mutations in the LEPR gene, represents one of the most well-characterized, albeit very rare forms of monogenic obesity. This condition was first described in humans by Clément et al in 1998 [4]. This landmark study demonstrated that homozygous LEPR mutations cause not only severe obesity and hyperphagia, but also significant pituitary dysfunction. These findings established leptin as a critical regulator of multiple endocrine functions beyond appetite control.

A recent systematic review observed that hypopituitarism affects approximately 34% of patients with LEPR deficiency, manifesting as hypogonadotropic hypogonadism (HH), growth hormone deficiency (GHD), and central hypothyroidism [5]. Over 90% of patients with LEPR mutations exhibit HH, while 30–50% have GH deficiency and 20–40% show central hypothyroidism [4,5]. These patients also face high risks of metabolic complications such as type 2 diabetes, dyslipidemia by adolescence [6]. We present a case illustrating these issues and discuss evidence-based management. The endocrine complications significantly impair growth, pubertal development, and long-term health outcomes, but remain under-recognized in clinical practice.

The development of targeted therapies has revolutionized treatment approaches for monogenic obesity. Setmelanotide (Imcivree™), an MC4R agonist approved for LEPR deficiency, has improved prospects for affected patients by directly targeting the disrupted melanocortin pathway [6]. This precision medicine approach represents a decisive shift from symptomatic management to addressing the underlying pathophysiology.

This case report presents a 14-year-old girl with homozygous LEPR mutation demonstrating the full spectrum of clinical manifestations associated with this condition, including severe obesity, metabolic complications, and hypopituitarism. We provide a detailed analysis of the clinical presentation, diagnostic workup, and management strategies, incorporating current evidence on the prevalence and characteristics of endocrine dysfunction in LEPR deficiency.

Case Report:

Patient Background and Genetic Diagnosis

We present a 14-year-old girl born to first cousin parents who presented to the pediatric endocrinology clinic at Al Jalila Children's Hospital in December 2022 with severe obesity and concerns regarding potential metabolic complications. The patient's early childhood history revealed onset of hyperphagia and rapid

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weight gain before age 2 years, consistent with monogenic obesity. Lifestyle interventions proved ineffective.

The patient was previously diagnosed with leptin receptor deficiency in 2018 following comprehensive next-generation sequencing (NGS) obesity panel testing revealed a homozygous likely pathogenic variant c.3132delC (p.Asn1045Thrfs*2) in exon 20 of the LEPR gene, after Initial targeted genetic testing for POMC, LEP, and MC4R mutations were negative. LEPR frameshift mutation creates a premature stop codon, triggering nonsense-mediated mRNA decay. This process results in complete loss of functional leptin receptor expression [7]. The consanguineous parentage is consistent with the autosomal recessive inheritance pattern of LEPR deficiency and highlights the increased risk of rare genetic conditions in populations with higher rates of consanguinity.

Clinical Presentation and Physical Examination

At presentation, the patient exhibited massive obesity (weight 146 kg; BMI 62.7 kg/m², height 154.3 cm (7th percentile), well below her mid parental target (165 cm, 75th percentile), a deficit of approximately 11 cm. This extreme degree of obesity was consistent with the severe phenotype typically observed in LEPR deficiency. She had significant growth failure relative to genetic potential.

Physical examination revealed generalized obesity with florid acanthosis nigricans, particularly affecting the neck, indicating severe insulin resistance. Blood pressure was significantly elevated at 143/98 mmHg. Marked lipomastia was present despite the patient's prepubertal status, reflecting her severe adiposity.

The patient exhibited the profound hyperphagia characteristic of LEPR deficiency. During her first clinic visit, she consumed three bags of potato chips, demonstrating the appetite dysregulation that defines this condition. Parents reported persistent food-seeking behaviors, poor satiety responses, and significant challenges with dietary restriction that had been present since early childhood. These behaviors included food hoarding, night-time eating, and emotional distress when food access was limited.

In an effort to manage her progressive weight gain, the patient underwent gastric bypass surgery at age 12 years. However, this intervention was unsuccessful, and she continued to gain weight post-operatively. This failure of bariatric surgery is consistent with the limited effectiveness of restrictive procedures in monogenic obesity, where the underlying appetite dysregulation persists despite surgery.

Symptoms suggestive of obstructive sleep apnea were present, including loud snoring, witnessed apnea, and daytime fatigue. These symptoms had worsened over time and significantly affected her quality of life and academic performance. She was completely prepubertal with no breast development, pubic hair growth, or menarche, despite her chronological age of 14.78 years.

Comprehensive laboratory evaluation revealed multiple significant abnormalities consistent with both metabolic complications of severe obesity and the characteristic endocrine dysfunction associated with LEPR deficiency (Table 1). The results demonstrated new-onset type 2 diabetes mellitus with elevated fasting glucose (160mg/dL) and HbA1c (7.0). The lipid profile demonstrated diabetic dyslipidemia with elevated triglycerides and LDL cholesterol), but low HDL cholesterol – ‘Diabetic Dyslipidemia’.

Pituitary hormone evaluation revealed multiple deficiencies characteristic of LEPR-associated hypopituitarism. Insulin-like growth factor-1 (IGF-1) was markedly reduced consistent with GHD explaining the patient's relative short stature. Reproductive hormone assessment demonstrated HH with undetectable gonadotropins, as is normally seen in LEPR patients.

Thyroid function testing revealed a pattern suggestive of central hypothyroidism, with low-normal free T4 and low free T3 despite normal TSH (2.1 mIU/L). This pattern, while subtle, is consistent with central hypothyroidism.

Bone age assessment using the Greulich-Pyle method showed a mildly advanced bone age of 15.0 years compared to the chronological age of 14.78 years. Using standard bone age prediction tables, this finding indicated that approximately 98.9% of the patient's growth potential had been completed, with an estimated final height of ~155 cm, excluding use of growth hormone therapy for height, even if other contraindications were not present.

The slightly advanced bone age, despite growth hormone deficiency, likely reflects the complex interplay between severe obesity, insulin resistance, and altered growth factor metabolism in the setting of LEPR deficiency. Hyperinsulinemia can accelerate skeletal maturation through insulin-like growth factor pathways, independent of growth hormone status.

To confirm the preliminary diagnosis of Type 2 diabetes a continuous glucose monitoring (CGM) sensor was placed for 14-day monitoring. The CGM data definitively confirmed diabetes with only 71% time in target glucose range (70-180 mg/dL), demonstrating significant glycemic variability and poor glucose control (**Figure 2**). Peak glucose levels exceeded 250 mg/dL, while nocturnal glucose levels remained consistently elevated above 180 mmol/L.

Given the complex presentation involving severe obesity, diabetes, and multiple endocrine deficiencies, a comprehensive multidisciplinary approach was required. Immediate priorities included diabetes control, cardiovascular risk reduction, and initiation of hormone replacement therapy for hypopituitarism.

Targeted Therapy for LEPR Deficiency

Setmelanotide (Imcivree™): The cornerstone of treatment for obesity due to LEPR variants is setmelanotide, an MC4R agonist approved for the treatment of obesity due to LEPR deficiency. Unfortunately, this high-cost orphan drug was not readily available in our center and required procurement from overseas on a named-patient basis, at another center, which nevertheless took several months.

Setmelanotide represents the first targeted therapy that directly addresses the underlying pathophysiology of leptin receptor deficiency [8]. The drug works by directly activating the melanocortin-4 receptor (MC4R), effectively bypassing the upstream block in the leptin-melanocortin pathway caused by loss of LEPR signaling (See Figure 1). By directly stimulating MC4R, setmelanotide can restore satiety signaling and promote weight loss even in the absence of functional leptin receptor signaling [9].

Clinical trials have demonstrated significant efficacy in patients with LEPR deficiency, with 45% of participants achieving at least 10% weight loss at one year, accompanied by substantial reductions in hunger

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scores (mean reduction of 43.7%) [10]. The therapy was expected to address both the weight management challenges and the profound hyperphagia that significantly impacted the patient's quality of life and family dynamics.

Other treatment

We recommended that our patient start semaglutide, given the anticipated delays in obtaining setmelanotide, as bridge therapy. Semaglutide is approved for adolescent obesity and offers the additional benefit of improved diabetes control and potential cardiovascular benefits [11]. While not specifically targeting the underlying genetic defect like setmelanotide, GLP-1 receptor agonists can provide meaningful weight loss and metabolic benefits. In addition, we advised metformin for glycemic control and statin-ezetimibe combination therapy for hyperlipidemia.Unfortunately the family chose not to accept these therapies. We further advised levothyroxine and estradiol replacement to bring about pubertal development, but the latter was also refused. We considered growth hormone treatment for its metabolic benefits including improved body composition and insulin sensitivity from, but her sleep apnea ruled this out.

Discussion:

This case exemplifies the complex clinical presentation and management challenges associated with leptin receptor deficiency, highlighting both the significant prevalence of hypopituitarism in affected patients and the need for targeted precision therapy to improve the lives of these patients.

A recent review analyzed data from 88 published LEPR-deficient patients worldwide, and found that 34% of patients develop one or more pituitary hormone deficiencies [5]. Clément et al reported that over 90% of patients with *LEPR* mutations exhibit hypogonadotropic hypogonadism (HH), while 30–50% have GH deficiency and 20–40% show central hypothyroidism [4,5]. These patients also face high risks of metabolic complications (e.g., diabetes, dyslipidemia) by adolescence, as in our case [6]. These complications significantly impact growth, pubertal development, and long-term health outcomes. The underlying pathophysiology of hypopituitarism relates to the critical role of leptin signaling in hypothalamic-pituitary function. Leptin receptors are expressed throughout the hypothalamic-pituitary axis, and the absence of functional leptin signaling disrupts normal neuroendocrine regulation affecting multiple hypothalamic nuclei responsible for regulation of the hypothalamic-pituitary axis [12].

HH, manifesting as absent pubertal development despite chronological age of 14 years, represents the most common endocrine abnormality in LEPR deficiency. [4] GHD contributes to the short stature observed in many affected patients, despite the presence of severe obesity, while central hypothyroidism may further complicate metabolic dysfunction and weight management efforts.

The specific mutation identified in our patient (c.3132delC; p.Asn1045Thrfs*2) represents a severe loss-of-function variant resulting in complete absence of functional leptin receptor protein. This frameshift mutation affects all isoforms of the leptin receptor, including the long form (LEPRb) responsible for intracellular signaling, eliminating any possibility of residual leptin signaling and contributing to the very severe

phenotype observed [13]. The complete penetrance and severe phenotype associated with null mutations underscore the critical role of leptin signaling in human energy homeostasis and endocrine regulation.

The availability of setmelanotide represents a paradigm shift in the management of patients with LEPR variants, offering the first targeted therapy specifically designed to address the underlying pathophysiology of genetic obesity disorders. The drug's mechanism of action, involving direct MC4R activation, bypassing upstream pathway defects and restores satiety signaling in patients with LEPR deficiency [14].

The regulatory approval of setmelanotide by both the FDA (2020) and EMA (2021) for patients aged 6 years and older with obesity due to LEPR deficiency represents a significant milestone in precision medicine for rare genetic conditions. However, access challenges, including high cost limits availability, and our patient had to go to another facility to receive treatment.

While setmelanotide is the gold standard for LEPR deficiency treatment, alternative approaches may provide benefits. GLP-1 receptor agonists, including semaglutide, have shown promise in treating various forms of obesity, including some genetic forms [15]. These medications work through pathways independent of the leptin-melanocortin system, potentially providing therapeutic benefits even in the presence of upstream pathway defects.

The failure of bariatric surgery in our patient illustrates the limitations of surgical interventions in genetic obesity conditions where the fundamental appetite dysregulation persists. This experience emphasizes the importance of understanding the underlying pathophysiology when selecting treatment approaches and highlights why targeted precision therapies are more appropriate for monogenic obesity.

Unfortunately, the prognosis for patients with LEPR deficiency has historically been poor, with high morbidity and mortality rates related to severe obesity and its complications. Recent studies have highlighted the significant health burden, with main causes of death in childhood including pulmonary and gastrointestinal infections, while adults face increased risks from diabetes complications, cardiovascular events, and obstructive sleep apnea [16]. Oxidative stress levels are notably higher in patients with genetic leptin and leptin receptor mutations, contributing to accelerated development of complications and emphasizing the need for comprehensive antioxidant strategies and cardiovascular risk reduction [16]. The multisystem nature of LEPR deficiency requires coordinated care involving multiple subspecialties and long-term monitoring for complications.

This case contributes to the growing body of literature documenting the clinical spectrum of LEPR deficiency and supports the importance of precision medicine approaches in managing rare genetic conditions. Continued research into optimal treatment strategies, long-term outcomes, and use of adjunct therapies such as GLP1 analogs should further improve care for patients with this challenging condition.

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Author Contributions: MA: Patient care, data collection, manuscript drafting. NKST: Patient care, clinical supervision, manuscript review and editing, final approval.

Ethics Statement: This case report was conducted in accordance with the Declaration of Helsinki. Institutional review board approval was not required for this case report as per institutional guidelines.

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Data Availability Statement: The datasets used and analyzed during the current study are available from the corresponding author on reasonable request, subject to patient privacy considerations.

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Patient Consent: Written informed consent was obtained from the patient's parents for publication of this case report and any accompanying images. The patient's identity has been protected throughout the manuscript.

Word Count: Approximately 2,100 words

Table 1: Comprehensive laboratory results demonstrating metabolic complications and endocrine dysfunction. Values outside the reference range are highlighted, showing evidence of diabetes, dyslipidemia, and multiple pituitary hormone deficiencies characteristic of LEPR deficiency.

Table 1: Laboratory Results

Parameter	Result	Reference Range	Interpretation
Metabolic Parameters			
Fasting Glucose	160 mg/dL	<100 mg/dL	Diabetic range
HbA1c	7.0%	<5.6%	Diabetic range
Total cholesterol	177 mg/dl	100-169 mg/dl	High
LDL- Cholesterol	132 mg/dl	<99 mg/dl	High
HDL-Cholesterol	31 mg/dL	50-70 mg/dL	Low
Triglycerides	334	<89 mg/dL	High
Endocrine Evaluation			
IGF-1	68.3 ng/ml	146-480 ng/dl	Low
IGFBP-3	3490 ng/ml	3500-7512 ng/ml	Low
FSH	< 0.4 mIU/ml	0.16-4.1 IU/L	Undetectable
LH	< 0.1 mIU/ml	0.02-0.3 IU/L	Undetectable
TSH	2 uIU/ml	0.47 - 3.4 uIU/ml	Normal
Free T4	10.7 pmol/L	9.5 - 16 pmol/L	Low-normal
Free T3	3.6 pmol/L	3.84 - 6 pmol/L	Low
Morning Cortisol	385 nmol/L	171-536 nmol/L	Normal

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Figure 1: Hypothalamic appetite regulation via the leptin-leptin receptor-MC4R pathway

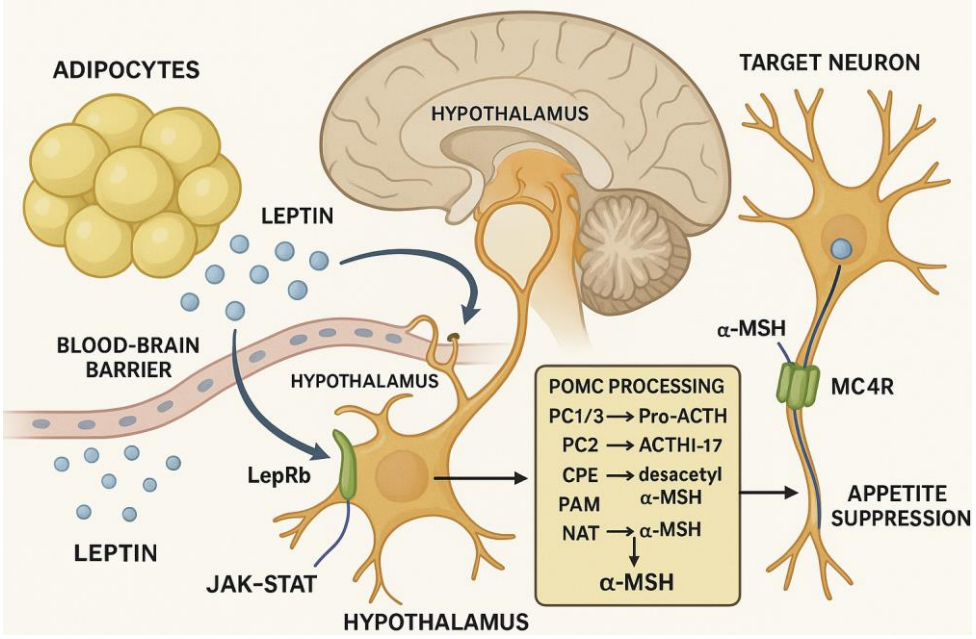


Figure Legend:

Leptin, secreted by adipocytes in proportion to fat stores, crosses the blood-brain barrier and binds to leptin receptors (LepRb) on pro-opiomelanocortin (POMC) neurons in the hypothalamic arcuate nucleus. Leptin binding activates the JAK-STAT (Janus kinase-signal transducer and activator of transcription) intracellular signaling pathway, leading to increased POMC gene expression and peptide processing. The POMC precursor undergoes sequential enzymatic cleavage: prohormone convertase 1/3 (PC1/3) cleaves POMC to generate pro-adrenocorticotropic hormone (Pro-ACTH), which is further processed by prohormone convertase 2 (PC2) to produce ACTH1-17. Carboxypeptidase E (CPE) removes C-terminal basic residues, followed by peptidyl α-amidating monooxygenase (PAM) amidation to generate desacetyl α-melanocyte-stimulating hormone (α-MSH). N-acetyltransferase (NAT) acetylates the peptide to produce the final bioactive α-MSH. α-MSH is released from POMC neurons and binds to melanocortin 4 receptors (MC4R) on target neurons, activating cyclic adenosine monophosphate (cAMP) signaling pathways that ultimately suppress appetite and reduce food intake. Setmelanotide as an MC4R agonist so bypasses any proximal block in the Leptin-Leptin Receptor-MC4R pathway. Figure was created with Manus AI.

Abbreviations: ACTH, adrenocorticotropic hormone; cAMP, cyclic adenosine monophosphate; CPE, carboxypeptidase E; JAK-STAT, Janus kinase-signal transducer and activator of transcription; LepRb, leptin receptor long form; MC4R, melanocortin 4 receptor; α-MSH, α-melanocyte-stimulating hormone; NAT, N-acetyltransferase; PAM, peptidyl α-amidating monooxygenase; PC1/3, prohormone convertase 1/3; PC2, prohormone convertase 2; POMC, pro-opiomelanocortin.

Figure 2: Continuous glucose monitoring data showing glycemic patterns over 14 days. The graph demonstrates poor glycemic control with only 71% time in target range (70-180 mg/dL), frequent hyperglycemic excursions exceeding 250mg/dL, and persistently elevated nocturnal glucose levels.

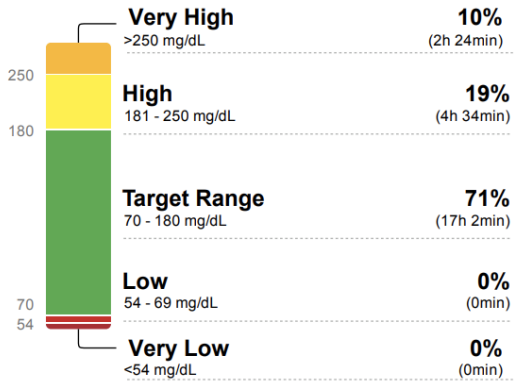
GLUCOSE STATISTICS AND TARGETS

22 December 2022 - 4 January 2023	**14 Days**
% Time Sensor is Active	**82%**

Ranges And Targets For	Type 1 or Type 2 Diabetes
Glucose Ranges Target Range 70-180 mg/dL	**Targets** % of Readings (Time/Day) Greater than 70% (16h 48min)
Below 70 mg/dL	Less than 4% (58min)
Below 54 mg/dL	Less than 1% (14min)
Above 180 mg/dL	Less than 25% (6h)
Above 250 mg/dL	Less than 5% (1h 12min)
Each 5% increase in time in range (70-180 mg/dL) is clinically beneficial.	

Average Glucose	**156** mg/dL
Glucose Management Indicator (GMI)	**7.0% or 54 mmol/mol**
Glucose Variability	**39.3%**
Defined as percent coefficient of variation (%CV)	

TIME IN RANGES



Glucose Patterns (14 Days)