

Review Article

The Pattern of Cerebral Palsy in Iraqi Children

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Abstract

Background: Cerebral palsy is a heterogeneous condition associated with a non-progressive lesion causing permanent disorder of movement with limited mobility. It 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. Little is known about the pattern of cerebral palsy in Iraq. The aim of this paper is to describe the pattern of cerebral palsy in a sample of Iraqi children observed by a single pediatrician in a tertiary pediatric referral center.

Patients and methods: During nine months period (August, 2018 to April, 2019), thirty four patients with cerebral palsy (19 males and 15 females) were observed at the neuropsychiatry clinic at the Children Teaching hospital of Baghdad Medical City. Their ages ranged from four months to twelve years. The patients were treated with individualized treatment plans providing a combination of various interventions including nutritional support, muscle relaxants, oral pyridinol, intramuscular piracetam, citicoline (oral and injectable), intramuscular cerebrolisin, and intramuscular nandrolone decanoate.

Aim: Little is known about the pattern of cerebral palsy in Iraq. The aim of this paper is to describe the pattern of cerebral palsy in a sample of Iraqi children.

Results: Twenty three patients had spastic cerebral palsy affecting all limbs (spastic quadriplegia) including twelve boys and eleven girls. Four patients including three boys and one girl didn't have significant spasticity. Four patients had hemiplegic spastic cerebral palsy including one boy and three girls. Three boys had ataxic cerebral palsy. Three male patients had a definite history of birth asphyxia including two patients with spastic cerebral palsy and one patient with ataxic cerebral palsy. Two female patients had history of meningitis before the diagnosis of cerebral palsy. One patient had spastic cerebral palsy and one patient aged ten months didn't have significant spasticity, but the possibility of later development of spasticity couldn't be excluded. One female had affected brother with spastic cerebral palsy and imaging study showing findings similar to the findings of leukodystrophy.

Eye abnormalities were present in six patients: Two patients (a boy and a girl) had refractive error, two patients (a boy and a girl) had squint, and in the girl it resulted in torticollis, and two patients (a boy and a girl) had significant reduction of visual acuity most probably resulting from optic atrophy, and the male patient was almost totally blind. Four patients had history of epileptic seizures including three male patients, and one girl, and in two boys seizures required long term anticonvulsant therapies. A boy with spastic cerebral palsy had a pelvic MRI showing atrophic undescended right testis located in the mid-inguinal region, and a girl with spastic cerebral palsy had secundum atrial septal defect.

Nineteen patients had brain imaging studies including ultrasonography, CT-scan and MRI. Three of these nineteen patients had normal findings. Nine patients had evidence of diffuse brain atrophy on brain imaging studies. Two of these nine patients had additional unique findings including one patient had polymicrogyri and dilatation of the basal cisterns, and a second patient had bilateral parietal lobe white matter peri-ventricular leukomalacia.

Seven patients had a unique findings other than evidence of diffuses brain atrophy: One patient had left hemi-brain atrophy, one patient had vermian hypoplasia with retro-cerebellar hypoplasia, one patient had absence of the inferior vermis and cerebellum atrophy, one patient had diffuse hypo density of both cerebral hemispheres suggesting hypoxic brain ischemia caused by birth asphyxia, one patient had evidence of hypoxic ischemic encephalopathy, one patient had large right sided temporoparietal gliotic changes with large communicating porencephaly cyst, and one patient had mild dilatation of the right ventricle.

Conclusion: Spastic cerebral palsy affecting all limbs (Spastic quadriplegia) accounted for about 67% of all Iraqi children with cerebral palsy. Radiologic evidence of diffuse brain atrophy was present in more than 25% of the patients with cerebral palsy in this series.

Keywords: Cerebral palsy; Pattern; Brain atrophy

Introduction

Cerebral palsy is a condition results from abnormal development or damage to the regions 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 permanent disorder of movement, posture, and limitation of mobility. The movement disorder generally leads to gross motor developmental delay and in moderate to severe cases motor developmental milestones such as walking May never be achieved [1-4]. In addition to movement problems, patients with cerebral palsy may have cognitive impairment leading to difficulties with learning, and speech.

The spastic type of cerebral palsy is by far the most common type accounting for about 70% of all cases. In this type, mobility impairment is 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. In less severe cases, the patient can walk, but experience gait difficulties mostly in the form of tip-toeing gait.

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There are generally no universally agreed 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 ankle [1-3].

Patients and Methods

During nine months period (August, 2018 to April, 2019), thirty four patients with cerebral palsy (19 males and 15 females) were observed at the neuropsychiatry clinic at the Children Teaching hospital of Baghdad Medical City. Their ages ranged from four months to twelve years.

The patients were treated 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 protocol for this research was approved by the scientific committee of Iraq head quarter of Copernicus Scientists International Panel and conforms to the provisions laid out in the Declaration of Helsinki (as revised in Edinburgh 2000). Consent has been obtained from the parents of the patients to publish their photographs in the article.

Results

Twenty three patients had spastic cerebral palsy affecting all limbs (Spastic quadriplegia) including twelve boys and eleven girls. Four patients including three boys and one girl didn't have significant spasticity. Four patients had hemiplegic spastic cerebral palsy including one boy and three girls. Three boys had ataxic cerebral palsy.

Three male patients had a definite history of birth asphyxia including two patients with spastic cerebral palsy and one patient with ataxic cerebral palsy. Two female patients had history of meningitis before the diagnosis of cerebral palsy, one patient had spastic quadriplegic cerebral palsy and one patient aged ten months didn't have significant spasticity, but the possibility of later development of spasticity couldn't be excluded.

One female had affected brother with spastic cerebral palsy and imaging study showing findings similar to the findings of leukodystrophy. Eye abnormalities were present in six patients: Two patients (a boy and a girl) had refractive error. Two patients (a boy and a girl) had squint in the girl it resulted in torticollis. Two patients (a boy and a girl) had significant reduction of visual acuity most probably resulting from optic atrophy, and the male patient was almost totally blind. Four patients had history of epileptic seizures including three male patients, and one girl, and in two boys seizures required long term anticonvulsant therapies.

A boy with spastic cerebral palsy had a pelvic MRI showing atrophic undescended right testis located in the mid-inguinal region, and a girl with spastic cerebral palsy had atrial septal defect. Nineteen patients had brain imaging studies including ultrasonography, CT-scan and MRI. Three of these nineteen patients had normal findings. Nine patients had evidence of diffuse brain atrophy on brain imaging studies. Two of these nine patients had additional unique findings including one patient had polymicrogyria and dilatation of the basal

cisterns, and a second patient had bilateral parietal lobe white matter peri-ventricular leukomalacia. Seven patients had unique findings other than evidence of diffuse brain atrophy: One patient had left hemi-brain atrophy. One patient had vermian hypoplasia with retro-cerebellar hypoplasia. One patient had absence of the inferior vermis and cerebellar atrophy. One patient had diffuse hypo density of both cerebral hemispheres suggesting hypoxic brain ischemia caused by birth asphyxia.

One patient had evidence of hypoxic ischemic encephalopathy. One patient had large right sided temporo-parietal gliosis changes with large communicating porencephaly cyst. One patient had mild dilatation of the right ventricle.

Patients with spastic cerebral palsy affecting the four limbs (Spastic quadriplegia) included the two patients who had the most severe condition in this series when first seen the two patients had impaired consciousness. They were not opening their eyes, and were showing almost no spontaneous movements and they responded poorly too painful stimuli. Both patients had poor feeding, and both had seizures and required long term anti-convulsants. Both patients showed improvement after several months of treatment courses consisting of a combination of various interventions including nutritional support, muscle relaxants, oral pyritinol, intramuscular piracetam, citicoline (oral and injectable), intramuscular cerebrolysin, and intramuscular nandrolone decanoate.

Figure 1 shows the older of the two patients with the most severe disorder who was first seen at about the age of six years after more than ten months on therapy. He showed improved muscle tone with significant reduction of spasticity and he was able to: Sit alone on the chair with good head control. Recognize his mother. Express happiness by smiling and laughing. One of the patients with spastic cerebral palsy had a definite history of birth asphyxia (Figure 2). One female patient with spastic cerebral palsy had meningitis before the onset of cerebral palsy which was associated with evidence of diffuse



Figure 1: A boy with severe spastic cerebral caused by brain atrophy after several months of treatment, he was able to sit alone on the chair with good head control and he was expressing happiness by smiling.



Figure 2: The patient with spastic cerebral palsy caused by birth asphyxia. At the age of sixteen months he was still unable to sit on the chair without slipping.

brain atrophy on brain MRI. Before treatment at about the age of one year (Figure 3A), she had marked spasticity with torticollis and limited spontaneous movement and poor feeding.

She was not recognizing mother face nor responding to voice. The girl received four treatment courses over about four months (Table 1).

After four treatment courses (Figure 3B and C), the girl showed improved muscle tone with significant reduction of spasticity, and she was able to: Sit alone on the chair with good head control. Recognize his mother and turning to voice. Three of the patients with spastic quadriplegia had ocular abnormalities including a girl with refractive error and two patients (a boy and a girl) with serious reduction of visual acuity most probably caused by optic atrophy.

The girl with refractive error was first seen at about the age of about seventeen months during August 2018. She was unable to sit alone on a chair and spasticity limited her movements. She received five treatment courses (Table 2).

After treatment (Figure 4), she could sit on the chair and was able to stand holding furniture and making few steps and her speech development was initiated.

The second patient with spastic quadriplegia and ocular abnormality was a ten year old girl with severe growth retardation (Her weight was 12 kilograms) and optic atrophy. When she was first



Figure 3: (A) The female patient with post-meningitis brain atrophy and spastic quadriplegia. Before treatment at about the age of one year she had marked spasticity with torticollis and limited spontaneous movement and poor feeding. She was not recognizing mother face nor responding to voice. (B, C) After four treatment courses the girl showed improved muscle tone with significant reduction of spasticity, and she was able to sit alone on the chair with good head control.

Table 1: Treatment courses received by the patient with post-meningitis brain atrophy and spastic quadriplegia.

First course (one month)
Oral baclofen 2.5 mg three times, increased to 5 mg daily over ten days.
Cerebrolysin 1 ml daily give intramuscularly for 30 days.
Oral citicoline 2 ml (200 mg) in the morning.
Second course (one month)
Oral baclofen 5 mg three times.
Intramuscular piracetam 1.5 ml (300 mg) every other day (10 doses).
Oral citicoline 2 ml (200 mg) in the morning.
Third course (one month)
Oral baclofen 10 mg two times.
Intramuscular cerebrolysin 1 ml daily for 20 days.
Oral citicoline 2 ml (200 mg) in the morning.
Nutritional support mainly in the form of royal jelly oral capsules given once daily.
Fourth course (one month)
Oral baclofen 5 mg three times.
Intramuscular cerebrolysin 1 ml every third day, 10 doses over 1 month.
Oral citicoline 3 ml (300 mg) in the morning.

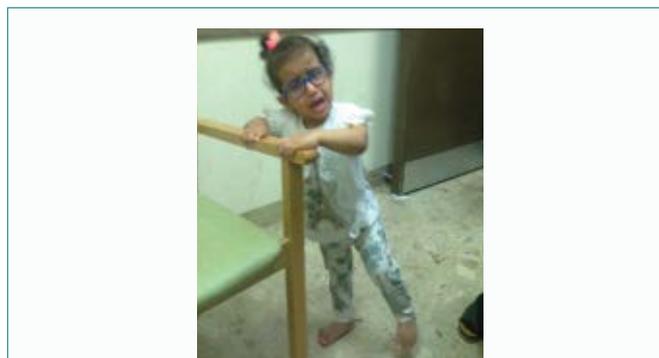


Figure 4: The girl with spastic quadriplegia and refractive error. After treatment, she could sit on the chair and was able to stand holding furniture and making few steps and her speech development was initiated.

Table 2: The five treatment courses received by the girl with spastic quadriplegia and refractive error.

First course (one month) started at about the age of 17 months during August 2018
Baclofen (oral) 2.5 mg three times daily increased to 5 mg three times daily over 10 days.
Oral citicoline 3 ml (300 mg) in the morning
Second course
Baclofen (oral) 10 mg twice.
Intramuscular citicoline 3 ml (375 mg) every third day, 10 doses.
Third course
Baclofen (oral) 10 mg three times.
Intramuscular cerebrolysin 1 ml every third day, 10 doses.
Fourth course started on the 17 th of December,2018
Baclofen (oral) 10 mg three times.
Diazepam (oral) 2 mg at night for 10 days.
Oral citicoline 3 ml in the morning
Intramuscular cerebrolysin 1 ml every other day, 10 doses
Fifth course started at the age of 23 months
Baclofen (oral) 10 mg three times.
Intramuscular piracetam 2 ml (400 mg) every third day, 10 doses over one month.
Intramuscular nandrolone decanoate 12.5 mg.
Oral Citicoline 3 ml (300 mg) in the morning

seen on the fourth of March, 2019 (Figure 5A), she had very poor feeding, and marked spasticity that limited her movements. She was unable to sit on chair with head control. She didn't show obvious alertness or response to voice and was not saying any word.

The first course of treatment included

Baclofen (oral) 2.5 three times increased to 25 mg daily over 10 days. Intramuscular cerebrolysin 1 ml daily 10 doses oral citicoline 2 ml (200 mg), daily Nutritional support mainly in the form of royal jelly oral capsules.

After treatment (Figure 5B), she showed improvement in feeding and spasticity. She became more alert to the environment and responding to voices. She was able to sit on a chair with better head control. She was also expressing happiness by smiling. The third patient with spastic quadriplegia and ocular abnormality was a three-year old boy who was blind (Figure 6) and had optic atrophy One of the patients with spastic quadriplegia who had a tendon lengthening operation, but was able to walk only while holding furniture (Figure 7).

The older patient with spastic quadriplegia was a twelve-year old boy who was the only patient who was rather overweight, and had normal speech development. He was unable to stand or walk even when holding furniture and was using a wheelchair (Figure 8).

A boy with spastic cerebral palsy (Figure 9) with brain MRI showing evidence of diffuse brain atrophy, and had a pelvic MRI



Figure 5: (A). The girl with spastic quadriplegia and optic atrophy. She had very poor feeding and marked spasticity that limited her movements. She was unable to sit on chair with head control. She didn't show obvious alertness or response to voice and was not saying any word. (B). After treatment, the girl showed improvement in feeding and spasticity, and she became more alert to the environment and responding to voices. She was able to sit on a chair with better head control. She was also expressing happiness by smiling.



Figure 6: A three-year old boy with spastic quadriplegia who was blind and had optic atrophy.



Figure 7: A boy with spastic quadriplegia who had a tendon lengthening operation but was able to walk only while holding furniture.

showing atrophic undescended right testis located in the mid-inguinal region. Figure 10 shows a girl with spastic cerebral palsy had small secundum atrial septal defect.

Twelve patients with spastic quadriplegia had brain imaging studies including ultrasonography, CT-scan and MRI.

Three patients had normal findings on brain imaging studies including the patient in (Figure 4) who had normal brain ultrasound

and CT-scan, and a six year old girl (Figure 11) with growth retardation (weight:12 kilograms) who didn't have marked hypertonia and didn't need muscle relaxants. The girl had history of seizures and had normal brain CT-scan. She was unable to walk and was not speaking. Initial treatment included intramuscular cerebrolysin 1 ml daily for 10 days and oral citicoline 2 ml (200 mg) in the morning.



Figure 8: The older patient with spastic quadriplegia was a twelve-year old boy who was the only patient who was rather overweight and had normal speech development.



Figure 9: A six-year boy with spastic cerebral palsy had a pelvic MRI showing atrophic on descended right testis located in the mid-inguinal region. He was unable to stand or walk even when holding furniture.



Figure 10: A girl with spastic cerebral palsy who had small secundum atrial septal defect. She had significant spasticity that limited her movements and was unable to stand or walk even when holding furniture.

The third patient with spastic quadriplegia and normal findings on brain imaging studies was a six month old girl. When she was first seen on the 25th of March, 2019 (Figure 12A), spasticity markedly limited her movements, and she was not obviously alert to the environment and was not responding to voice. She was initially treated with intramuscular cerebrolysin 1 ml daily for 10 days and oral baclofen 5 mg twice daily. When she was seen on the 18th of April, 2018, (Figure 12B), she had less limitation to movements, was more alert and responsive to voice.

Seven patients with spastic quadriplegia had evidence of diffuse brain atrophy on brain imaging studies including the three patients on (Figures 1, 3 and 9), three girls (Figures 13-15) and a boy who had brain atrophy associated with polymicrogyri and dilatation of the basal cisterns. The brain MRI of the girl with diffuse brain atrophy in (Figure 15) also showed bilateral parietal lobe white matter periventricular leukomalacia and mildly dilated lateral ventricle.

The boy who had brain atrophy associated with polymicrogyri and dilatation of the basal cisterns was first seen on the 25th of February, 2019 (Figure 16A). He had limited spontaneous movements, poor head control, and was unable to sit alone on the chair.

The first one month course of treatment started on the 25th of February, 2019 included: Oral baclofen with an initial dose of 2.5 mg once daily increased gradually over one month to 5mg twice daily.



Figure 13: Seventeen months old girl weighing 5.5 kilograms was seen 4th of March, 2019. She had spastic quadriplegia caused by brain atrophy.



Figure 14: Three years and five months old girl weighing 5.5 kilograms with spastic quadriplegia associated with diffuse brain atrophy.



Figure 11: A six year old girl with spastic quadriplegia who didn't have marked hypertonia and didn't need muscle relaxants. She was unable to walk and was not speaking.



Figure 15: The girl with spastic quadriplegia and diffuse brain atrophy associated with bilateral parietal lobe white matter periventricular leukomalacia and mildly dilated lateral ventricle.



Figure 12: (A). The third patient with spastic quadriplegia and normal findings on brain imaging studies was a six month old girl with spasticity markedly limited her movements, and she was not obviously alert to the environment and was poorly responding to voice. (B). After the first treatment course, she had less limitation to movements, was more alert and responsive to voice.

Intramuscular cerebrolysin 1 ml daily for 10 days followed by 1 ml every other day (20 doses were given over one month). Oral citicoline 2 ml (200mg) daily in the morning after treatment (Figure 16B), spasticity improved with more spontaneous movement and the boy was able to sit alone on the chair for short time. In two patients (A boy and girl) with spastic quadriplegia, imaging studies showed findings other than diffuse brain atrophy. The brain MRI of the girl in figure-10 who had small secundum atrial septal defect showed mild dilatation of the right ventricle with normal third and fourth ventricles.

The CT-scan of a four-month old boy with spastic quadriplegia and a history of birth asphyxia showed diffuse hypo density of both cerebral hemispheres sparing the cerebellar hemispheres and the basal ganglia suggesting hypoxic brain ischemia caused by birth asphyxia.

The boy was first seen on the 15th of April, 2019 (Figure 17) with poor spontaneous movements and poor head control. Initial treatment included oral baclofen 2.5 mg three times daily and intramuscular cerebrolysin 1 ml daily, 10 doses.

The four patients (Three boys and one girl) who didn't have significant spasticity included:

1. A thirteen months old boy (Figure 18) weighing 11 Kilograms who was first seen on the 8th April, 2019. He was unable to stand and was not babbling. Brain CT scan showed evidence of brain atrophy with prominent lateral ventricles. He was initially treated with intramuscular cerebrolysin 1 ml daily for days 10 and oral citicoline 2 ml (200 mg) in the morning.
2. Two years and five months old boy (Figure 19A) weighing 12 kilograms was first seen on the 4th of March, 2019. He had convergent squint of the left. He was unable to walk alone and was walking with difficulty holding furniture (Figure 19B). He was unable to feed himself neither with a bottle nor with spoon and was not saying any word. He didn't need muscle relaxant and he was initially treated with intramuscular cerebrolysin 1 ml every other day, 10 doses, and oral citicoline 200 g daily in the morning.
3. A ten months old girl who was hospitalized for two weeks (4 to 14, July, 2018) at the age of two and half months because of meningitis. She had seizures and her brother was epileptic. She was first seen on the 24th of January, 2019, at about the age of 10 months (Figure 20). She was hypotonic had poor spontaneous movement and poor head control and was unable to sit. She was initially treated with intramuscular cerebrolysin 1 ml daily, 10 doses, and oral citicoline 1 ml (100 mg) daily in the morning.
4. A twenty-two month old boy (Figure 21) weighing 8.45 Kilograms with hemi-brain atrophy. He was unable to stand and walk and was not speaking. He was initially treated with intramuscular cerebrolysin 1 ml every fifth day and intramuscular citicoline 2 ml (250 mg) every fifth day.

The four patients (One boy and three girls) with hemiplegic cerebral palsy included:

1. A one-year old girl (Figure 22) with hemiplegic cerebral palsy and squint causing torticollis Brain MRI showed signal intensity involving the right thalamic, midbrain and hippocampus region. Dilation of the right horn of the right lateral ventricle was also present suggesting ischemic injury.
2. A nine-year old boy (Figure 23) who was first seen on the 4th of March, 2019. He had abnormal gait caused by right hemiplegia. He also had epileptic seizures that were controlled with carbamazepine.
3. A seven-year old girl with hemiplegia cerebral palsy with weakness on the right side of the body She had gait abnormality mostly in form of dragging her right leg. She was able to make squat without obvious difficulty, and she could take and carry weight with her left arm for good time. However, she had obvious difficulty in taking weight with her right arm, and she was only able to carry the weight momentarily after taking the weight with her left arm



Figure 16A: The boy who had brain atrophy associated with polymicrogyri and dilatation of the basal cisterns was first seen at the age of 9 months on the 25th of February 2019. He had limited spontaneous movements, poor head control, and was unable to sit alone on the chair.



Figure 16B: The boy who had brain atrophy associated with polymicrogyri and dilatation of the basal cisterns. After treatment, spasticity improved with more spontaneous movement and the boy was able to sit alone on the chair for short time.



Figure 17: The boy with spastic quadriplegia caused by hypoxic brain ischemia. He had poor spontaneous movements and poor head control.

and putting it in her right hand (Figure 24). The girl was doing well at first year primary school and the decision was made to follow up the girl and encouraging physiotherapy as her disability was not regarded to be significantly serious.

4. A five-year old girl with Hemiplegia cerebral palsy with weakness on the left side of the body She had gait abnormality mostly in form of dragging her left leg. She could take and carry weight with her right arm for some time, but she could not take the same weight with her left arm, and she could not prevent herself from



Figure 18: A thirteen-month old boy with cerebral palsy without significant hypertonia associated with brain atrophy. He was unable to stand and was not babbling.



Figure 19: (A). A two years and five months old boy had cerebral palsy without significant hypertonia and had convergent squint of the left. He was unable to walk alone and was walking with difficulty holding furniture. He was unable to feed himself neither with a bottle nor with spoon and was not saying any word.
(B). A two years and five months old boy had cerebral palsy without significant hypertonia and had convergent squint of the left. He was unable to walk alone and was walking with difficulty holding furniture.



Figure 20: Ten months old girl with post-meningitis cerebral palsy. She was hypotonic had poor spontaneous movement and poor head control and was unable to sit.

using her right arm when she was encouraged to keep trying. The girl was able to take a pen to try copying a line and a circle, but she couldn't (Figure 25). Brain CT scan showed large right sided temporo-parietal gliotic changes with large communicating porencephaly cyst. The decision was made to follow up the girl and encouraging physiotherapy as her disability was not regarded to be significantly serious.

The three male patients with ataxic cerebral palsy included:

1. A boy whose brain CT scan showed absence of the inferior vermis and cerebellar atrophy, and the posterior fossa was filled with CSF.
2. A two year boy (Figure 26A) weighing 14 kg. He was hypotonic unable to sit, unable to stand or walk and was not speaking. Brain MRI showed evidence of vermian hypoplasia with retro-cerebellar hypoplasia. He was initially treated with intramuscular cerebrolysin 1ml daily for 10 days and oral citicoline 2 ml (200 mg) in the morning. Treatment resulted in a significant improvement in muscle tone and he could sit alone on a chair (Figure 26B).
3. Three years and 3 months old boy (Figure 27A) weighing 15.5 kg with ataxic cerebral palsy caused by birth asphyxia. He had abnormal movements with poor balance and was unable to stand erect even when holding furniture. He was just babbling and was unable to drink from cup. Brain MRI showed generalized cerebral atrophy. He was initially treated with intramuscular cerebrolysin 3 ml daily for 10 days, followed by cerebrolysin 5 ml every other day (20 doses of cerebrolysin over one moth), with addition of oral citicoline 3 ml (300 mg) daily in the morning citicoline. Treatment resulted in: Reduction in abnormal movements with improvement of balance and became able to stand comfortably erect holding furniture (Figure 27B). Improvement in fine motor skills with the ability of holding small objects and holding a pencil with help of parents to copy a circle and a square (Figure 27C).



Figure 21: A twenty-two month old boy with cerebral palsy without significant hypertonia caused by hemi-brain atrophy. He was unable to stand and walk and was not speaking.



Figure 22: A one-year old girl with hemiplegic cerebral palsy caused by ischemic injury and squint causing torticollis.



Figure 23: A nine-year old boy who had right hemiplegic cerebral palsy.



Figure 24: A seven-year old girl with hemiplegic cerebral palsy with weakness on the right side of the body. She had obvious difficulty in taking weight with her right arm, and she was only able to carry the weight momentarily after taking the weight with her left arm and putting it in her right hand.



Figure 25: A five-year old girl with left hemiplegic cerebral palsy. She could take and carry weight with her right arm for some time but she could not take the same weight with her left arm and she could not prevent herself from using her right arm when she was encouraged to keep trying. The girl was able to take a pen to try copying a line and a circle but she couldn't.



Figure 26: (A). A two year boy with ataxic cerebral palsy caused by vermian hypoplasia and retro-cerebellar hypoplasia. He was hypotonic unable to sit unable to stand or walk and was not speaking. (B). A two year boy with ataxic cerebral palsy caused by vermian hypoplasia and retro-cerebellar hypoplasia. Treatment resulted in a significant improvement in muscle tone and he could sit alone on a chair.



Figure 27A: Three years and 3 months old boy with ataxic cerebral palsy caused by birth asphyxia. He had abnormal movements with poor balance and was unable to stand erect even when holding furniture.



Figure 27B: Treatment of the boy with ataxic cerebral palsy resulted in reduction in abnormal movements with improvement of balance and became able to stand comfortably erect holding furniture.



Figure 27C: Treatment of the boy with ataxic cerebral palsy resulted in improvement in fine motor skills with the ability of holding small objects and holding a pencil with help of parents to copy a circle and a square.

Discussion

The spastic type of cerebral palsy is by far the most common type accounting for about 70% of all cases [2,4]. In this series of Iraqi patients with cerebral palsy, spastic cerebral palsy affecting all limbs accounted for about 67% of all patients. 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.

The use of oral pyritinol with the judicious use of nandrolone decanoate given by intramuscular injection intermittently has been reported to be associated with a beneficial effect on motor development and learning in patients with cerebral palsy [1,2]. The use of oral pyritinol, intramuscular piracetam, citicoline (oral and injectable), intramuscular cerebrolysin, and intramuscular nandrolone decanoate has been reported to have a beneficial effect in a very severe form of spastic cerebral palsy associated with radiologic evidence of brain atrophy [3].

Recently, Al Mosawi [5] described the treatment of six patients (3 girls and 3 boys) with spastic cerebral palsy with individualized treatment plans providing a new combination of interventions including nutritional support, muscle relaxants and the use of oral pyritinol, intramuscular piracetam, citicoline (oral and injectable), intramuscular cerebrolysin, and intramuscular nandrolone decanoate. Treatment aimed primarily at improving motor development particularly standing and walking. The patients' age in the study of Al Mosawi ranged from 22 months to three years. All patients were unable to stand or walk, and had poor speech development. Four patients had severe cerebral palsy and were even not able to sit. The other two patients had moderately severe disorder and were unable to stand or walk. All the patients in the study of Al Mosawi were not saying any word or were saying only few words.

After treatment, all the patients experienced improvement in motor development without the occurrence of any side effect. Five patients were able to stand with support, and four of them were also able to walk few steps with support.

The sixth patient remained unable to stand and the limited benefit of treatment was attributed to some degree of deformity and muscle contracture. In all patients treatment was associated with initiation of speech development or improved speech. In the study of Al Mosawi [5], it was possible to demonstrate improvement in fine motor skills in three patients. In this series, patients were treated 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 use of these relatively new therapies was based on our previous extensive experience with the use of these disorders in various disorders [6-10].

Conclusion

Spastic cerebral palsy affecting all limbs accounted for about 67% of all Iraqi children with cerebral palsy. Radiologic evidence of diffuse brain atrophy was present in more than 25% of the patients with cerebral palsy in this series.

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