

RESEARCH ARTICLE

The Twenty-First Reported Patient with Complete Remission of the Core Autistic Features: A Child with a Previously Unrecognized Autism–Craniofacial Dysmorphism Syndrome

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ABSTRACT

Background: Autism spectrum disorder is a heterogeneous neurodevelopmental disorder characterized by impaired social communication and restricted or repetitive behaviors. Although current management focuses primarily on behavioral, educational, and pharmacological interventions to alleviate associated symptoms, no universally accepted curative therapy exists. We previously reported substantial improvement and complete remission of the principal autistic features following individualized treatment regimens centered on intramuscular cerebrolysin. We report a child with syndromic atypical autism associated with distinctive craniofacial dysmorphism who demonstrated complete remission of the two principal diagnostic features of autism after prolonged individualized treatment. **Objectives:** This study presents the 21st global case of autism in which the two major diagnostic features (poor social interaction and communication) were cured through an individualized treatment regimen including intramuscular cerebrolysin as the main curative agent. **Patients and Methods:** We have previously reported a 6-year-old boy who presented with global developmental delay and syndromic atypical autism characterized by severe impairment of social interaction, poor responsiveness to his name, reduced eye contact, stereotypic hand flapping, delayed speech and language development, and marked deficits in adaptive functioning. Physical examination revealed multiple craniofacial dysmorphic features, including low-set posteriorly rotated ears, mild hypertelorism, down-slanting palpebral fissures, a broad nasal bridge with a bulbous nasal tip, flattened malar regions, mild retrognathia, a short philtrum, a thin upper lip, and a short neck, suggesting an unrecognized syndromic disorder. The patient was treated with an individualized therapeutic regimen based on our previously published protocol, using intramuscular cerebrolysin as the principal therapeutic agent in combination with selected adjunctive medications. **Results:** Progressive clinical improvement was observed throughout treatment. After the initial month, the patient demonstrated improved eye contact and reduced hyperactivity. The therapeutic regimen was subsequently adjusted according to the clinical response. After 10 months of treatment, the patient exhibited complete remission of the two principal diagnostic features of autism, becoming consistently responsive to his name and establishing normal eye contact. However, significant cognitive impairment and behavioral abnormalities persisted, necessitating continuation of treatment and rehabilitation. During the 11th month, adjunctive omega-3 supplementation and N-acetylcysteine were incorporated based on published evidence. **Conclusion:** This case represents the 21st documented patient in whom complete remission of the principal autistic features was achieved following an individualized cerebrolysin-based therapeutic regimen. The findings add to our previously

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published clinical experience and suggest that this therapeutic approach merits further investigation in well-designed prospective controlled studies. Nevertheless, despite remission of the core autistic features, continued educational, behavioral, and rehabilitative interventions remain essential to address persistent cognitive and adaptive impairments.

Keywords: Craniofacial dysmorphism, cure, new clinical entity, syndromic autism

INTRODUCTION

Autism spectrum disorder (ASD) is a heterogeneous group of chronic neurodevelopmental disorders characterized by early-onset impairments in social communication and interaction, accompanied by restricted and repetitive patterns of behavior, interests, or activities. Diagnosis is primarily clinical and is based on characteristic behavioral manifestations, particularly deficits in social interaction and communication. Cardinal diagnostic features include poor responsiveness to one's name, reduced eye contact, delayed language development, impaired use and understanding of language, and repetitive motor behaviors such as hand flapping, spinning, or stereotyped movements.

The marked variability in speech, language, cognitive development, and adaptive functioning contributes to the clinical heterogeneity of autism. Historically, the mildest form of autism was first described by the Soviet pediatric psychiatrist Grunya Efimovna in 1925, who referred to the condition as *autistic psychopathy* [Figure 1a]. In 1944, Hans Asperger [Figure 1b] described children with similar clinical characteristics, and in 1981 Lorna Wing [Figure 1c] introduced the term *Asperger syndrome* for this phenotype.

The classic form of autism, commonly known as Kanner syndrome, was described by Leo Kanner [Figure 1d] in 1943. Although many affected individuals have normal or above-average intelligence, Kanner syndrome is typically associated with significant delay in speech and language development. During the 1980s, autism became increasingly recognized as part of the broader category of pervasive developmental disorders (PDDs), particularly in the United Kingdom. Asperger syndrome was incorporated into the World Health Organization's International Classification of Diseases, Tenth Revision in 1992 and into the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) in 1994.

Traditionally, autism has been classified into several clinical subtypes. Typical autism generally refers to individuals with autism and below-average intelligence without severe intellectual disability. In contrast, atypical autism includes autism associated with significant intellectual disability as well as regressive autism, in which previously acquired social and communication skills are lost after an initial period of apparently normal development. Because distinguishing among these subtypes is often challenging in clinical practice, the American Psychiatric Association introduced the umbrella term ASD in the fifth edition of the DSM-5 in 2013. This terminology reflects the difficulty in accurately diagnosing the specific types of autism. A worth-mentioning older classification of ASDs, the PDDs, included three main types: Autism, Rett syndrome, and disintegrative childhood disorder, also called Heller syndrome.

Considering this classification and the rare syndromic types of autism, we can gain some understanding of how complex these disorders are. Autism is a clinically and genetically heterogeneous condition that is generally regarded as a functional neurodevelopmental disorder without consistent structural neuroimaging abnormalities. Consequently, routine brain imaging is not universally recommended. Nevertheless, neuroimaging studies have reported abnormalities in selected patients, including arachnoid cysts, agenesis of the corpus callosum, imaging findings suggestive of vasculitis in Heller syndrome, and abnormalities related to coexisting neurological disorders such as cerebral palsy.^[1-18]

Current management of ASDs focuses primarily on improving symptoms and functional outcomes through behavioral interventions, educational support, speech and language therapy, social skills training, and selected pharmacological therapies. Medications including risperidone, baclofen, and ondansetron have been used to reduce behavioral



Figure 1: (a) Grunya Efimovna, a Soviet pediatric psychiatrist. (b) Hans Asperger, an Austrian physician. (c) Lorna Gladys Wing, an English psychiatrist. (d) Leo Kanner, a pioneer of child psychiatry

disturbances and hyperactivity in selected patients.^[1,2,5,7,10,11,15-19] Despite numerous therapeutic approaches, including nutritional supplementation with pyridoxine, magnesium, thiamine, biotin, folic acid, and omega-3 fatty acids, no treatment has been universally accepted as curative. Because the core impairments of autism involve social communication, an effective disease-modifying therapy should ideally improve the principal diagnostic features, particularly responsiveness to one's name, eye contact, and social engagement.

Over the past decade, we have investigated an individualized therapeutic approach using intramuscular cerebrolysin as the principal treatment. Previous publications from our group have described marked improvement and, in selected patients, complete remission of the principal autistic features following individualized treatment regimens that included cerebrolysin.^[11,20-22] Between December 2017 and November 2019, we evaluated 116 children with different forms of autism at the Children Teaching Hospital, Baghdad Medical City. Their ages ranged from 2 to 16 years, and the clinical characteristics and treatment outcomes were reported in several publications and later compiled into a book translated into multiple languages.

Most patients exhibited severe speech delay, with the exception of those diagnosed with Asperger syndrome. Many were non-verbal, whereas others could produce only a limited number of words before treatment. The therapeutic protocol primarily targeted the core manifestations of autism, including impaired social interaction, poor responsiveness to name, limited eye contact, and reduced social engagement. Treatment was also associated with

improvements in speech development and repetitive behaviors.^[11-15] Although long-term follow-up was not possible for every patient, documented cases demonstrated substantial clinical improvement, including complete remission of the principal autistic features in a number of patients.^[11,16,17,20-27]

These encouraging observations have led us to adopt this individualized therapeutic approach in our clinical practice while continuing to document patient outcomes through peer-reviewed publications.^[12-17] Nevertheless, even after marked improvement in the core autistic features, many patients continue to require educational, behavioral, and rehabilitative support to address residual cognitive, learning, and adaptive challenges.

Cerebrolysin is a peptide preparation composed of approximately 85% free amino acids and 15% biologically active low-molecular-weight peptides, including neurotrophic factors that are believed to exert neuroprotective and neurorestorative effects. The drug has been used in a variety of neurological and neurodevelopmental disorders, including intellectual disability, cerebral palsy, brain atrophy, myelomeningocele, spinal muscular atrophy, Charcot–Marie–Tooth disease, kernicterus, and agenesis of the corpus callosum with colpocephaly.^[28-39]

A subset of ASDs is syndromic and is characterized by congenital anomalies or dysmorphic features that suggest an underlying genetic disorder. We previously reported patients with syndromic autism associated with Williams syndrome and Coffin–Siris syndrome from Iraq and other countries [Figures 2 and 3].^[25,40] We also described a 6-year-old boy with autism, cognitive delay, and a distinctive pattern of craniofacial dysmorphism that

did not correspond to any recognized syndrome.^[41] Identification and detailed characterization of such patients may contribute to the recognition of previously unrecognized syndromes and facilitate advances in personalized diagnosis and management.

We have previously reported a 6-year-old boy with autism, cognitive delay, and a distinctive pattern of craniofacial dysmorphism not consistent with any recognized syndrome.^[41] This case represents the 20th global case of autism in which the two major diagnostic features (poor social interaction and communication) were cured through an



Figure 2: Iraqi girl with Coffin–Siris syndrome and autism. She had distinctive facial features including thick eyebrows, a depressed and wide nasal bridge, and a large mouth with thick everted upper and lower lips, hypertrichosis, and sparse scalp hair, particularly in the temporal regions



Figure 3: A boy from India with Williams syndrome and autism. He had a wide mouth with prominent lower lip, puffiness around the eyes, and long upper lip length

individualized treatment regimen including intramuscular cerebrolysin as the main curative agent.

PATIENTS AND METHODS

We previously reported a 6-year-old boy who presented with global developmental delay and autistic behaviors. Developmental milestones were delayed across the cognitive, language, and adaptive domains. The patient exhibited the cardinal clinical features of autism, including impaired social interaction, poor responsiveness to his name, reduced eye contact, and stereotypic behaviors, particularly repetitive hand flapping. During clinical evaluation, he was hyperactive, failed to respond consistently when called by name, and made minimal eye contact [Figure 4].

Developmental Profile

- Speech and language: Marked delay in both expressive and receptive language development
- Motor development: Mild delay in fine motor skills, whereas gross motor development was relatively preserved
- Adaptive functioning: Significantly impaired. The child required assistance with feeding because he was unable to use a spoon effectively and demonstrated delays in age-appropriate activities of daily living.



Figure 4: At the clinic he was not responding to name and had poor eye contact

Family History

The patient was born to non-consanguineous parents. There was no family history of ASD, developmental delay, intellectual disability, or congenital anomalies.

Physical Examination

Weight, height, and head circumference were within the normal range for age. Physical examination revealed multiple craniofacial dysmorphic features [Figure 5], including:

- Low-set, posteriorly rotated ears
- Mild hypertelorism
- Down-slanting palpebral fissures
- Broad nasal bridge with a bulbous nasal tip
- Full cheeks with flattened malar regions
- Mild retrognathia (micrognathia)
- Short, poorly defined philtrum
- Thin upper lip
- Short neck.

No additional systemic abnormalities were identified. Cardiovascular, renal, and musculoskeletal examinations were unremarkable, with no clinically evident congenital anomalies. At the time of reporting, neuro-imaging and metabolic investigations had not been performed.

RESULTS

Based on the presence of significant cognitive impairment in addition to the core autistic features,

the patient was diagnosed with syndromic atypical autism. Treatment was individualized according to our previously published therapeutic protocol for ASDs.^[1,2,5,10,11,15-17,20-22]

Treatment Course

During the 1st month, the patient received intramuscular cerebrolysin (4 mL every other day), oral risperidone (0.5 mg nightly), and oral piracetam (800 mg each morning). Following this initial course, his parents reported noticeable behavioral improvement. Clinical examination demonstrated better eye contact and reduced hyperactivity. In view of this favorable response, cerebrolysin was continued at a maintenance schedule of 4 mL every 3rd day (10 injections monthly) while risperidone and piracetam were maintained.

During the 2nd and 3rd months, cerebrolysin was continued at the maintenance dose. Piracetam was increased to 800 mg twice daily, whereas risperidone was continued unchanged.

Beginning in the 4th month, risperidone was replaced with olanzapine (2.5 mg nightly), and omega-3 supplementation was added. During the 5th month, the olanzapine dose was increased to 5 mg nightly, and oral prochlorperazine (5 mg daily) was introduced.

During the 6th and 7th months, prochlorperazine was replaced with trifluoperazine (1 mg daily), whereas magnesium (250 mg) combined with Vitamin B6 (100 mg) was added. The patient continued to receive cerebrolysin, olanzapine, piracetam, and omega-3 supplementation.



Figure 5: (a) The boy displayed multiple dysmorphic features. (b) The boy displayed multiple dysmorphic features. (c) The boy displayed multiple dysmorphic features

From the 8th through the 10th months, oral baclofen (10 mg nightly) was added to the treatment regimen. The remaining medications were continued without significant modification.

After completing 10 months of treatment (April 25, 2026), the patient exhibited complete remission of the two principal diagnostic features of autism, demonstrating consistent responsiveness to his name and normal eye contact. Nevertheless, significant cognitive impairment and behavioral abnormalities persisted. Consequently, treatment was continued to optimize cognitive and behavioral outcomes.

During the 11th month, N-acetylcysteine (600 mg daily) was added to the treatment regimen, whereas cerebrolysin, olanzapine, baclofen, piracetam, trifluoperazine, and omega-3 supplementation were continued. Omega-3 supplementation was introduced based on the findings of Doaei *et al.*,^[42] whereas N-acetylcysteine was added in accordance with the evidence reported by Lee *et al.*^[43]

DISCUSSION

The patient described in this report represents the 21st documented patient worldwide in whom complete remission of the two principal diagnostic features of autism was achieved following an individualized therapeutic regimen centered on intramuscular cerebrolysin.

Our findings further support our previous observations suggesting that individualized cerebrolysin-based therapy may substantially improve the core manifestations of autism in selected patients.^[11,20-22] In the present case, treatment was associated with progressive improvement in social interaction, culminating in restoration of responsiveness to name and normalization of eye contact after 10 months of therapy. Despite remission of the principal autistic features, substantial cognitive impairment and behavioral abnormalities remained, indicating that continued pharmacological treatment, educational intervention, behavioral therapy, and long-term rehabilitation are essential to maximize functional outcomes.

Cerebrolysin, piracetam, and citicoline have previously demonstrated favorable safety profiles in a variety of pediatric neurological and neuropsychiatric disorders, including developmental

and neurogenetic conditions.^[10,29,32,34-39] The favorable clinical response observed in the present patient further supports continued investigation of this therapeutic strategy in carefully designed prospective studies.

CONCLUSION

This case represents the 21st documented patient in whom complete remission of the principal autistic features was achieved following an individualized cerebrolysin-based therapeutic regimen. The findings add to our previously published clinical experience and suggest that this therapeutic approach merits further investigation in well-designed prospective controlled studies. Nevertheless, despite remission of the core autistic features, continued educational, behavioral, and rehabilitative interventions remain essential to address persistent cognitive and adaptive impairments.

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CONFLICT OF INTEREST

None.

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