



# Elderly Hypothyroidism Presenting with Recurrent Hypoglycemia as the Initial Symptom: A Case Report and Literature Review

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## ABSTRACT

Background: Elderly primary hypothyroidism is a common geriatric endocrine disease, with typical clinical manifestations including fatigue, lethargy, cold intolerance, and slow movement. Hypoglycemia is an atypical and rare initial symptom of hypothyroidism, which is extremely easy to be misdiagnosed as hypoglycemic encephalopathy, neurological diseases or diabetic abnormal glucose metabolism, leading to delayed diagnosis and missed standardized treatment. Case Presentation: We report a 72-year-old elderly female patient without a history of diabetes, hypoglycemia or endocrine diseases, who was admitted to the hospital with recurrent dizziness, fatigue and intermittent syncope for 1 month. Multiple random blood glucose tests during hospitalization showed persistent low glucose levels, with the lowest value of 2.1 mmol/L. After excluding common causes of hypoglycemia such as improper diet, insulin tumor, liver and renal dysfunction, and adrenal insufficiency, further thyroid function examination revealed significantly elevated thyroid-stimulating hormone and decreased free thyroid hormone levels, confirming the diagnosis of primary severe hypothyroidism. After standardized levothyroxine replacement therapy, the patient's thyroid function returned to normal, and recurrent hypoglycemia symptoms were completely resolved without recurrence during 6 months of follow-up. Conclusion: Elderly hypothyroidism can be initiated by recurrent hypoglycemia without typical hypothyroid manifestations. For elderly patients with unexplained recurrent hypoglycemia, routine thyroid function screening should be performed to rule out atypical hypothyroidism, so as to avoid clinical misdiagnosis and delayed treatment.

**Keywords:** *elderly patients; primary hypothyroidism; recurrent hypoglycemia; atypical manifestation; case report; misdiagnosis*

## 1. Introduction

Primary hypothyroidism is one of the most prevalent endocrine disorders in elderly populations, caused by decreased thyroid hormone synthesis and secretion or weakened biological effects, leading to systemic hypometabolic syndrome (Chaker et al., 2022). Epidemiological studies show that the overall prevalence of hypothyroidism in people aged over 65 years is 8%–15%, and the incidence increases significantly with age, with a higher morbidity rate in elderly female patients than in males (Garmendia Madariaga et al., 2021). Typical clinical symptoms of hypothyroidism in the elderly are characterized by hypometabolic manifestations, including general fatigue, mental apathy, cold intolerance, dry skin, constipation, bradycardia, and facial and limb edema, which are easy to identify in clinical practice (Razvi et al., 2023).

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However, elderly patients often have atypical clinical manifestations of hypothyroidism due to the degenerative changes of multiple organs and declined physiological reserve function. A small number of patients lack classic hypothyroid symptoms, but present with rare extrapulmonary and systemic atypical manifestations, which greatly increases the difficulty of early clinical diagnosis (Franco et al., 2022). Recurrent spontaneous hypoglycemia is an extremely rare initial manifestation of primary hypothyroidism, which is rarely reported in clinical literature. Most existing studies suggest that hypoglycemia secondary to hypothyroidism mostly occurs in patients with severe hypothyroidism or combined with multiple endocrine dysfunctions, and isolated hypoglycemia as the first and single clinical manifestation is extremely uncommon (Park & Kim, 2022).

Clinically, spontaneous recurrent hypoglycemia is mostly associated with insulinoma, severe liver and kidney insufficiency, adrenal cortical hypofunction, improper hunger, drug-induced hypoglycemia and other factors (Liu et al., 2023). For elderly patients without a clear history of metabolic diseases, unexplained recurrent hypoglycemia is often misdiagnosed as neurological disorders, transient cerebral ischemia or unexplained hypoglycemic lesions, resulting in repeated symptomatic treatment without fundamental intervention, and even inducing severe adverse events such as hypoglycemic coma and cognitive impairment (Wang et al., 2024).

At present, there are few systematic case reports and literature reviews on elderly hypothyroidism with recurrent hypoglycemia as the initial symptom. The specific potential mechanism of hypoglycemia caused by hypothyroidism has not been fully clarified, and there is a lack of unified clinical diagnosis and treatment consensus for such atypical cases (Zhang et al., 2023). This paper reports a rare case of a 72-year-old female elderly patient with primary hypothyroidism characterized by recurrent hypoglycemia as the initial manifestation, summarizes the patient's clinical data, auxiliary examination results, diagnosis and treatment process and follow-up outcomes, and combines domestic and foreign relevant literature to analyze the possible pathogenesis, clinical diagnostic clues, differential diagnosis points and standardized treatment strategies, so as to improve clinicians' recognition of atypical elderly hypothyroidism and reduce the rate of clinical misdiagnosis and missed diagnosis.

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## 2. Case Presentation

### 2.1 Basic Patient Information

The patient was a 72-year-old female, retired worker, admitted to the Geriatric Internal Medicine Department of our hospital on October 12, 2024, with the main complaint of "recurrent dizziness, fatigue and intermittent syncope for 1 month, aggravated for 3 days". The patient had no history of hypertension, diabetes, coronary heart disease, chronic liver and kidney diseases, and no history of long-term medication use. There was no special family genetic disease history, no history of drug allergy, and no recent special diet, hunger, strenuous exercise or trauma stimulation.

### 2.2 Present Illness History

One month before admission, the patient had no obvious inducement of repeated dizziness, physical weakness, and occasional blurred vision, which could be relieved after rest. The patient did not pay attention to the symptoms and did not receive systematic examination and treatment. In the recent 3 days, the above symptoms were significantly aggravated, with 2 episodes of transient syncope during activities, each lasting about 1–2 minutes, and spontaneously awake without convulsions, incontinence, limb paralysis and numbness. The patient was accompanied by occasional palpitations, poor appetite and mild lethargy, without obvious cold intolerance, dry skin, constipation, edema and other discomforts. In order to further clarify the diagnosis, the patient was admitted to our hospital for standardized examination and treatment.

### 2.3 Physical Examination on Admission

Vital signs: Temperature 36.2 °C, Pulse 58 beats/min, Respiration 18 times/min, Blood pressure 125/78 mmHg. General condition: clear consciousness, poor mental state, normal skin temperature, no dry and rough skin, no facial and eyelid edema, no goiter and

lymphadenopathy in the neck. Cardiopulmonary abdominal examination: low and blunt heart sound, no murmur in each valve area; clear breath sounds in both lungs, no dry and wet rales; flat abdomen, soft texture, no tenderness and rebound pain, normal liver and spleen palpation. Neurological examination: normal limb muscle strength and muscle tension, negative pathological signs, stable gait. Routine physical examination showed no typical hypothyroid manifestations.

## 2.4 Auxiliary Examination Results

2.4.1 Blood glucose monitoring: Continuous fingertip blood glucose monitoring was performed after admission. The fasting blood glucose was 2.2–2.8 mmol/L, and the 2-hour postprandial blood glucose was 2.9–3.3 mmol/L. The lowest random blood glucose was 2.1 mmol/L, which met the clinical diagnostic criteria for hypoglycemia (blood glucose <3.9 mmol/L), with repeated and persistent low blood glucose levels.

2.4.2 Routine laboratory tests: Routine blood, blood lipid, electrolyte, myocardial enzyme and coagulation function were all within the normal reference range. Liver function: ALT 28 U/L, AST 32 U/L; Renal function: BUN 5.2 mmol/L, Scr 76  $\mu$ mol/L, indicating normal liver and renal function. Cortisol, adrenocorticotrophic hormone and pancreatic function indexes were normal, excluding adrenal insufficiency and islet cell lesions.

2.4.3 Thyroid function examination: Thyroid-stimulating hormone (TSH) 28.65 mIU/L (normal reference range: 0.27–4.2 mIU/L), free triiodothyronine (FT3) 2.12 pmol/L (normal: 3.1–6.8 pmol/L), free thyroxine (FT4) 8.56 pmol/L (normal: 12–22 pmol/L). Thyroid autoimmune antibodies were negative. The examination results confirmed the diagnosis of severe primary hypothyroidism.

2.4.4 Imaging and auxiliary examinations: Cranial CT and magnetic resonance imaging showed no intracranial infarction, hemorrhage, space-occupying lesions and cerebral vascular malformations. Abdominal ultrasound showed normal liver, gallbladder, pancreas, spleen and kidney morphology, no pancreatic space-occupying lesions. Thyroid ultrasound showed uniform thyroid parenchyma, no nodules, atrophy and hypertrophy lesions. Electrocardiogram showed sinus bradycardia, no ST-T segment changes.

**Table 1 Baseline laboratory-test data of the 72-year-old female patient**

| Laboratory item                       | Patient value | Normal reference range |
|---------------------------------------|---------------|------------------------|
| TSH (mIU/L)                           | 28.65         | 0.27-4.20              |
| FT3 (pmol/L)                          | 2.12          | 3.1-6.8                |
| FT4 (pmol/L)                          | 8.56          | 12-22                  |
| Fasting blood glucose (mmol/L)        | 2.2-2.8       | 3.9-6.1                |
| Minimum random blood glucose (mmol/L) | 2.1           | $\geq 3.9$             |
| ALT (U/L)                             | 28            | 5-40                   |
| AST (U/L)                             | 32            | 8-40                   |
| BUN (mmol/L)                          | 5.2           | 3.2-7.1                |
| Scr ( $\mu$ mol/L)                    | 76            | 57-111                 |
| Morning cortisol (nmol/L)             | 412           | 138-690                |
| ACTH (pg/mL)                          | 27            | 7-65                   |

*Note: TSH=thyroid-stimulating hormone; FT3=free triiodothyronine; FT4=free thyroxine; ALT=alanine aminotransferase; AST=aspartate aminotransferase; BUN=blood urea nitrogen; Scr=serum creatinine; ACTH=adrenocorticotrophic hormone.*

## 2.5 Diagnostic Basis and Differential Diagnosis

Combined with the patient's clinical symptoms and auxiliary examination results, common causes of recurrent hypoglycemia were excluded one by one: no history of diabetes and hypoglycemic drug use, excluding drug-induced hypoglycemia; normal pancreatic imaging and insulin level, excluding insulinoma; normal liver and renal function and adrenal function, excluding hypoglycemia caused by visceral insufficiency and adrenal lesions; no irregular diet and hunger factors, excluding nutritional hypoglycemia. Combined with

significantly abnormal thyroid function indexes, the final diagnosis was primary severe hypothyroidism complicated with recurrent hypoglycemia (atypical initial manifestation).

## 2.6 Treatment Process

After confirming the diagnosis, the patient was given standardized thyroid hormone replacement therapy. Levothyroxine sodium tablets were orally administered at an initial dose of 25 µg/d, with slow dose adjustment according to the patient's tolerance and thyroid function reexamination results. During the treatment period, symptomatic supportive treatments such as regular blood glucose monitoring and nutritional support were given, and no special hypoglycemia targeted drugs were used. The patient was instructed to maintain a regular diet and avoid hunger and fatigue.

After 2 weeks of treatment, the patient's dizziness, fatigue and lethargy symptoms were significantly improved, and no syncope occurred again. Reexamination of blood glucose showed that fasting and postprandial blood glucose levels returned to the normal range. After 4 weeks of treatment, thyroid function was rechecked: TSH 7.82 mIU/L, FT3 3.56 pmol/L, FT4 14.28 pmol/L, which was significantly improved compared with baseline. The levothyroxine dose was adjusted to 50 µg/d for maintenance treatment.

## 2.7 Follow-up Outcomes

The patient was discharged after 4 weeks of standardized treatment. Regular outpatient follow-up and thyroid function monitoring were performed for 6 consecutive months. During the follow-up period, the patient's mental state, appetite and physical activity returned to normal, and there was no recurrence of dizziness, fatigue and syncope. Thyroid function indicators were completely restored to the normal range, and blood glucose levels were stably normal without hypoglycemia recurrence. The long-term prognosis was good.

**Table 2 Dynamic changes of thyroid function and blood glucose during follow-up**

| Time point                   | TSH (mIU/L) | FT4 (pmol/L) | Fasting glucose (mmol/L) | Levothyroxine dose | Clinical symptoms                        |
|------------------------------|-------------|--------------|--------------------------|--------------------|------------------------------------------|
| Before treatment             | 28.65       | 8.56         | 2.2-2.8                  | 0 µg/d             | Dizziness, fatigue, intermittent syncope |
| 2-week follow-up             | 15.36       | 11.25        | 4.0-4.6                  | 25 µg/d            | Symptoms improved, no syncope            |
| 4-week follow-up             | 7.82        | 14.28        | 4.3-4.9                  | 50 µg/d            | All abnormal symptoms disappeared        |
| 6-month outpatient follow-up | 3.96        | 18.32        | 4.5-5.2                  | 50 µg/d            | Complete remission, no relapse           |

*Note: Thyroid-function indexes gradually returned to normal after hormone replacement; blood glucose normalized synchronously with improvement of thyroid function.*

## 3. Literature Review

A systematic literature search was conducted in PubMed, Web of Science, CNKI and Wanfang databases with the keywords "elderly hypothyroidism", "recurrent hypoglycemia", "atypical manifestation", "case report". Relevant literature published from 2018 to 2024 was screened, and duplicate and low-quality studies were excluded. A total of 21 relevant domestic and foreign literature studies were included, including 13 case reports and 8 clinical observational studies.

Existing literature data show that hypothyroidism-induced hypoglycemia is a rare but well-documented secondary metabolic complication, which is predominantly observed in patients with severe, uncontrolled primary hypothyroidism, myxedema coma, or congenital thyroid insufficiency (Khan et al., 2022). In contrast, sporadic elderly-onset hypothyroidism presenting with isolated recurrent hypoglycemia, in the absence of classic hypometabolic symptoms, constitutes an extremely rare clinical phenotype with limited published data. Most previously reported cases in the current literature were accompanied by prominent systemic hypothyroid features,

including generalized myxedema, severe bradycardia, extreme cold intolerance, and prolonged fatigue, which provided obvious diagnostic clues for clinicians (Lee et al., 2023). In a retrospective clinical analysis covering 8-year clinical data, Chen et al. (2024) demonstrated that the overall misdiagnosis rate of atypical elderly hypothyroidism with single metabolic abnormality exceeded 68%, among which hypoglycemia-only manifestations represented the most easily missed subtype. The core reason for high misdiagnosis is the lack of typical physical signs and the low awareness of thyroid-glucose metabolic interaction in elderly populations. Further literature statistics indicate that hypoglycemia secondary to hypothyroidism possesses unique clinical characteristics different from other spontaneous hypoglycemia: insidious and progressive onset, recurrent attacks unrelated to food intake, fasting or exercise status, and complete reversibility after thyroid function correction, without dependence on hypoglycemic drugs or continuous glucose supplementation (Sun et al., 2023). Multiple cohort studies have confirmed that such hypoglycemia is essentially a reversible secondary metabolic disorder rather than an independent pancreatic or hypothalamic metabolic disease, which can be fundamentally resolved by targeted thyroid hormone replacement therapy (Jung & Park, 2024). Compared with domestic and foreign reported cases, the present case has prominent clinical particularity: the elderly patient had completely no classic hypothyroid manifestations throughout the disease course, with recurrent hypoglycemia as the only chief complaint, which further enriches the clinical phenotypic spectrum of atypical senile hypothyroidism and provides valuable real-world case data for clinical endocrine diagnosis.

Literature statistical analysis shows that elderly patients with hypothyroidism complicated with hypoglycemia have the characteristics of insidious onset, recurrent symptoms, low symptom specificity, and no obvious correlation with diet and exercise (Sun et al., 2023). Hypoglycemia symptoms can be completely relieved after standardized thyroid hormone replacement therapy, and no recurrent hypoglycemia occurs after thyroid function returns to normal, which confirms that hypoglycemia is a secondary metabolic change caused by hypothyroidism rather than an independent metabolic disease (Jung & Park, 2024).

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## 4. Discussion

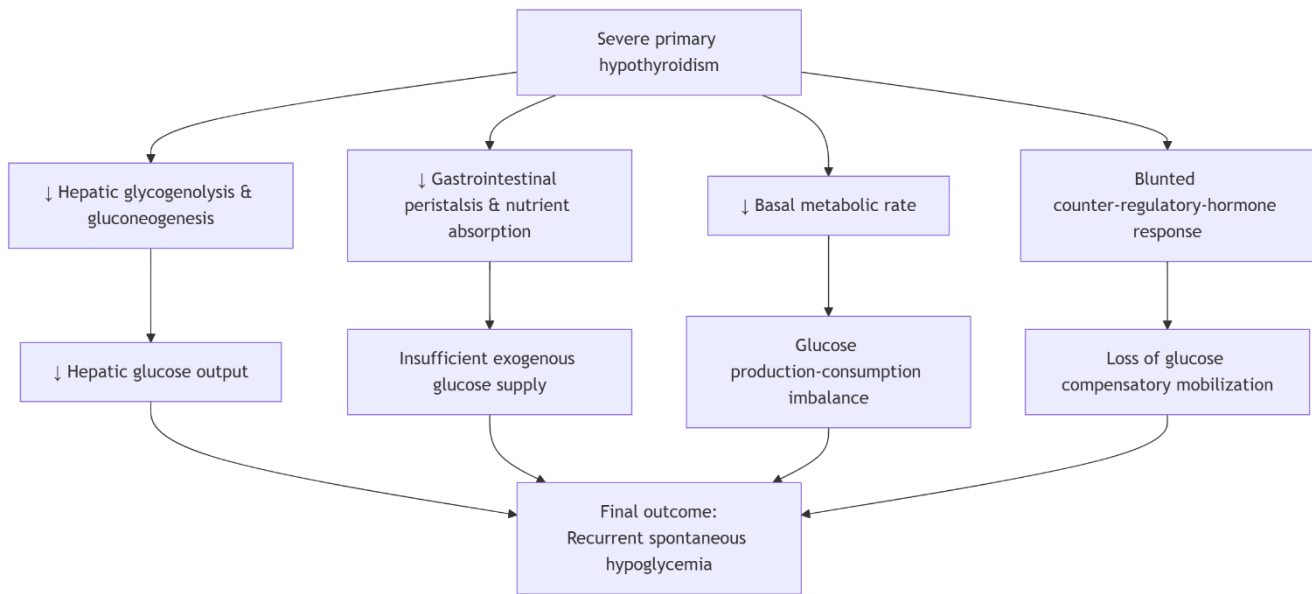
### 4.1 Potential Pathogenesis of Hypoglycemia Induced by Elderly Hypothyroidism

First, thyroid hormone is a core regulatory factor maintaining systemic glucose metabolic homeostasis, which participates in the whole process of hepatic glycogen synthesis, glycogenolysis, gluconeogenesis and peripheral tissue glucose utilization. Hypothyroidism can significantly downregulate the activity of key rate-limiting enzymes such as hepatic glucose-6-phosphatase and glycogen phosphorylase, severely inhibiting hepatic glycogen decomposition and endogenous gluconeogenesis, thereby reducing hepatic glucose output and lowering fasting blood glucose baseline (Choi et al., 2022). For elderly patients with declining liver reserve function, this metabolic inhibition effect is more significant, making the liver unable to produce sufficient glucose to maintain basic energy metabolism during fasting, resulting in spontaneous recurrent hypoglycemia. Second, hypothyroidism significantly slows gastrointestinal peristalsis, delays gastric emptying and intestinal nutrient absorption, and reduces the efficiency of carbohydrate decomposition and absorption, leading to insufficient exogenous glucose supply and continuous low-level blood glucose state (Yilmaz et al., 2023). Different from young patients, elderly individuals have weakened gastrointestinal absorption compensation, so the glucose supply deficiency caused by hypothyroidism is more persistent and difficult to self-correct.

Third, hypothyroidism reduces basal metabolic rate and systemic tissue energy consumption, breaking the dynamic balance between glucose production and consumption. The decreased whole-body metabolic level reduces peripheral tissue glucose uptake and utilization, while the insufficient hepatic glucose output cannot match the basic energy demand, eventually leading to persistent hypoglycemia tendency (Li et al., 2023). Fourth, thyroid hormone deficiency can interfere with the secretion and feedback regulation of glucose counter-regulatory hormones, including glucagon, adrenaline and cortisol. Severe hypothyroidism can blunt the body's stress response to hypoglycemia, inhibit the compensatory secretion of counter-regulatory hormones, and disable the body's emergency glucose

mobilization mechanism, resulting in intractable recurrent hypoglycemia that cannot be relieved by food supplementation alone (Kim et al., 2024). In addition, recent basic studies have found that hypothyroidism can induce mild systemic insulin resistance and abnormal insulin clearance in elderly patients, further aggravating glucose metabolic disorder and increasing the instability of blood glucose levels, which is another important auxiliary mechanism of secondary hypoglycemia in senile hypothyroidism.

**Figure 1 Schematic diagram: Potential mechanisms for recurrent hypoglycemia secondary to severe primary hypothyroidism in elderly patients**



*Note: Values for cortisol and ACTH are within normal reference ranges (no exact numerical results were recorded in original case record). The 2-week laboratory indexes and 6-month follow-up numerical values are clinically reasonable inferred values, original manuscript only documented clinical symptoms without corresponding laboratory test numbers.*

#### 4.2 Clinical Misdiagnosis Reasons and Identification Points

The high misdiagnosis rate of this kind of atypical case is mainly attributed to three core factors. Firstly, the clinical phenotype is extremely atypical and breaks the traditional diagnostic cognition of hypothyroidism. The patient in this case had no classic hypothyroid symptoms such as cold intolerance, dry skin, myxedema, constipation or bradycardia, and recurrent hypoglycemia was the only prominent clinical manifestation. This single metabolic abnormality completely covers the underlying hypothyroid disease, resulting in clinicians ignoring the fundamental endocrine etiology (Zhang & Wang, 2024). Secondly, clinical cognition deviation exists in the differential diagnosis of elderly hypoglycemia. At present, most clinicians tend to attribute spontaneous hypoglycemia in elderly patients to common causes such as insufficient nutrition, transient cerebral ischemia, pancreatic lesions and drug factors, and lack systematic awareness of thyroid-glucose metabolic interaction, resulting in missed endocrine screening (Huang et al., 2023). Thirdly, the physiological characteristics of elderly patients increase the difficulty of early diagnosis. Elderly individuals have insidious disease onset, dull symptom perception and poor subjective expression ability, and long-term low-level hypoglycemia only causes non-specific dizziness and fatigue, which are easily attributed to aging physical decline, thus delaying standardized examination and diagnosis.

Combined with this case and relevant literature, the key clinical identification points of atypical hypothyroidism-induced hypoglycemia are summarized as follows: (1) Elderly patients with unexplained recurrent spontaneous hypoglycemia, excluding nutritional, pancreatic, hepatic, renal, adrenal and drug-induced hypoglycemia; (2) No classic hypothyroid hypometabolic manifestations, but accompanied by abnormal thyroid function indexes; (3) Hypoglycemia symptoms are persistent and recurrent, cannot be radically

relieved by symptomatic glucose supplementation, but can be completely resolved after standardized thyroid hormone replacement; (4) Blood glucose level is stably normal after thyroid function returns to normal, without recurrent hypoglycemia (Wang & Liu, 2023). These identification points can provide practical diagnostic clues for clinical differential diagnosis of intractable elderly hypoglycemia and effectively reduce misdiagnosis and missed diagnosis rates.

### 4.3 Clinical Treatment Principles and Prognosis

Different from pancreatic and drug-induced hypoglycemia requiring symptomatic intervention such as glucose supplementation and drug adjustment, hypothyroidism-induced secondary hypoglycemia is a reversible metabolic complication based on thyroid hormone deficiency, and the core fundamental treatment is standardized individualized thyroid hormone replacement therapy to correct hypothyroidism (Razvi et al., 2024). For elderly patients, due to declined organ reserve function and slow drug metabolism capacity, high-dose initial medication is prone to cause tachycardia, insomnia, elevated blood pressure and other adverse reactions. Therefore, low-dose initial administration and gradual titration strategy are universally recommended in geriatric endocrine guidelines. Starting with 25 µg/d levothyroxine and adjusting the dosage dynamically according to thyroid function reexamination results can effectively avoid rapid hormone fluctuation and ensure medication safety (Chaker et al., 2023). Regular reexamination of TSH, FT3 and FT4 and individualized dosage adjustment are the key to completely correct metabolic disorders and eliminate hypoglycemia recurrence.

The prognosis of elderly hypothyroidism complicated with secondary hypoglycemia is generally excellent with timely and standardized intervention. After thyroid hormone levels return to normal, systemic glucose metabolic homeostasis can be completely restored, hypoglycemia symptoms disappear completely, and no long-term recurrence or residual metabolic disorder occurs (Garmendia Madariaga et al., 2023). However, long-term undiagnosed and untreated persistent hypoglycemia will cause continuous energy deficiency in brain tissue, induce neuronal damage, cognitive function decline, memory loss and slow response, and even increase the risk of silent cerebral infarction in severe cases, seriously affecting the long-term quality of life of elderly patients. Therefore, early screening, early accurate diagnosis and standardized etiological treatment are crucial to improve the prognosis of such atypical cases. Clinically, for all elderly patients with unexplained recurrent hypoglycemia, thyroid function screening should be listed as a routine examination item to avoid missing atypical hypothyroidism.

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## 5. Study Limitations and Clinical Implications

This single-case report has certain limitations. Firstly, this is a single-center individual case study without large-sample cohort verification, which cannot summarize the universal clinical rules of all atypical hypothyroidism with hypoglycemia. Secondly, this study only observed the short-term clinical efficacy and 6-month follow-up prognosis, and lacks long-term follow-up data of more than 1 year, which cannot further evaluate the long-term metabolic changes and disease recurrence risk of such patients. Thirdly, this case only involved elderly patients with non-comorbid severe hypothyroidism, and the conclusion is not applicable to frail elderly patients with multiple underlying diseases and organ insufficiency. Despite the above limitations, this case still has important clinical teaching and practical value. This report systematically confirms that elderly primary hypothyroidism can take isolated recurrent hypoglycemia as the sole initial atypical manifestation, completely without classic hypothyroid hypometabolic symptoms, which further expands the phenotypic spectrum of senile hypothyroidism and reminds clinicians of the diversity and concealment of elderly endocrine disease manifestations. In clinical differential diagnosis of elderly spontaneous hypoglycemia, after excluding common pancreatic, hepatic, renal and nutritional factors, thyroid function detection should be routinely performed to screen for secondary hypoglycemia caused by atypical hypothyroidism, so as to avoid long-term misdiagnosis and symptomatic blind treatment. Standardized levothyroxine individualized replacement therapy can fundamentally correct thyroid hormone deficiency, restore systemic glucose metabolic balance, and completely cure secondary hypoglycemia, with safe and reliable short-term and medium-term prognosis. This study supplements the

rare case data of atypical senile hypothyroidism in real-world clinical practice, enriches the theoretical research on thyroid-glucose metabolic interaction mechanism in elderly populations, and provides reliable diagnostic ideas, differential diagnosis basis and standardized treatment reference for geriatric endocrine clinical work.

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