



The First Documented Iraqi Case of Progeria: Clinical Discovery, Rare-Disease Medicine, and a Vision for Transforming Iraqi Medicine from Knowledge Consumer to Knowledge Produce

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OPEN ACCESS

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Received Date: 11 Sep 2026

Accepted Date: 09 Oct 2026

Published Date: 10 Oct 2026

Citation:

Aamir Jalal Al-Mosawi. The First Documented Iraqi Case of Progeria: Clinical Discovery, Rare-Disease Medicine, and a Vision for Transforming Iraqi Medicine from Knowledge Consumer to Knowledge Produce. *WebLog J Genet Genomic Res.* wjggr.2026.j1003. <https://doi.org/10.5281/zenodo.23337005>

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Abstract

Background: Hutchinson-Gilford progeria syndrome is an exceptionally rare genetic disorder, usually caused by a pathogenic variant in the LMNA gene, and characterized by progressive features of accelerated aging beginning in early childhood. Most affected children appear clinically normal at birth and during early infancy. Although Hutchinson-Gilford progeria syndrome has been recognized for more than a century, it remains extremely rare and individual case reports continue to contribute to its clinical and geographical documentation.

Objective: To report the first documented case of Hutchinson-Gilford progeria syndrome in Iraq and describe its characteristic clinical phenotype.

Patient and Methods: A boy who had apparently normal development during birth, infancy, and early childhood developed a progressive severe progeroid phenotype at approximately 10 years of age. Clinical assessment revealed characteristic physical manifestations of premature aging, including body habitus, skin, hair, facial appearance, eyes, and dentition. The clinical findings were compared with the established phenotype of Hutchinson-Gilford progeria syndrome and other childhood-onset progeroid disorders.

Results: The predominant abnormality was progressive generalized loss of subcutaneous fat, resulting in a markedly thin, skeletal appearance. Additional findings included thin, tight, atrophic skin with prominent superficial blood vessels, alopecia, markedly aged facial features, prominent eyes with bilateral ectropion, and abnormal dentition.

The overall clinical phenotype was strongly suggestive of Hutchinson-Gilford progeria syndrome.

Conclusion: This report documents, to our knowledge, the first reported case of Hutchinson-Gilford progeria syndrome in Iraq. The case emphasizes the importance of careful clinical recognition and documentation of rare progeroid phenotypes. In rare-disease medicine, detailed phenotyping remains essential for diagnosis and provides an important foundation for subsequent molecular investigation and inclusion of previously under-recognized patients in the international medical literature.

Keywords: Progeria; Iraq; Rare Disease

Introduction

Progeria, or Hutchinson-Gilford progeria syndrome, is the classic form of progeroid syndrome. It is a very rare genetic disorder, usually caused by a de novo pathogenic variant in the LMNA gene and inherited in an autosomal-dominant manner. It is characterized by features of accelerated aging beginning in early childhood. Most children with progeria appear clinically normal at birth and during early infancy [1-5].

Progeria was first described by Jonathan Hutchinson (Figure-1A) in 1886 and subsequently described in greater detail by Hastings Gilford (Figure-1B) in 1897 [1, 2].



Figure 1A: Jonathan Hutchinson (July 23, 1828-June, 23 1913), an English surgeon.

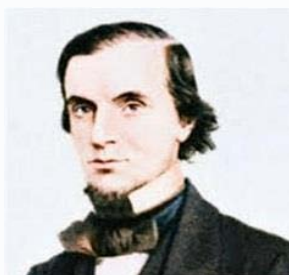


Figure 1B: Hastings Gilford (July 2, 1861-September 6, 1941), an English surgeon.



Figure 1C: Leonard Atkins (1922-2019), a Boston physician and pathologist.

Gilford introduced the term “progeria” in 1904. The condition is now generally known as Hutchinson-Gilford progeria syndrome.

The patient described by Gilford in 1897 exhibited wrinkled skin with an aged or senile appearance, baldness, and a combination of premature and immature development. Gilford noted that there was no mental retardation; although he described certain “psychic” characteristics that he considered reminiscent of old age.

In his 1904 publication “Progeria: a form of senilism,” Gilford introduced the term “progeria” to describe the characteristic premature senile degeneration observed in childhood. The word refers to premature old age and is derived from Greek roots associated with “before” and “old age.” Gilford classified the condition as a form of “senilism” because of the striking resemblance between the physical deterioration in affected children and the changes associated with normal aging.

Gilford also used the term “ateleiosis” to describe a contrasting

developmental condition characterized by persistent juvenile or immature characteristics into adulthood, which he described as “continuous youth.” Thus, in his 1904 work, Gilford contrasted ateleiosis (“continuous youth”) with progeria (“premature old age”) [3, 4].

In 1954, Leonard Atkins (Figure-1C) from Boston suggested that since the original description of cases of progeria by Hutchinson in 1886 and Gilford in 1897, only 22 cases have been reported in the medical literature. He reported an additional case with post-mortem findings.

According to Leonard Atkins childhood cases of progeria or Hutchinson-Gilford progeria syndrome show a conspicuous resemblance to each other. Patients appear normal at birth; dwarfism being observed within the first few years of life.

Patients also has characteristic facies with a beaklike nose, a relatively small face with receding chin, loss of hair, exophthalmos, prominent scalp veins, markedly decreased subcutaneous fat, muscular atrophy, arthritic joint deformities, shortened clavicles, coxa valga, and normal or above normal intelligence [6].

In 1972, Franklin L. DeBusk from Florida reviewed the world literature and counted 60 documented cases with Hutchinson-Gilford progeria syndrome in the medical literature. He also described four patients. He emphasized that patients with this syndrome have amazingly similar appearance, and may exhibit scleroderma-like skin with early growth retardation. Patients may also have reduced soft-tissue, poor modeling of long bones, and coxa valga [7].

In 2009, Coutinho *et al.*, emphasized the rarity of Hutchinson-Gilford progeria syndrome and reported the presence over 150 cases documented worldwide since its first description by Jonathan Hutchinson in 1886 [8].

Therefore, Hutchinson Gilford progeria syndrome is an exceptionally rare genetic disorder. Historically, the number of cases reported in the medical literature increased from 22 cases documented by Atkins in 1954 to approximately 60 cases reported by DeBusk in 1972 and more than 150 cases in subsequent literature reviews [6-8].

We have previously reported an uncountable number of the first described disorders in Iraq [9-87]. Here, we report the first documented case of Hutchinson Gilford progeria syndrome in Iraq.

Patients and Methods

At approximately 10 years of age, following an apparently normal birth and normal early infancy and toddlerhood, the boy (Figure-2) developed a progressive severe progeroid (premature-aging) phenotype, characterized by:

- Marked loss of subcutaneous fat (lipoatrophy), resulting in a very thin, skeletal appearance of the face and body.
- Thin, tight, atrophic skin with prominent superficial blood vessels.
- Alopecia/scalp hair loss and markedly aged facial features.
- Prominent eyes with bilateral ectropion, characterized by outward eversion of the lower eyelids and exposure of the palpebral conjunctiva.
- Abnormal dentition, contributing to the characteristic facial appearance (Figure 2A-2E).



Figure 2A:



Figure 2D:



Figure 2B:



Figure 2C:



Figure 2E:

Results

The predominant clinical abnormality is progressive generalized loss of subcutaneous fat associated with a severe premature-aging (progeroid) phenotype, suggesting a childhood-onset progeroid syndrome or progeroid lipodystrophy disorder such as Hutchinson-Gilford progeria syndrome and other genetic progeroid syndromes.

Discussion

The history of medicine is often remembered through major discoveries made in large research institutions, technologically advanced laboratories, and highly specialized centers. Yet an important part of medical progress has always originated elsewhere: at the bedside, in the careful observation of an unusual patient, and in the clinician's decision to investigate, document, compare, and publish what does not fit familiar diagnostic categories.

My clinical career and scholarly work represent an unusual example of this form of clinician-led discovery in Iraq.

Over approximately two decades, my work has focused particularly on rare and ultra-rare disorders, unusual clinical associations, dysmorphic syndromes, neurodevelopmental disorders, renal diseases, neurological conditions, and congenital malformations.

I have published more than 40 newly described clinical syndromes and disorders and many reports of very rare or ultra-rare disease presentations. These observations were derived primarily from direct patient care, clinical examination, imaging, follow-up, literature comparison, and publication rather than from large-scale genomic programs [9-87].

My published encyclopedia of new and ultra-rare disorders which has been translated into several European languages [80-86], explicitly describes this methodology as involving repeated recognition of distinctive phenotypes, exclusion of known disorders, corroborative neuroimaging and laboratory information, and eventual replication when additional cases became available. My observations were subsequently disseminated through peer-reviewed publications and academic monographs.

This approach is particularly important in rare-disease medicine because the first challenge is often not molecular confirmation but recognition.

This approach led to reports spanning several areas of medicine. My published encyclopedia [80-86] groups the discoveries into neurodevelopmental and psychiatric syndromes, autism-associated disorders, dysmorphic and multisystem syndromes, and renal and endocrine disorders.

Examples include a syndrome characterized by mental retardation, periventricular white-matter hyperintensity, optic atrophy and retinitis pigmentosa; a syndrome involving cerebral palsy, hydrocephalus and a KIDINS220 mutation; autism associated with precocious puberty; autism with focal cortical atrophy and epilepsy; facial dysmorphism associated with triphalangeal toes and unilateral renal agenesis; and an autosomal dominant form of nonsyndromic unilateral renal hypoplasia.

The first documented reports in Iraq

One of the most distinctive components of my experience was the accumulation of first documented reports in Iraq including numerous disorders for which the reports are identified as the first documented Iraqi cases. Among them are Bardet-Biedl syndrome, genetic vitamin-D-resistant hypophosphatemic rickets, Coffin-Siris syndrome, nephropathic cystinosis, Lowe oculo-cerebro-renal syndrome, Aicardi syndrome, Mostyn Embrey syndrome, Beckwith-Wiedemann syndrome, Berry-Treacher Collins syndrome, congenital Chevalier Jackson syndrome, Prader-Labhart-Willi syndrome, Lesch-Nyhan syndrome, Hurler-Scheie syndrome, Maroteaux-Lamy syndrome, nonsyndromal hemifacial microsomia, and Angelman syndrome.

The list continues with Niemann-Pick disease, congenital nephrotic syndrome of the Finnish type, Rett syndrome, Dandy-Walker syndrome, Townes-Brocks syndrome, Sanjad-Sakati-Richardson-Kirk syndrome, Adams-Oliver syndrome, extended Michelin tire baby syndrome, Ruprecht-Majewski-Bosma syndrome, Ekman-Lobstein syndrome, phenylketonuria, homocystinuria, Semmola-Meryon-Duchenne syndrome, congenital myotonic muscular dystrophy, congenital absence of the radii without thumb aplasia, Virchow-Seckel syndrome, congenital cystic adenomatoid lung malformation, pediatric Wohlfart-Kugelberg-Welander syndrome, childhood Huntington disease, Goldberg-Shprintzen syndrome, childhood Seeligmüller-Strümpell-Philip disease, Cornelia de Lange syndrome, pediatric Bartter syndrome, Béguez-César syndrome, dysimptomatic Wiskott-Aldrich syndrome, crossed unfused renal ectopia, agenesis of the corpus callosum with colpocephaly, Pallister-Hall syndrome, Noonan syndrome, the syndrome of congenital facial palsy and unilateral anotia, identified as a first documented Iraqi cases.

Thus, my contribution was not confined to a single specialty. It crossed pediatrics, neurology, nephrology, endocrinology, ophthalmology, radiology, clinical genetics, and dysmorphology.

An important distinction is that my work was not limited to documenting diseases already known elsewhere but absent from Iraq. Several reports also occupied unusual positions in the international literature.

I have described the 35th global case of cutis laxa type II (Debré type), the 130th case of Townes-Brocks syndrome, and the 52nd case of Ruprecht-Majewski-Bosma syndrome. It also describes the first Arab case of Aicardi syndrome associated with corneal opacity and the second global pediatric case of unilateral Vogt-Koyanagi-Harada syndrome.

Other observations extended recognized phenotypes. Goldberg-Shprintzen syndrome was reported with unilateral anorchia in one case, while the extended Michelin tire baby syndrome was associated with mental retardation, cryptorchidism, and hearing impairment [9-87].

The importance of these observations is that rarity itself was not the sole criterion for publication. An unusual association could provide information about the boundaries of a syndrome and suggest that the recognized phenotype was broader than previously appreciated.

This paper documents, to our knowledge, the first reported case of Hutchinson-Gilford progeria in Iraq.

This case also reflects the broader value of clinician-led discovery. Careful observation, detailed phenotyping, literature comparison, longitudinal follow-up, and publication can transform an unusual clinical encounter into a lasting contribution to medical knowledge. In the genomic era, accurate clinical phenotyping remains essential because recognition of the phenotype may precede and guide molecular diagnosis.

More broadly, my experience with rare diseases in Iraq supports a vision of transforming Iraqi medicine from primarily a consumer of medical knowledge into a contributor and producer of new medical knowledge.

Essential principles are simple: recognize unusual patients, document carefully, investigate systematically, educate others, and contribute the findings to the international medical community.

Conclusion

Hutchinson-Gilford progeria syndrome is an exceptionally rare genetic disorder characterized by progressive premature aging beginning in childhood. This paper documents, to our knowledge, the first reported case of Hutchinson-Gilford progeria in Iraq. The patient's characteristic progeroid phenotype highlights the importance of careful clinical recognition and documentation of rare disorders, particularly in settings where molecular diagnostic facilities may be limited.

Acknowledgement

The author has the copyright of all the figures (Sketches) included in this paper.

All the necessary consents have been obtained.

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