MEDICO RESEARCH CHRONICLES
ISSN NO. 2394-3971
DOI No. 10.26838/MEDRECH.2020.7.4.441
Contents available at www.medrech.com
OPEN ACCESS

CEREBRAL PALSY: A UNIQUE ILLUSTRATED EXPERIENCE

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ARTICLE INFO

Article History
Received: July 2020
Accepted: August 2020
Keywords: Cerebral palsy, multi-factorial therapies, illustrative experience.

ABSTRACT

ORIGINAL RESEARCH ARTICLE

Background: Cerebral palsy is a heterogeneous condition associated with a non-progressive lesion causing permanent disorder of movement with limited mobility, and is generally associated with gross motor developmental delay. In moderate to severe cases of cerebral palsy, motor developmental milestones such as walking may never be achieved. Impaired cognition and delayed speech are also commonly seen. The aim of this paper is describe our illustrated experience with cerebral palsy with emphasis on treatment with multi-factorial therapies.
Patients and methods: Seventeen patients with cerebral palsy are described in this paper including two female patients whose early treatment courses were included in previous publications , and 15 new cases (11 males and 4 females) observed during seven months period (May-November,2019). Their ages raged from 10 months to 9 years. Ten patients had significant spasticity limiting their movements. All patients had developmental delay including delayed speech. Nine patients were unable to sit without support, including a patient with significant dystonia, and a patient who could stand and walk with support but was unable to sit without support. Only two patients were able to walk alone, but slowly and with difficulty. Two patients had history of birth asphyxia, and one patient had a genetic condition with 2 of his brothers being affected. The patients were treated based on our published experiences with individualized treatment plans providing a combination of various interventions including nutritional support, muscle relaxants, oral pyritinol, intramuscular piracetam, citicoline (oral and injectable), intramuscular cerebrolysin, and intramuscular nandrolone decanoate. The aims of these therapies include overcoming spasticity, repairing the brain and improving its function, and ultimately improving mobility and advancing development.
Results: All patients experienced improvement in motor development without the occurrence of any side effect. However, it was not possible

to document the details of treatments and follow-up for all patients, but it was possible to provide an illustrated demonstration of improvement in seven patients.
Conclusion: Cerebral palsy is a heterogeneous condition, and the emergence of a single therapeutic agent that offers a comprehensive effect to improve its manifestations is very unlikely is the near future. Therefore, the use of evidence-based multi-factorial therapies is advisable. Adequate muscle relaxation is vital to prevent the complications of contractures which appear to cause a progressive disability.

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INTRODUCTION

Cerebral palsy is a condition resulting from abnormal development or damage mostly to parts of the brain that control movement, balance, and posture. A minority of cases of cerebral palsy, about 2% could be attributed to an inherited genetic cause, and most inherited cases are expected to be autosomal recessive. The brain abnormalities in cerebral palsy cause a non-progressive, but a permanent disorder of movement, posture, and limitation of mobility. In addition to movement problems, patients with cerebral palsy may have cognitive impairment leading to difficulties with learning, and speech. The spastic cerebral palsy may account for up to 70% of all cases. In this type, mobility impairment can be worsened by hypertonia caused by an upper motor neuron lesion in the brain and the corticospinal tract or the motor cortex. Although the neurologic lesion in spastic cerebral palsy is non-progressive, secondary orthopedic complications are generally progressive and disabling because of the developments of joint deformities and joint contractures [1, 2, 3, 4].

The movement disorder is generally associated with gross motor developmental delay, and in moderate to severe cases, motor developmental milestones such as walking may never be achieved without appropriate treatments (Figure-1). Figure-2 shows a boy with spastic cerebral palsy who was treated with multi-factorial therapy early during the second year and was able to stand with support at about the age of three years and he was mostly considered a normal child at about the age of five years. Unfortunately, details of his treatment and follow-ups are not available.
In less severe cases, the patient can walk, but experience gait difficulties mostly in the form of tip-toeing gait [1, 2, 3, 4].

There are generally no universally agreed on specific therapies for cerebral palsy. Treatment of spastic cerebral palsy is essentially aiming at improving mobility through muscle relaxation and physiotherapy. Muscle relaxants are used to improve spasticity and prevent deformities and contractures. However, muscle relaxants have not been reported to have an important effect on motor development. Many patients with moderate and severe spastic cerebral palsy develop flexion deformities especially equinus or planter deformity of the ankles [5, 6, 7].

This paper aims to describe our illustrated experience with cerebral palsy with emphasis on treatment with multi-factorial therapies.

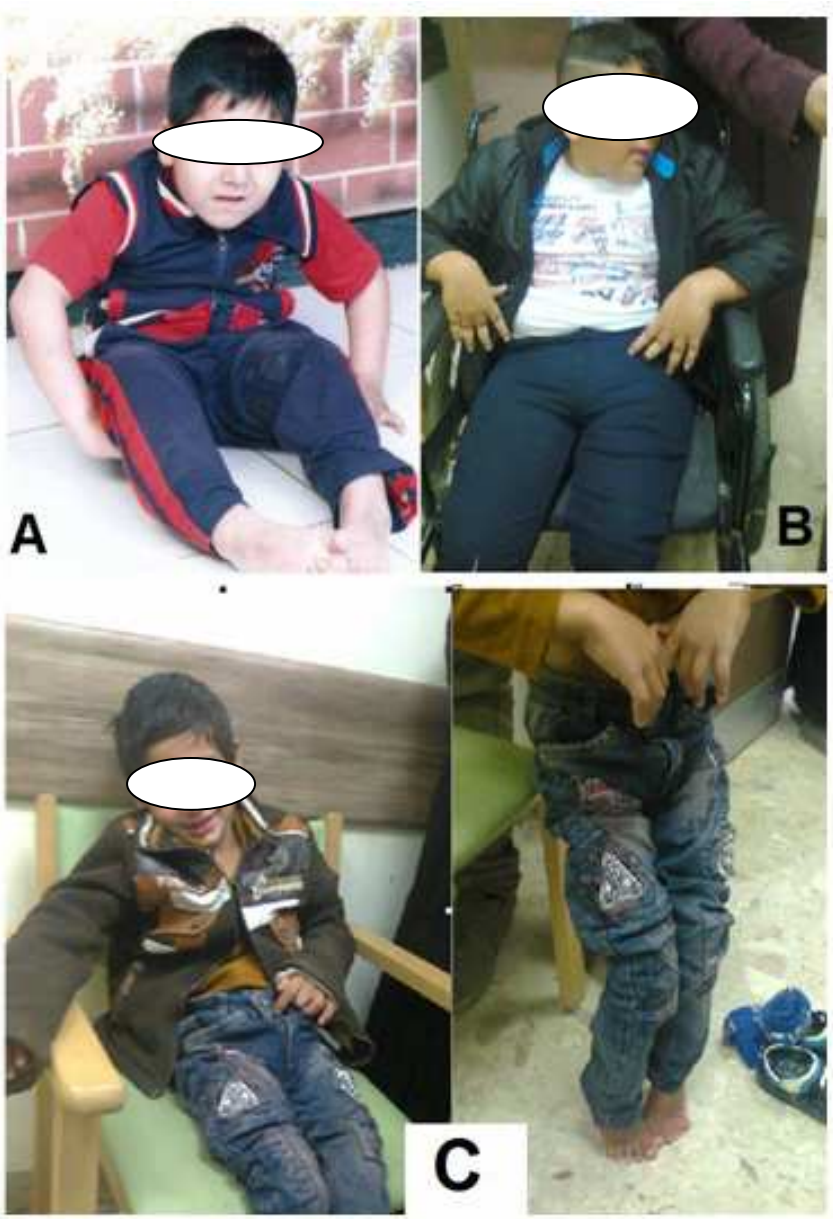

Figure-1: Patients with spastic cerebral palsy with the development of flexion deformities: (A) A boy who remained unable to stand or walk at the age of six years (B) A twelve-year-old boy who had normal speech development, but he was unable to stand or walk even when holding furniture and was using a wheelchair. (C) A six-year boy who was unable to stand or walk even when holding furniture

Figure-2: A boy with spastic cerebral palsy who was treated with multi-factorial therapy early during the second year of life for more than one year, and was able to stand with support at about the age of three years. He was mostly considered a normal child at about the age of five years. Unfortunately, details of his treatment and follow-ups are not available.

PATIENTS AND METHODS

Seventeen patients with cerebral palsy are described in this paper including two female patients whose early treatment courses were included in previous publications [4, 5], and 15 new cases (11 males and 4 females) observed during seven months' period (May-November, 2019). Their ages ranged from 10 months to 9 years.

Ten patients had significant spasticity limiting their movements. All patients had developmental delays including delayed speech. Nine patients were unable to sit without support (Figure-3 and Figure-4), including a patient with significant dystonia (Figure-5), and a patient who could stand and walk with support but was unable to sit without support (Figure-6). Two patients had tendon lengthening surgery at the hip (Patient-10), and at ankles (Patient-11), and both were able to stand with support, but not able to walk even with support (Figures 7 and 8). Patient-12 (Figure-9) could stand with the help of parents using a foot-supporting device and step one or 2 steps very slowly, while patient-13 (Figure-10) at the clinic refused to stand or walk, but at home, she was able to walk of a walking aid. Only two patients (Patients 14 and 15) were able to walk alone, but slowly and with difficulty. Two patients had a history of birth asphyxia (Patient-1 in figure-3A, and patient-14 in figure-11A), and one patient had a genetic condition with 2 of his brothers being affected (Patient-15 in figure-11B). Brain imaging studies were available for ten patients; Table-1 summarizes the brain imaging studies of the patients.

The patients were treated based on our published experiences [5,6,7] with individualized treatment plans providing a

combination of various interventions including nutritional support, muscle relaxants, oral pyritinol, intramuscular piracetam, citicoline (oral and injectable), intramuscular cerebrolysin, and intramuscular nandrolone decanoate. The aims of these therapies include overcoming spasticity, repairing the brain and improving its function, and ultimately improving mobility and advancing development.

However, it was not possible to keep records of the treatment details and follow-up of all patients, but it was possible to provide an illustrated demonstration of improvement in seven patients. Table-2 the treatment of 9 patients

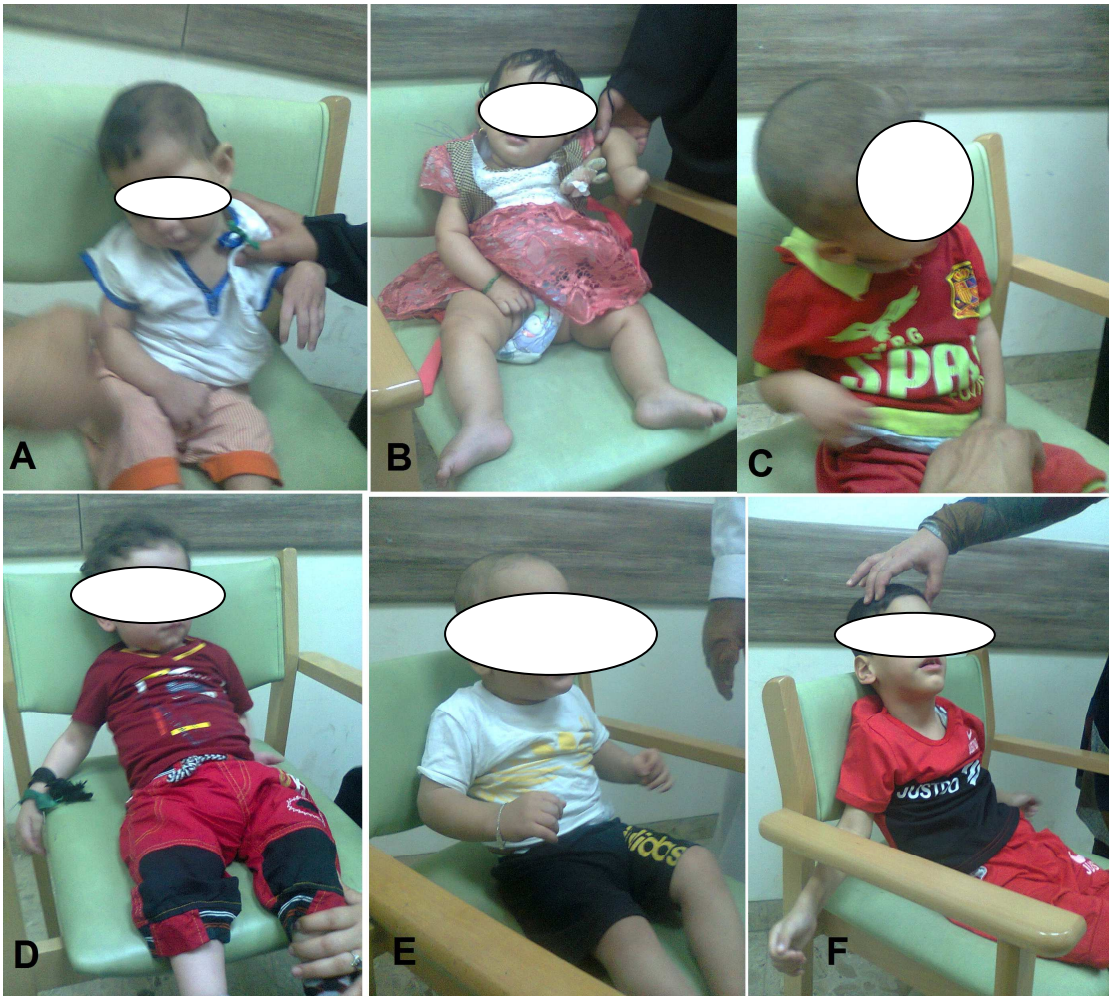

Figure-3: Six of the nine patients who were unable to sit without support (A: Patient-1, B: Patient-2, C: Patient-3, D: Patient-4, E: Patient-5, F: Patient-6. They were not saying any word. Patient-1 had poor spontaneous movements and poor head control and was not stable when seated on a hair. He also had poor feeding. Patients 2, 3, and 6 also had poor head control. Patient-3 was babbling without saying any, but he could feed himself with bottle with the assistance of the mother.

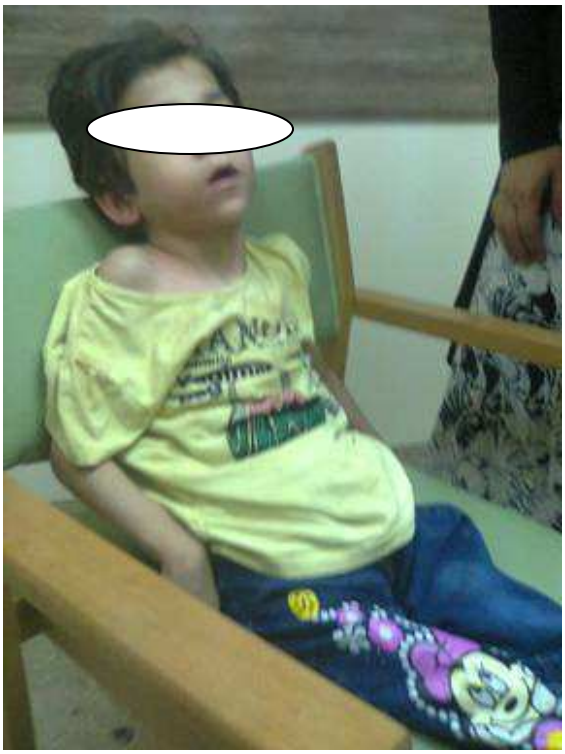

Figure-4: One of the nine patients who were unable to sit without support (Patient-7). She was emaciated and had poor alertness with very poor spontaneous movements and was saying any word

Figure-5: A patient with significant dystonia who was unable to sit without support (Patient-8), and he was not saying any word

Figure-6: A patient who could stand and walk very slowly with support but was unable to sit without support (Patient-9)

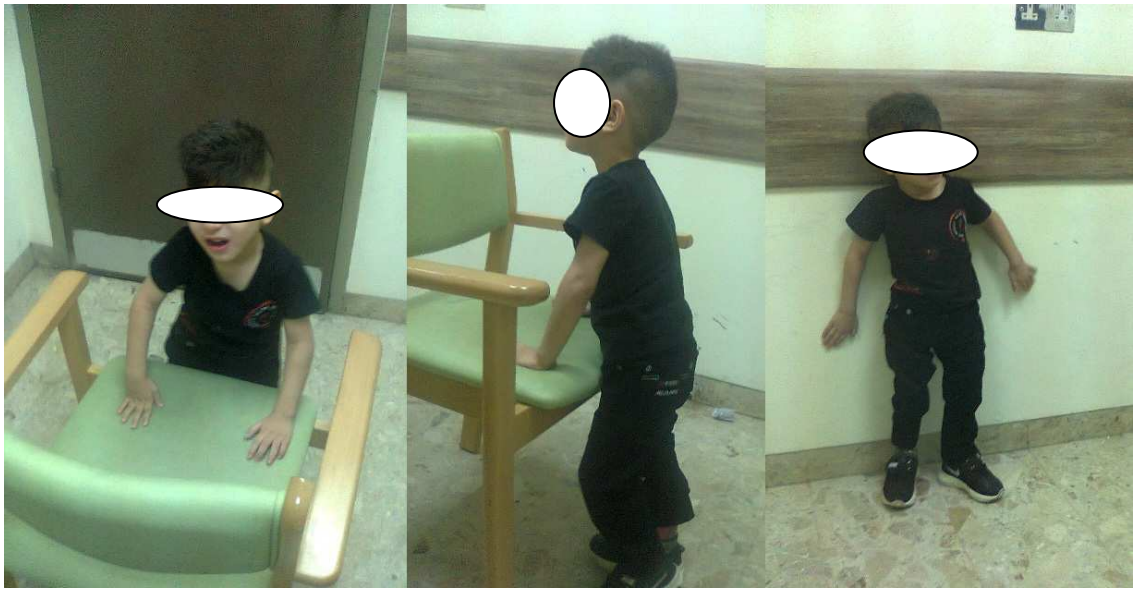

Figure-7: Patient-10 had tendon lengthening surgery at the hip and was able to stand with support but not able to walk even with support

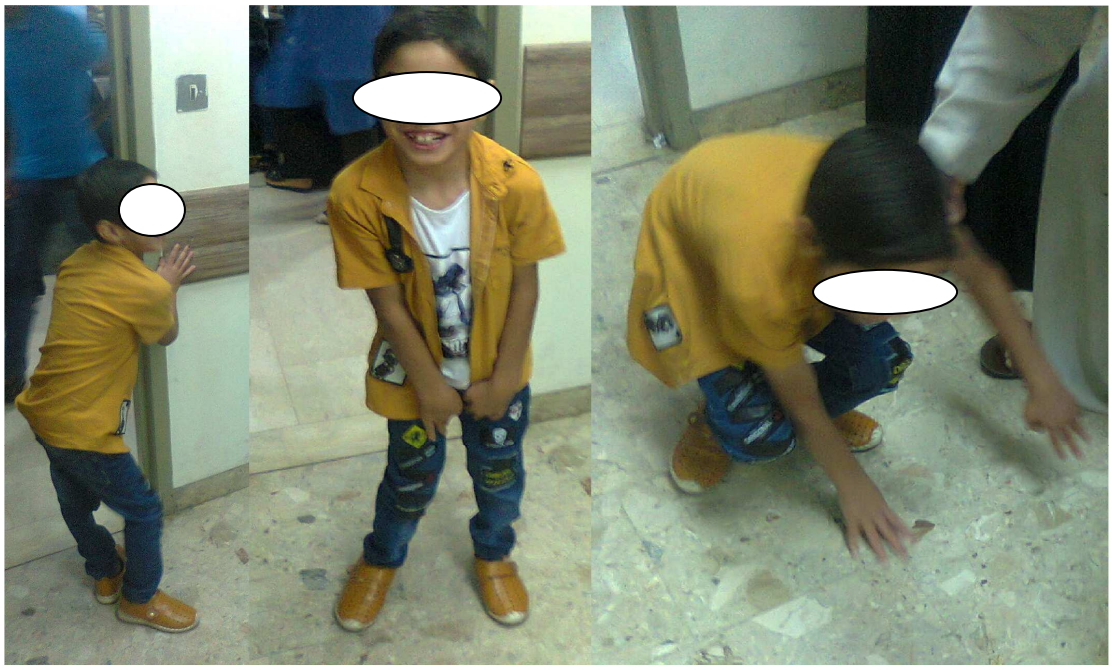

Figure-8: Patient-11 had tendon lengthening surgery at the ankle, and was able to stand with support but not able to walk even with support and fell when tried to make a step. At the clinic, the boy had poor speech development, but was cooperative and smiling. He had some wasting and mild spasticity of all limbs. He tried to copy a circle, but he couldn't

Figure-9: Patient-12 was unable to stand or walk alone, but could stand with the help of parents using foot supporting device and step one or 2 steps very slowly. His speech was acceptable to his parent, but he didn't say a single word at the clinic. He could drink from a cup, but he could not use a spoon to eat. Parents said he could scribble. He was spastic, and he was receiving low dose of Baclofen 5 mg three times daily

Figure-10: Patient-13. at the clinic, she refused to stand or walk, but at home she was able to walk of a walking aid

Figure-11: Only two patients (A: Patients-14 and B: Patients-15) were able to walk alone, but slowly and with difficulty. Both patients had poor speech development and cognitive impairment, and they were not understanding simple questions. Patient-15, at the age of nine, he was saying very few words and was not understanding spoken language including greeting by the doctor. When he asked stand on one foot and to take a pen a copy a line, he couldn't understand

Table-1: The brain imaging studies of the patients				
	Sex	**Age**	**Characteristics**	**Brain imaging**
1	M	10 mo	Spastic	Ultrasound: Bilateral dilatation of the lateral ventricles, 14mm, and mild dilatation of the third ventricles.
2	F	1 yr	No significant spasticity, brisk reflexes	Ultrasound: Normal
3	M	18 mo	No significant spasticity	MRI: Evidence of mild brain atrophy.
4	M	2 yr	Spastic	N/A
5	M	2 yr	Spastic	Ultrasound was normal, but MRI showed evidence of brain atrophy.
6	M	5 yr	No significant spasticity	Evidence of brain atrophy
7	F	6 yr & 2 mo	Spastic	MRI: Bilateral enlargement of the subarachnoid space in the frontal, temporal, & anterior parietal regions, mildly dilated ventricles.
8	M	3yr &4 mo	Spastic & dystonic	N/A
9	M	3yr	Spastic	MRI: Evidence of moderate atrophic changes & bilateral parietal lobes deep white matter periventricular leukomalacia & mildly dilated lateral ventricles.
10	M	5 yr	Spastic	N/A
11	M		Spastic	CT-scan: Evidence of atrophic changes in the temporal region.
12	M	3 yr & 9 mo	Spastic	N/A
13	F	4yr	Spastic	CT-scan: Evidence of mild diffuse atrophic changes
14	M	6yr & 6 mo	No significant spasticity	N/A
15	M	9 yr	No significant spasticity	CT: Evidence of brain atrophy indicated by mild dilatation of the 3rd & lateral ventricles

Table-2: Treatment courses
Patient-1(Figure-3A)
First course [Started on the 9th of May-2019]
Oral baclofen 5 mg three times daily. Oral citicoline 2 ml daily in the morning. Intramuscular piracetam 1ml (200mg) every other day received 10 doses over 20 days.
Second course of treatment [Started on the 13th of June, 2019]
Oral baclofen 5 mg three times daily. Oral citicoline 2 ml daily in the morning. Intramuscular cerebrolysin 1ml every other day received 10 doses over 20 days. Oral royal jelly once daily.
Patient-3 (Figure-3C)
Intramuscular cerebrolysin 1ml daily. Oral citicoline 2 ml (200 mg) daily in the morning. Royal jelly twice daily.
Patient-4 (Figure-3D)
First course
Oral baclofen 10 mg twice daily. Intramuscular cerebrolysin 1ml in the morning daily for 20 days. Oral citicoline 3ml (300 mg) daily in the morning.
Second course of treatment
Oral baclofen 10 mg twice daily. Intramuscular citicoline 3ml (375 mg) in the morning every third day (10 doses over 30 days).
Patient-6 (Figure-3E)
Intramuscular cerebrolysin 5ml every third day (10 doses over 30 days). Oral citicoline 3 ml (300 mg) daily in the morning. Royal jelly twice daily.
Patient-7 (Figure-4)
Oral baclofen 5 mg three times daily. Oral citicoline ml (200 mg) daily in the morning. Royal jelly three times daily.
Patient-8 (Figure-3F)
Oral baclofen 10 mg three times daily. Intramuscular piracetam 3 ml (600mg) on alternate days (15 doses) Intramuscular citicoline 3 ml (275 mg) on alternate days (15 doses)
Patient-11 (Figure-8) [Treatment started on the 4th of July, 2019]
Oral baclofen 2.5 mg three times a day, increased within 2 weeks to 10 mg twice daily. Intramuscular cerebrolysin 5ml every other day in the morning (10 doses). Amino acid supplementation. Single Intramuscular injection of Nandrolone decanoate 25 mg.

Table-2: Treatment courses (Cont.)

Patient-12 (Figure-9)
Oral baclofen 5 mg increased to 10 mg twice daily. Single intramuscular injection of nandrolone decanoate 25 mg was given with providing nutritional support in the form of royal jelly. Oral citicoline 2 ml (250 mg) in the morning. The parents were advised to abandon the foot supporting device which was used as a walking aid and try to train him to stand and walk alone with the initiation of treatment.
Patient-15 (Figure-11B)
First course of treatment [started on the 2nd of May]
Intramuscular cerebrolysin 3ml every other day received 10 doses over 20 days. Oral citicoline 3 ml daily in the morning. Royal jelly twice daily.
Second course of treatment [Started on the 26th of May,2019]
Intramuscular cerebrolysin 5ml every third day (10 doses to be given over 30 days). Oral citicoline 3 ml daily in the morning. Royal jelly twice daily.

Patients 16 and 17 were two female patients whose early treatment courses were included in a previous publication [4, 5].

Patient-16 was a girl with severe spastic cerebral who was seen at about the age of two years with markedly delayed motor development. She was unable to sit unsupported and was not crawling She was treated with muscle relaxants including oral baclofen and intermittent use of oral diazepam 2mg at night. Oral baclofen was gradually increased to 30 mg daily. Also, she received ten courses of multi-factorial therapies (Table-3). Nutritional support was mainly provided in the form of oral royal jelly capsules and she also received amino acid supplementation for several weeks.

Patient-17 was a three-year-old girl with spastic cerebral palsy who was unable to neither stand nor walk and had poor fine motor skills. She was not saying any word. She was unable to sit unsupported and was not crawling She was treated with muscle relaxants including oral baclofen and intermittent use of oral diazepam 2mg at night. She received four courses of treatment (Table-4). Nutritional support was mainly provided in the form of oral royal jelly capsules and she also received amino acid supplementation for several weeks.

Table-3: Course of treatment received by patient-16
First course [Started during March 2018]
Oral baclofen 5mg three times daily. Piracetam 2ml (400mg) was given intramuscularly every three days (4 doses). Citicoline 2ml (250mg) given intramuscularly every three days (4 doses).
Second course [Started on the 29th of March, 2018]
Oral baclofen 5mg three times daily. Oral pyritinol 3ml (60mg) daily for one month in the morning. Oral Citicoline 2ml (200mg) daily for one month in the afternoon. Nandrolone decanoate 12.5mg intramusular injetion.

Third course [Started on the 30th of April, 2018]
Oral baclofen 10 mg twice times daily. Oral pyritinol 3ml (60mg) daily in the morning for one month. Nandrolone decanoate 12.5mg intramusular injetion. Oral Citicoline 2ml (200 mg) daily for one month given in the afternoon. Oral royal jelly capsules once daily.
Fourth course [Started on the 16th of August, 2018]
Oral baclofen 10 mg twice times daily. Piracetam 3ml (600mg) given intra-muscularly every three days (10 doses). Oral Citicoline 3 ml (200 mg) daily for one month given in the morning. Oral royal jelly capsules three times daily.
Fifth course [Started on the 27th of September, 2018]
Oral baclofen 10 mg in the morning, 5mg in the afternoon, 10 mg at night. Piracetam 3ml (600mg) given intra-muscularly every three days (10 doses). Intramuscular citicoline 3 ml (375 mg) every three days (10 doses). Oral royal jelly capsules three times daily.
Sixth course [Started on the 27th of December, 2018]
Oral baclofen 10 mg three times daily. Oral diazepam 2mg at night for 20 days. Cerebrolysin 1ml every third day, 10 dose were given over one month. Nandrolone decanoate 12.5mg intramusular injetion.
Seventh course [Early during the year 2019]
Oral baclofen 10 mg three times daily. Cerebrolysin 3ml every third day, 10 given over one month. Oral Citicoline 3ml (300mg) daily for one month given in the morning.
Eighth course [Started on the 4th of April, 2019]
Oral baclofen 10 mg three times daily. Oral diazepam 2mg at night for 20 days. Nandrolone decanoate 12.5mg intramusular injetion. Oral royal jelly capsules twice times daily.
Ninth course [Started on the 2nd of May, 2019]
Oral baclofen 10 mg three times daily. Oral diazepam 2mg at night for 20 days. Oral royal jelly capsules twice times daily.
Tenth course [Started on the 8th of August, 2019]
Oral baclofen 10 mg three times daily. Oral diazepam 2mg at night for 20 days. Nandrolone decanoate 25 mg intramusular injetion. Oral royal jelly capsules three times daily.

Table-4: Course of treatment received by patient-17
First course

Oral baclofen 5mg twice daily. Oral pyritinol 3ml (60 mg) in the morning daily for one month. Citicoline 2ml (250) was given intramuscularly every three days (10 doses).
Second course
Oral baclofen 5mg twice daily. Oral pyritinol 3ml (60 mg in the morning daily for one month. Nandrolone decanoate 12.5mg intra-muscularly. Oral royal jelly capsules three times daily.
Third course [Started o the 29th of April, 2018]
Oral baclofen 5mg three times daily. Oral pyritinol 3ml (60 mg) in the morning daily for one month. Cerebrolysin 1ml every other day was given by intramuscular injection (10 doses).
Four-course
Oral baclofen 10 mg twice daily. Nandrolone decanoate 12.5mg intra-muscularly. Amino acid supplementation
Fifth course [Started 9th of July 2018]
Oral baclofen 10 mg three daily. Piracetam 2.5 ml (500mg) given intramuscularly every three days (6 doses). Oral royal jelly capsules twice daily.
Sixth course [Started 29th of July 2018]
Oral baclofen 10 mg three daily. Oral diazepam 2mg at night for 10 days. Oral royal jelly capsules twice daily. Nandrolone decanoate 25 mg intra-muscularly.
Seventh course [Started 15th of August 2018]
Oral baclofen 10 mg three daily. Oral royal jelly capsules three times daily. Nandrolone decanoate 25 mg intra-muscularly.
Eighth course [Started 17th of August 2018]
Oral baclofen 10 mg three daily. Oral diazepam 2mg at night for 10 days. Citicoline 2ml (250) was given intramuscularly every three days (10 doses). Oral royal jelly capsules three times daily.

RESULTS

All patients experienced an improvement in motor development without the occurrence of any side effects. However, it was not possible to document the details of treatments and follow-up for all patients, but it was possible to provide an illustrated demonstration of improvement in seven patients.

After initial treatment, patient-1 experienced lessening of spasticity which is attributed to baclofen, and he had improved feeding and head control which is attributed to improved brain function associated with the use of citicoline piracetam, and he was no longer falling when seated on the chair (Figure-12).

After the initial treatment, patient-7 experienced a lessening of spasticity and had

markedly improved alertness, much more spontaneous movements, and improved feeding with significantly improved nutritional status (Figure-13).

After the initial treatment, Patient-11 was able to stand supporting himself o the wall (Figure-14) and could walk supporting himself on the wall rather rapidly and showed improved fine motor skills when tried to copy a circle and a square. The family also reported improved in his speech. He continued on oral baclofen and received another dose of intramuscular injection of nandrolone decanoate 50 mg.

After one week of treatment, the parents of patient-12 reported that after the nandrolone decanoate injection, for few days he showed an obvious increase in strength and was able to stand and walked alone for three days, but thereafter his strength lessened but was still able to stand by himself but with supporting himself to the wall and walk (Figure-15). His fine motor skills also improved and he was also able to feed himself with a spoon but with some spilling and was able to copy a poor circle and a poor square (Figure-15). Baclofen was increased and 5 mg was given during the midday, oral citicoline and royal jelly capsule continued. Ten doses of intramuscular piracetam 400 mg was prescribed to be given every other day.

After the first course, patient-15 he had less difficulty with walking, and markedly improved cognition and understanding:

A-When he asked to stand on one foot, tried but for a short time while holding furniture. B-When he asked to take a pen a copy a line, he tried but he couldn't (Figure-16). Improved speech and saying more words. Improved social interaction by replying to the doctors greeting and replying to goodbye.

After six treatment courses, patient-16 was able to sit and stand with support (Figure-17A). Treatment was also associated with improved speech. After the tenth treatment course, she experienced an improvement in her ability to stand and walk with support (Figure-17B).

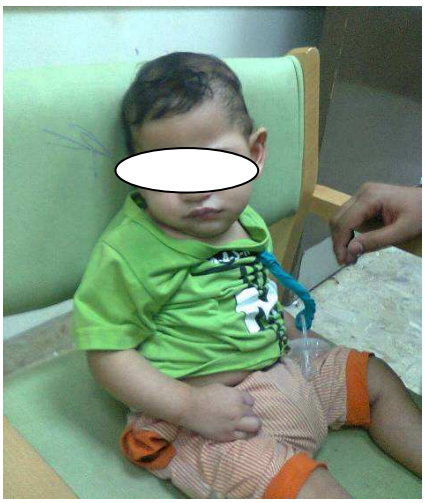

Figure-12: After treatment, patient-1 had better head control was no longer falling when seated on the chair

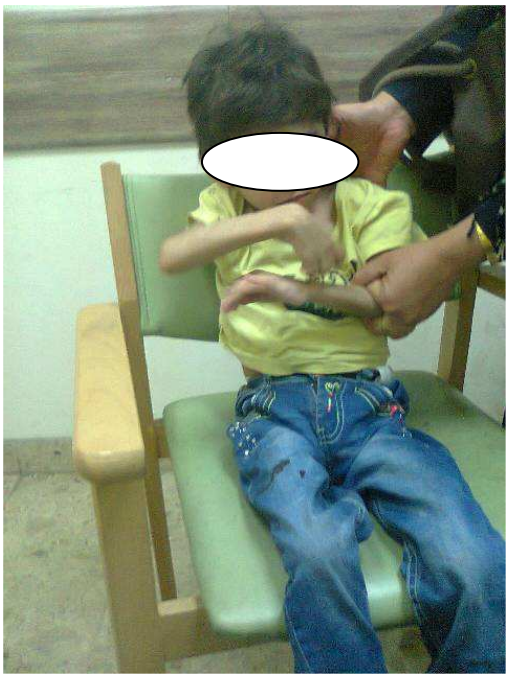

Figure-13: After the initial treatment, patient-7 had markedly improved alertness, much more spontaneous movements and improved feeding with significantly improved nutritional status

Figure-14: After the initial treatment, Patient-11 was able to stand supporting himself o the wall

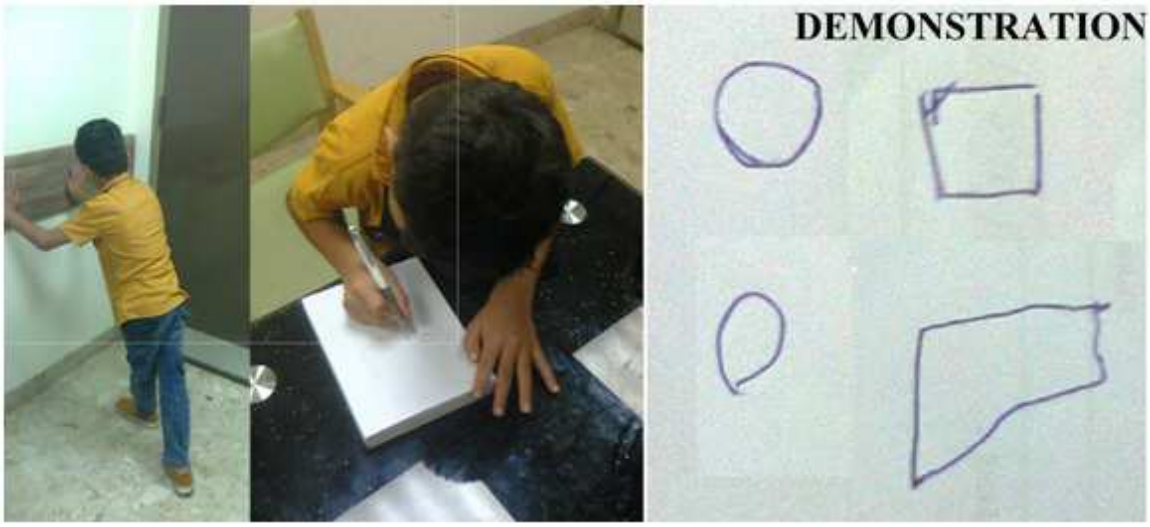

Figure-15: After one week of treatment, patient-12 was able to stand by himself but with supporting himself to the wall and walk. He also had improved fine motor skills and was able to copy a poor circle and a poor square

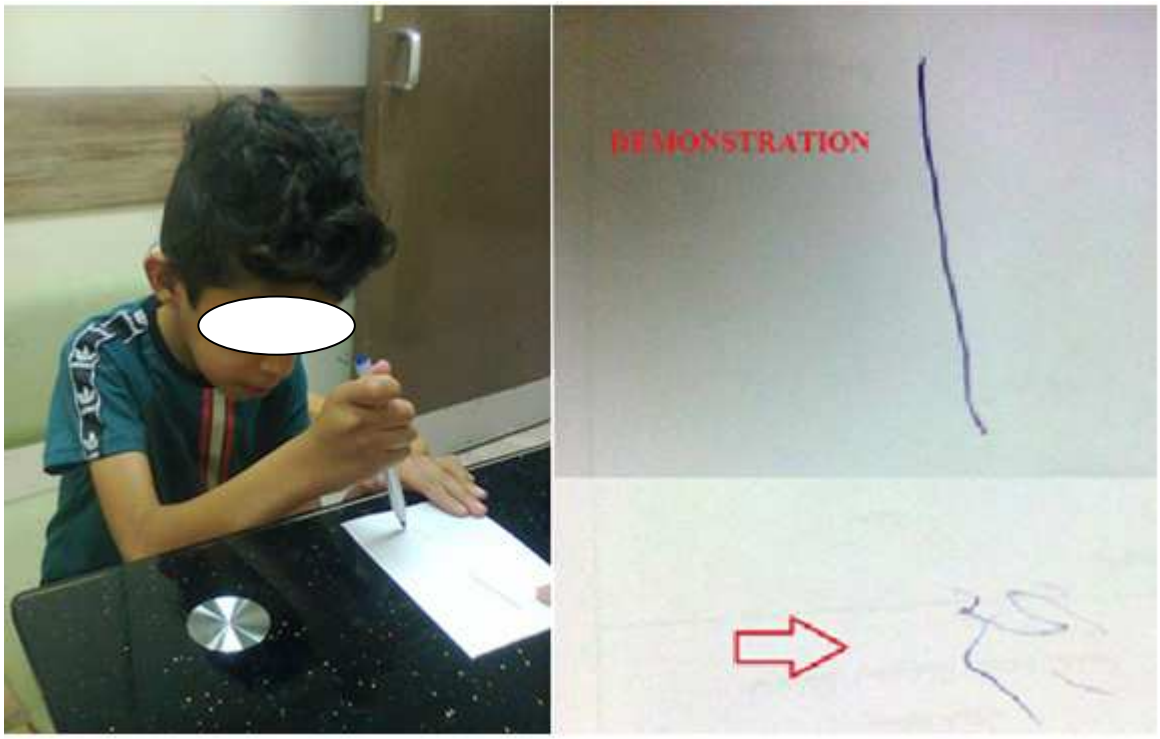

Figure-16: After the first course of treatment, patient 15 experienced obvious cognitive improvements, and when he was asked to take a pen a copy a line, he tried but he couldn't

Figure-17A: After the sixth treatment course, patient-16, she was able to sit and stand with support, but with difficulty

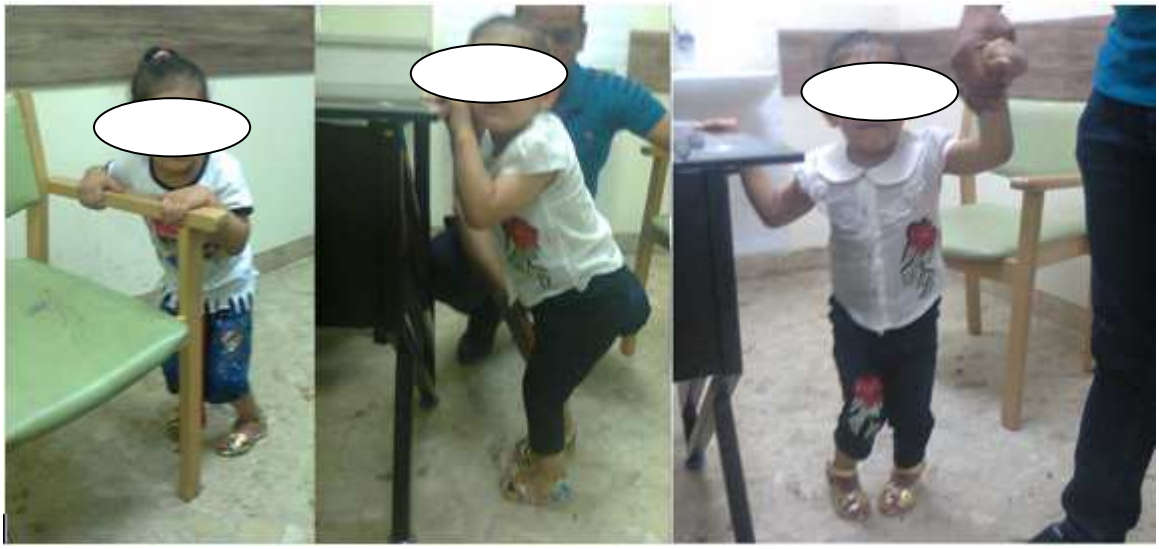

Figure-17 B: After the tenth treatment course, patient-16, experienced improvement in her ability to stand and walk with support

After the 4th course of treatment (Figure-18A), patient-17 was able to stand and walk holding furniture. She showed improved fine motor skills, and was able to hold a pen to try to copy a circle, and she could copy a circle, but the circle was not very good Treatment was also associated with initiation of speech development. After the 8th course of treatment, she was able to walk holding furniture confidently and in good speed. She also showed improved ability to copy a circle (Figure-18B).

Discussion

Citicoline is a mononucleotide made of ribose, pyrophosphate, cytosine and choline. It is a water-soluble naturally occurring substance that is generally grouped with the B vitamins. It is also considered a form of the essential nutrient choline [8, 9].

Citicoline has been recently used with benefits in the treatment of childhood neuropsychiatric disorders including, pervasive developmental disorders (including Rett syndrome) [10-17], brain atrophy [3, 18], kernicterus [19], and cerebral palsy [4, 5, 6].

Piracetam can beneficially influence impaired brain function by improving neuronal and cognitive functions without acting as a sedative or stimulant, increasing blood flow and oxygen consumption in the brain, and improving the function of the neurotransmitters and brain neurotransmission.

The modes of action of piracetam have been attributed to differential effects on subtypes of glutamate receptors without GABAergic actions Piracetam has no significant side effect nor has acute toxicity at the doses used in human studies. The LD_{50} is 5.6 g/kg in rats and 20 g/kg in mice, indicating extremely low acute toxicity [3, 4, 20].

Figure-18A: Patient-17, after the 4th course of treatment, she was able to stand and walk holding furniture slowly and with some difficulty. She also showed improved fine motor skills and was able to hold a pen to try to copy a circle, but the circle was not very good

Figure-18B: Patient-17, after the 8th course of treatment, she was able to walk holding furniture confidently and at a good speed. She also showed improved ability to copy a circle

Cerebrolysin has recently been safely used in the treatment of a variety of childhood neurological and psychiatric disorders including brain atrophy [3,18], cerebral palsy [4,5,6], kernicterus [19], agenesis of the corpus callosum [21], pediatric juvenile spinal muscular atrophy [22,23], Charcot Marie Tooth disease [24,25], myelomeningocele [26], autism, Rett syndrome [10-17], and mental retardation [27,28].

Cerebrolysin is a mixture of free amino acids (85%) and 15% biologically active low molecular weight amino acid sequences which include low molecular weight neuro-peptides (Brain-derived neurotrophic factor, glial cell

line-derived neurotrophic factor, nerve growth factor, ciliary neurotrophic factor [29].

Pyritinol, a pyrithioxine derivative, is a semi-synthetic water-soluble analog of vitamin B_6 (Pyridoxine Hcl).

It has been shown that cerebral blood supply is increased by pyritinol resulting in an improvement of nerve cell metabolism, and it was used with benefit in cerebral palsy [3, 27, 30].

Nandrolone decanoate has recently been used with benefit in the treatment of patients with cerebral palsy [30], brain atrophy [3, 18], refractory vitamin D-resistant rickets [31], and achondroplasia [32].

In contrast to 17- testosterone derivatives, nandrolone esters do not cause sodium sulfobromophthalein retention; therefore, hepatic complications are infrequent with their use in ordinary doses for short periods. The use of nandrolone has been reported to be associated with beneficial positive effects such as muscle strengthening [3, 18, 31, 32].

CONCLUSION

Cerebral palsy is a heterogeneous condition, and the emergence of a single therapeutic agent that offers a comprehensive effect to improve its manifestations is very unlikely in the near future. Therefore, the use of evidence-based multi-factorial therapies is advisable. Adequate muscle relaxation is vital to prevent the complications of contractures which appear to cause a progressive disability.

ACKNOWLEDGEMENT

The author would like to acknowledge his gratitude for the parents who willingly accepted publishing the photos of their children.

Conflict of interests: None.

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