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Werner Syndrome Masquerading as Type 2 Diabetes: A Diagnostic Odyssey Leading to Precision Medicine

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Conflict of interest: None declared

Patient: Male, 50-year-old
Final Diagnosis: Rare disease
Symptoms: Werner syndrome
Clinical Procedure: Polydipsia • polyphagia • polyuria • weigh loss
Specialty: —

Objective: Genetics

Background: Werner syndrome (WS), also called adult progeria, is a rare hereditary progeroid disorder characterized by highly heterogeneous clinical manifestations, often leading to delayed diagnosis or misdiagnosis. Diabetes mellitus affects ~55% of patients with WS but is frequently misclassified as type 1 or type 2 diabetes due to overlapping metabolic features. This report aims to highlight the diagnostic challenges of WS-associated diabetes, emphasize the role of genetic testing in establishing the correct etiological diagnosis, and demonstrate how precision treatment concerning the underlying genetic defect can improve glycemic control and patient outcomes.

Case Report: A 50-year-old man was admitted to the First Affiliated Hospital of Xinjiang Medical University in September 2024 with a history of poorly controlled diabetes. He exhibited diabetes mellitus, hyperlipidemia, hoarseness, and atrophy of subcutaneous fat and muscles in the face and limbs. His parents were consanguineous. Genetic sequencing revealed a homozygous mutation in the *WRN* gene (c.1846G>C, p.Ala616Pro), confirming the diagnosis of WS. His diabetes was subsequently reclassified as a specific type of monogenic diabetes rather than type 2 diabetes, and an appropriate precision treatment plan was developed.

Conclusions: This case underscores the importance of recognizing the association between WS and diabetes mellitus, particularly in patients with signs of premature aging. Early diagnosis and individualized management strategies are crucial for improving patient outcomes. Genetic testing plays a key role in confirming the diagnosis of WS and preventing misdiagnosis as type 1 or type 2 diabetes. This case also highlights the importance of identifying and managing rare diseases in clinical practice.

Keywords: Werner Syndrome • Progeria, Adult • Genetic Testing • Case Reports

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Introduction

Werner syndrome (WS), also called adult progeria, is a rare autosomal recessive genetic disorder first described by Otto Werner in 1904 [1]. It has an extremely low estimated incidence, approximately 1 in 10 million to 1 in 1 million individuals. Relatively higher incidence rates have been reported in regions such as Japan, Sardinia, India, and Pakistan. The primary characteristics of WS include premature aging and highly heterogeneous clinical manifestations. The disease is mainly caused by mutations in the *WRN* or *LMNA* genes. The *WRN* gene encodes the WRN protein [1], a member of the RecQ family of DNA helicases. This protein is involved in critical cellular processes, including DNA replication, repair, telomere maintenance, and apoptosis; it thus affects multiple physiological systems and results in diverse clinical manifestations [2]. The clinical features of WS are highly variable and heterogeneous. Patients are often misdiagnosed with type 1 or type 2 diabetes due to emaciation, lipid metabolism disorders, and insulin resistance [3,4]. Such misdiagnosis can lead to inappropriate treatment strategies and poor glycemic control, further exacerbating disease progression [5].

This report describes the diagnostic and therapeutic course of a patient with WS who exhibited hyperglycemia and hyperlipidemia. The case highlights the critical importance of recognizing the distinctive clinical features and genetic basis of WS, particularly in the context of diabetes management. Through detailed clinical evaluation, genetic testing, and individualized therapeutic interventions, we aim to improve awareness of this rare form of diabetes associated with a genetic disorder. This case also underscores the importance of early diagnosis and personalized management strategies.

Case Report

A 50-year-old man presented with polydipsia, polyuria, polyphagia, and weight loss beginning in 2013. He was admitted to our hospital, where his random blood glucose level was 19.5 mmol/L and triglyceride level was 19.53 mmol/L. He was initially diagnosed with type 2 diabetes, hyperlipidemia, osteopenia, and lacunar infarction. His treatment regimen included subcutaneous injections of biphasic insulin aspart 30 (18 units before breakfast and 16 units before dinner), oral metformin (0.5 g, 3 times daily), and fenofibrate (0.2 g once daily). Despite these interventions, glycemic control remained poor, and he was repeatedly admitted to tertiary hospitals over the following years. He received multiple insulin regimens, including premixed insulin, basal insulin, and basal-bolus insulin therapy, along with oral antidiabetic agents such as biguanides, sulfonylureas, and αglucosidase inhibitors. However, glycemic control remained suboptimal.

In 2018, the patient developed fatigue, progressive hair graying, alopecia, and reduced hair density; he reported feeling physically weaker than his peers. In 2019, he exhibited a symmetrical reduction in subcutaneous fat in the limbs and muscle atrophy, particularly involving the palms and soles. The following year, he experienced numbness in the limbs and blurred vision in both eyes. In 2022, after evaluation in our department, he was diagnosed with familial-partial-lipodystrophy-associated diabetes, diabetic peripheral neuropathy, and nonproliferative diabetic retinopathy. His treatment regimen was adjusted to include insulin aspart (16 units before each meal) and insulin degludec (38 units at bedtime), whereas his oral antidiabetic medications remained unchanged.

In 2023, the patient noticed a pronounced reduction in subcutaneous fat in the limbs, accompanied by facial fat atrophy and sunken cheeks. Genetic testing revealed a homozygous missense mutation in the *WRN* gene (c.1846G>C, p.Ala616Pro), confirming the diagnosis of WS. On March 19, 2024, he visited Peking Union Medical College Hospital and was diagnosed with generalized lipodystrophy, diabetes, stage II diabetic nephropathy, diabetic peripheral neuropathy, diabetic autonomic neuropathy, multiple atherosclerotic plaques, hyperlipidemia, cataracts, osteopenia, and grade 3 hypertension (very high risk). Six months later, he was readmitted to our department due to persistently poor glycemic control. Throughout the course of his illness, he denied hypoglycemic symptoms such as tremors and sweating. His triglyceride level peaked at 26.88 mmol/L, and he experienced weight loss of 20 kg.

The patient had achieved normal developmental milestones during infancy, including head control at 3 months, rolling over at 6 months, crawling at 8 months, and independent standing at 12 months. During childhood, his growth rate was comparable to that of his peers; however, he did not experience a pubertal growth spurt, resulting in short stature (final height: 170 cm). He was diagnosed with hypertension in 2013 (peak blood pressure: 160/110 mm Hg) but was nonadherent to antihypertensive therapy. Bilateral high-frequency sensorineural hearing loss was diagnosed in 2015 but remained untreated. In 2020, he was diagnosed with an anxiety disorder and treated with lorazepam, which was subsequently discontinued. He also reported poor sleep quality. Cataracts were diagnosed in 2024, and no surgical intervention had been performed. His surgical history included an appendectomy in February 2002 and cholecystectomy in 2016, both with uneventful recoveries.

On physical examination, the patient was 50 years old chronologically but appeared to be in his sixties due to premature aging features characteristic of WS. He had a high-pitched, hoarse voice. His height was 170 cm, weight was 62 kg, body mass index was 21 kg/m^2, and blood pressure was 145/94 mm Hg. He was conscious, alert, and fully oriented, with a Glasgow Coma

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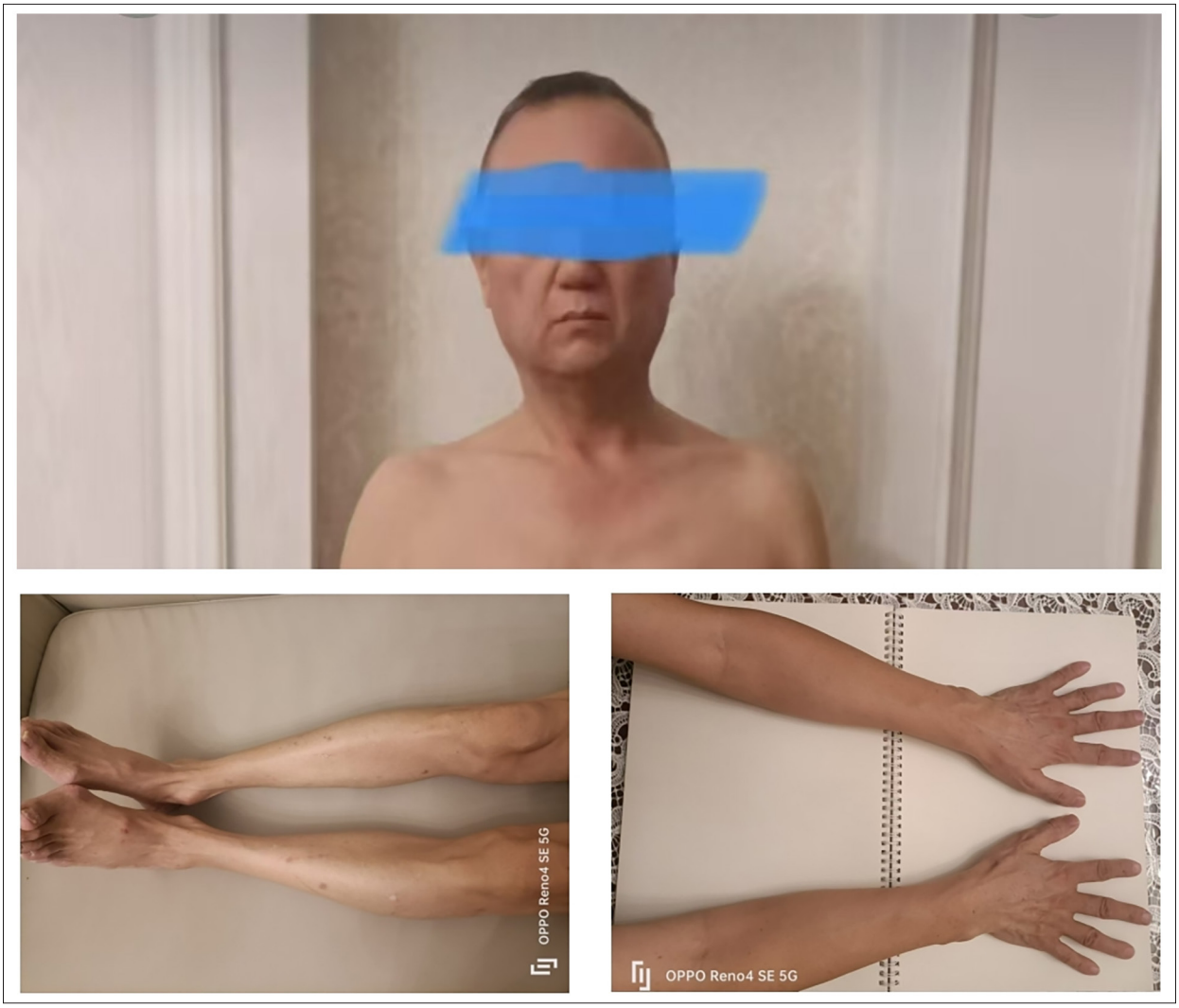

Figure 1. Atrophy of subcutaneous fat in the buttocks and limbs, accompanied by pronounced muscle atrophy of the lateral gluteal regions.

Scale score of 15. His hair was sparse and gray-white. Mild bilateral proptosis was present, and subcutaneous fat in the cheeks was greatly reduced. He had a pointed nose, resulting in a characteristic bird-like facial appearance. His limbs were slender; the skin of both feet appeared mildly erythematous, taut, and thin. Pronounced atrophy of subcutaneous fat was observed in the buttocks and limbs, accompanied by substantial muscle atrophy over the lateral gluteal regions (**Figure 1**). No acanthosis nigricans was observed.

Laboratory investigations revealed hypertriglyceridemia, insulin resistance with elevated blood glucose and glycated hemoglobin A1c levels, normal ketone body and plasma lactic acid levels, and negative diabetes-related autoantibodies (**Table 1**).

Whole-body magnetic resonance imaging demonstrated symmetrical atrophy of subcutaneous fat throughout the body, except in the chest region, as well as mild hepatic steatosis. Ultrasound examination of the neck and lower-extremity arteries revealed atherosclerosis with multiple plaques. Electromyography showed bilateral motor and sensory nerve damage in the lower limbs. Fundus photography confirmed diabetic retinopathy. Bone mineral density assessment demonstrated osteopenia, with a lowest T-score of -1.6. Electronic laryngoscopy revealed a raised lesion on the left side of the posterior nasopharyngeal wall with a generally smooth surface. Genetic testing by whole-exome sequencing identified a homozygous *WRN* gene mutation, c.1846G>C (p.Ala616Pro) (**Figure 2**). The patient's daughter, son, and 1 brother carried the mutation in the heterozygous state.

The treatment regimen was adjusted to include subcutaneous insulin degludec at 30 IU at bedtime and subcutaneous insulin lispro at 14 IU before each meal. Oral medications included

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Table 1. Laboratory investigations.

Test	Upon admission		Normal range
	Fasting	Postprandial	
PG	11.15 mmol/L	18.36 mmol/L	
CP	1.58 ng/mL	3.09 ng/L	
HbA1c	10.90%		4.8%-6.0%
TG	19.3 mmol/L		0.29-1.83 mmol/L
TC	8.40 mmol/L		2.8-5.7 mmol/L
KB	0.3 mmol/L		< 0.5 mmol/L
LA	1.3 mmol/L		0.7-2.2 mmol/L
ICA	(-)		(-)
IAA	(-)		(-)
ATA	(-)		(-)
GADA	(-)		(-)
T	3.37 ng/mL		4.94-32.01 ng/mL
LH	4.9 IU/L		0.57-12.07 IU/L
FSH	8.38 IU/L		2.64-13.13 IU/L

Abbreviations (alphabetical order): ATA, anti-tyrosine phosphatase antibody; CP, serum C-peptide; FSH, follicle-stimulating hormone; GADA, glutamic acid decarboxylase antibody; HbA1c, glycated hemoglobin A1c; IAA, insulin autoantibody; ICA, islet cell antibody; KB, ketone body; LA, lactic acid; LH, luteinizing hormone; PG, plasma glucose; T, testosterone; TC, total cholesterol; TG, triglycerides.

metformin 0.5 g, 4 times daily; pioglitazone 45 mg once daily; dapagliflozin 10 mg once daily; fenofibrate 0.2 g once daily; acipimox 0.25 g twice daily; and atorvastatin 10 mg at bedtime, along with additional symptomatic treatments as needed.

Discussion

WS is a genetic disorder characterized by premature aging. Typically, the earliest sign of WS is the absence of a pubertal growth spurt, resulting in short stature and low body weight [6]. During adulthood, patients gradually develop a range of clinical manifestations, including skin and hair abnormalities, voice changes, bilateral cataracts, diabetes mellitus, dyslipidemia, hypogonadism, osteoporosis, and atherosclerosis [7]. Additionally, patients with WS display an increased risk of developing malignant tumors and often die from malignancies or cardiovascular disease during middle age.

In the initial evaluation of this patient, the etiological classification of diabetes was unclear, leading to misdiagnosis with type 2 diabetes. The primary clinical manifestation was hyperglycemia accompanied by abnormalities in glucose and lipid metabolism.

The patient's nonobese body habitus and greatly elevated triglyceride levels suggested a strong association between lipid abnormalities and insulin resistance. Endocrine and metabolic abnormalities are common in patients with WS [8], predominantly manifesting as diabetes and hyperlipidemia; diabetes is the most common comorbidity. Yokote [9] found that *WRN*-deficient mice were more susceptible to hyperglycemia, insulin resistance, and dyslipidemia compared with wild-type mice fed the same diet. These findings suggest a role for the *WRN* gene in glucose and lipid homeostasis, although it remains unclear whether insulin resistance and diabetes in WS are related to telomere shortening. Some authors have speculated that insulin resistance in patients with WS is linked to reduced insulin receptor expression, impaired intracellular signaling following insulin-receptor binding, and visceral fat accumulation. At the time of the 2022 regimen adjustment, an insulin dose of 86 U/day failed to achieve satisfactory glycemic control, and the fasting C-peptide level did not decrease, suggesting strong insulin resistance. After treatment with metformin and pioglitazone to improve insulin sensitivity, the insulin requirement was substantially reduced, and glycemic control became relatively stable.

A review of the patient's growth and developmental history revealed the absence of a pubertal growth spurt. During

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Whole Exome Sequencing Results

Genetic variants potentially associated with clinical phenotype but with insufficient pathogenic evidence

Variant Gene	Nucleotide/Amino Acid Change	RS/HGMD-ID	Hom/Het/Hemi	ACMG Classification	DLKSDK-Daughter	DLKSDK-Son	DLKSDK-Brother
WRNNM_000553.6chr8:31091846	exon16c.1846G>Cp.Ala616Pro	-	Hom	Uncertain significance (Warm)	Het	Het	Het
SCNN1ANM_001038.6chr12:6354485	exon8c.1313G>Ap.Arg438Gln	rs551306856	Het	Uncertain significance (Tepid)	Het	Het	Het

*Hom: homozygous mutation; Het: heterozygous mutation; Hemi: hemizygous mutation

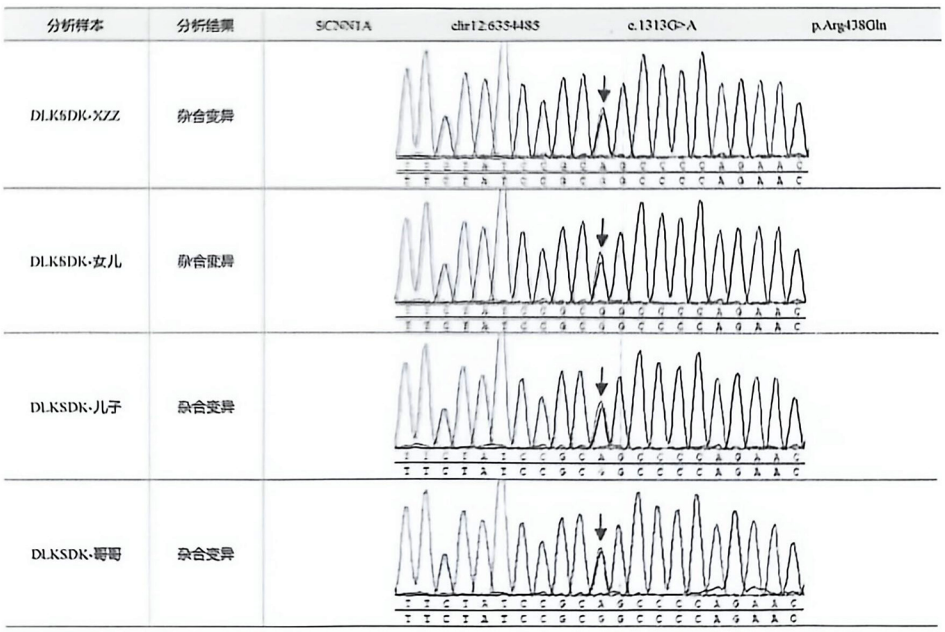

Figure 2. Whole-exome sequencing revealed a homozygous *WRN* gene mutation, c.1846G>C (p.Ala616Pro). The patient's daughter, son, and 1 brother carried the mutation in the heterozygous state.

adulthood, signs of premature aging, including alopecia and hair graying, gradually developed. The patient had been diagnosed with bilateral cataracts 1 year earlier, indicating early connective tissue involvement. Early clinical manifestations of WS are often subtle and easily overlooked in routine practice. Hyperglycemia, a key manifestation of underlying disease, is frequently detected incidentally. Oshima and Hisama [2] reported that approximately 55% of patients with WS exhibit diabetes mellitus. During glucose tolerance tests performed in 53 patients with WS, Tsukamoto et al found that 33% had hyperinsulinemia with a basal insulin level of 20 μU/mL; 67% exhibited peak insulin levels as high as 200 μU/mL [10]. Clinicians should maintain a high index of suspicion for genetically associated forms of diabetes in young, nonobese patients who primarily present with insulin resistance and evidence of pancreatic endocrine dysfunction.

This case substantially differed from the typical clinical features of both type 1 and type 2 diabetes. Although the patient developed diabetes in adulthood, he had a small body habitus and did not present with ketoacidosis at disease onset. There was no evidence of pancreatic autoantibodies or clinically significant pancreatic β-cell dysfunction, and the stimulated C-peptide level was > 0.6 nmol/L, suggesting type 1 diabetes was unlikely [11]. Additionally, although he required a daily insulin dose exceeding 80 U and had a preserved C-peptide level, the patient exhibited pronounced insulin resistance that could not be fully explained by type 2 diabetes alone. Given his slender limbs and loss of subcutaneous fat, lipodystrophic diabetes was also considered. However, the absence of obvious acanthosis nigricans, acromegaloid features, characteristic skin changes such as xanthomas, and major manifestations (eg, hepatosplenomegaly) complicated the diagnostic process.

According to American Diabetes Association classification criteria, patients displaying early-onset diabetes, nonobesity, negative pancreatic autoantibodies, and severe insulin resistance associated with distinctive facial features or developmental abnormalities should be evaluated for monogenic diabetes or secondary diabetes resulting from genetic syndromes. Our patient exhibited several characteristic features, including a prematurely aged appearance, sparse graying hair, severe loss of subcutaneous fat, early-onset cataracts, and decreased skin elasticity. Whole-exome sequencing identified a homozygous

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WRN gene mutation, c.1846G>C (p.Ala616Pro). His daughter, son, and 1 brother carried the mutation in the heterozygous state. These findings confirmed the diagnosis of WS caused by a *WRN* gene mutation. Classical WS is caused by homozygous or compound heterozygous loss-of-function mutations in the *WRN* gene. *WRN* is the only known gene in which mutations cause classical WS, and WS is the only known genetic disorder caused by null mutations in *WRN*. The *WRN* locus is located on chromosome 8p12 and consists of 34 coding exons spanning approximately 140 kb. More than 70 disease-causing mutations have been identified in patients with classical WS worldwide [1].

WS affects multiple organ systems and presents with a complex and heterogeneous clinical phenotype that can be mistaken for other syndromes involving distinctive facial features. However, it also has unique clinical characteristics. Familiarity with the diagnostic criteria is essential for accurate diagnosis. In 2014, the International Registry of Werner Syndrome established specific diagnostic criteria for the disorder [2]. The patient in the present case exhibited typical features of WS, including more than 3 cardinal signs and symptoms (distinctive facial features, short stature, premature hair graying, early-onset cataracts, and characteristic skin changes), as well as other common manifestations such as voice changes and abnormalities in glucose and lipid metabolism. Genetic testing played a crucial role in confirming the diagnosis.

Studies have shown that insulin therapy alone is often insufficient to improve insulin resistance in diabetes associated with WS. Metformin and thiazolidinediones can effectively enhance insulin sensitivity [1,12]. In the present case, the patient previously required high doses of insulin while maintaining poor glycemic control. After the addition of metformin and pioglitazone to improve insulin resistance, the insulin requirement decreased and glycemic control became relatively stable. However, thiazolidinediones have been reported to adversely affect bone health and can increase the risk of certain malignancies [13]. Therefore, careful risk assessment is warranted during long-term treatment with these agents. Dipeptidyl peptidase-4 inhibitors and glucagon-like peptide-1 receptor agonists have also demonstrated efficacy in some patients with WS [14,15]. Additionally, diet and exercise are essential for preventing increases in visceral fat and losses of skeletal muscle mass, thereby helping to slow diabetes progression in these patients [16].

Patients with WS have substantially increased risks of age-related diseases, including malignancies and cardiovascular disorders. Although no tumors have been identified in our patient to date, the presence of peripheral vascular atherosclerosis suggests an elevated cardiovascular risk. Regular clinical follow-up is recommended to facilitate the early detection and management of potential complications.

Strengths and Limitations of This Case

This case report contributes several unique aspects to the existing literature on WS-associated diabetes. First, this case is among few reported cases of WS originating from Central Asia (Xinjiang, China), thereby expanding the geographic and ethnic spectrum of documented *WRN* mutations. Most previously reported cases have originated from Japan, Sardinia, India, Pakistan, and the Middle East.

Second, the patient experienced a prolonged diagnostic delay of 11 years (2013-2024), during which his diabetes was misclassified as type 2 diabetes. This aspect highlights the persistent challenge of recognizing WS in clinical practice, particularly when features of premature aging emerge gradually and are attributed to unrelated conditions.

Third, we identified a homozygous missense mutation, c.1846G>C (p.Ala616Pro), in the *WRN* gene. To our knowledge, this specific mutation has not been previously reported, thus expanding the mutational spectrum of *WRN*. The only structurally similar mutation reported within the same exon region is the nonsense variant c.1111G>T (p.Glu371*) described in a Lebanese family by Jaafar et al [17], which differs in both location and functional consequence (nonsense versus missense).

Fourth, this case demonstrates successful precision therapy guided by genetic diagnosis. The treatment strategy was modified from insulin monotherapy (86 U/day with poor glycemic control) to a combination of basal-bolus insulin, metformin, pioglitazone, and a sodium-glucose cotransporter 2 inhibitor, resulting in reduced insulin requirements and improved glycemic stability. This approach is consistent with emerging evidence that insulin sensitizers may be more effective than insulin alone in managing insulin resistance associated with WS.

This report has several limitations. First, as a single-case report, its findings require validation in larger cohorts; however, the rarity of WS inherently limits the feasibility of large case series.

Second, follow-up after genetic diagnosis was relatively short. Although glycemic stability was observed during the 3-month period after treatment modification, longer-term outcomes (eg, cardiovascular events, malignancy risk, and survival) remain unknown.

Third, functional studies were not performed to validate the effect of the c.1846G>C mutation on *WRN* helicase activity. Pathogenicity was inferred from clinical findings and in silico prediction tools, including SIFT and PolyPhen-2.

Fourth, family screening was incomplete because only 3 first-degree relatives underwent genetic testing, and extended pedigree analysis was limited by geographic dispersion.

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Finally, patient-reported outcomes were not assessed via standardized quality-of-life instruments.

Conclusions

This case highlights the importance of recognizing the diverse clinical manifestations of WS and the critical role of genetic testing in establishing an accurate diagnosis. Identification of the *WRN* gene mutation c.1846G>C (p.Ala616Pro) underscores the need for precise etiological classification to avoid misdiagnosis as type 1 or type 2 diabetes. Early recognition and individualized treatment strategies may improve glycemic control and facilitate comprehensive management of this rare genetic disorder.

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Patient Consent

Written informed consent was obtained from the patient for publication of this report.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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