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The Initial Treatment of a Girl From Texas With Post-Infantile Acquired Cerebral Palsy Caused by Submersion Injury

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Abstract

Background: *Cerebral palsy is a heterogeneous condition that can cause a lifelong neurological impairments resulting from a non-progressive brain injury. It is commonly caused by antenatal, perinatal, and early postnatal and neonatal conditions. However, post-neonatal cases of acquired cerebral palsy have also been reported, and were commonly caused by infection. We have previously reported the uncommon situation of the occurrence of post-infantile acquired cerebral palsy caused by submersion injury in a girl from Qatar.*

Patients and methods: *During the first week of December, 2021, the mother of a three-year old child from Texas with acquired cerebral palsy consulted us about the possible therapies for the difficult condition of her child. The girl developed cerebral palsy after she survived a drowning accident and cardiac arrest at the age of 20 months (November 6, 2020).The child record was studied and an evidence-based therapeutic recommendation was made which included multi-factorial evidence-based therapies.*

Results: *After the initial one-month intensive multi-factorial therapies, improvements in alertness, posture, and head control were observed.*

Conclusion: *The management of severe brain damage remains challenging, and without the judicious use of evidence-based multi-factorial therapies, management is generally not expected to be rewarding.*

Keywords : Post-infantile acquired cerebral palsy, Texas USA, multi-factorial therapies.

Introduction

Cerebral palsy is a heterogeneous condition that can cause a lifelong neurological impairments resulting from a non-progressive brain injury. It is commonly caused by antenatal, perinatal, and early postnatal and neonatal conditions. However, post-neonatal cases of acquired cerebral palsy have also been reported, and were commonly caused by infection (Al Mosawi, 2008 ; Al Mosawi, 2017; Al Mosawi, 2017; Al Mosawi, 2019; Al-Mosawi, 2019; Al-Mosawi, 2019; Al-Mosawi, 2019; Al-Mosawi, 2020; Al-Mosawi, 2020; Al-Mosawi, 2022). We have previously reported the uncommon situation of the occurrence of post-infantile acquired cerebral palsy caused by submersion injury in a girl from Qatar (Al-Mosawi, 2022).
Patients and methods

Patients and methods

During the first week of December, 2021, the mother of a three-year old child from Texas with acquired cerebral palsy consulted us about the possible therapies for the difficult condition of her child. The girl developed cerebral palsy after she survived a drowning accident and cardiac arrest at the age of 20 months (November 6, 2020). The child record was studied and an evidence-based therapeutic recommendation was made.

The girl was born on the second of October, 2018, and was otherwise healthy until the 11th of June, 2020, when she experienced a submersion injury which resulted in cardiac arrest. After surviving the cardiac arrest, the diagnosis of severe hypoxic ischemic encephalopathy resulting in spastic quadriplegia was made on the seventh of December, 2020. The girl has been tracheostomy-dependent since the16th of April, 2021, and has been fed by gastric tube since the seventh of August, 2020. Seizure was first reported on the second of September, 2020.

The seizures and spasticity were controlled with oral

levetiracetam (KEPPRA) [750 mg twice daily started on June, 27/2021, and reduced to 500 mg twice daily on September, 29, 2021], oral clonazepam 0.25 mg 6 hourly, started on October, 5, 2021, and oral baclofen 20 mg three times daily, started on November, 29, 2021. Ketogenic diet was reported to be necessary for seizure control

She was also receiving gabapentin and carbidopa/levodopa with hope of reducing spasticity and dystonia.

Her condition was complicated by bilateral coxa valga, and right hip subluxation that did not reduce with frog-leg positioning, it observed on X-ray of the pelvis on June 4, 2021. Her illness was also complicated by S-shaped scoliosis which was observed on MRI September, 20, 2021.

Before October, 2021, the girl was showing poor response to the environment, and she had no spontaneous movements (Figure-1A). She had poor head control (Figure-1) and was unable to sit without support

Figure-1A: Before October, 2021, the girl was showing poor response to the environment, and had no spontaneous movements

Brain imaging studies included CT-scan on June 11, 2020, MRI on June, 15, 2020, and July, 23, 2020. These early two imaging studies suggested anoxic/hypoxic ischemic brain injury, and showed interval volume loss with widening of the extra-axial CSF spaces and sulci and increased ex vacuo dilatation of the supratentorial ventricular system. The posterior fossa contents showed ex vacuo dilatation of the fourth ventricle in the presence of prominence to the cerebellar folia. There was abnormal T2 hyperintensity with persistent unrestricted diffusion change returned from the bilateral posterior globus pallidus and putamen. There was bilateral and symmetric under-opercularization.

The MRI on July, 23, 2020 showed abnormal T2 hyperintensity returned from the bilateral posterior observed on and was likely representing gliosis. There was no restricted diffusion or abnormal susceptibility.

The hippocampi appear small. Midline structures demonstrate thinning of the corpus callosum and brainstem structures. Expected flow voids are demonstrated in the arteries about the circle of Willis.

The MRI on July, 23, 2020 suggested interval atrophy. The largest space within the sulci between the brain and inner table of the skull larger lateral ventricles third ventricle and fourth ventricle.

There was a focal encephalomalacia seen especially in the putamen with surrounding FLAIR hyperintensity consistent with microcystic gliosis. The thalami have become smaller in size/atrophic and contains some focal increased T2 and FLAIR hyperintensity especially on the left. Some atrophy of the caudate head bilaterally.

Minimal T2 hyperintensity posterior lateral aspect of the pons extending in the middle cerebellar peduncles bilaterally. There is also T2 hyperintensity seen in the peri-aqueductal gray region of the upper midbrain bilaterally extending toward the thalami finally, there is some volume loss in the region of hippocampi. There is no abnormal diffusion restriction.

Paranasal sinuses mastoid air cells middle ear show fairly extensive circumferential mucosal thickening in right greater left maxillary sinuses and fluid in some of the ethmoid air cells right greater than left and some fluid in mastoid air cells middle ears right greater than left.

Therefore, brain imaging diagnoses include progressive volume loss and atrophy with the development of cystic and microcystic encephalomalacia. Changes were probably most extensive within the deep gray matter nuclei including the putamen and thalami bilaterally. There was also diffuse volume loss of cortex and subcortical white matter including the hippocampi.

Her head circumference was 49 cm on September, 29, 2021 [Percentile 59.54 %].

The mother reported that on October 13, 2021, they started treating the girl with one of the protocols I have recommended in a previous publication which included oral citicoline 300 mg daily plus cerebrolysin 2.5ml given intra-muscularly every other day. Within 6 weeks, a reduction in tone and improved alertness were observed and spontaneous movements with movement of her fingers appeared after the initial treatment with cerebrolysin and citicoline (Figure-1B).

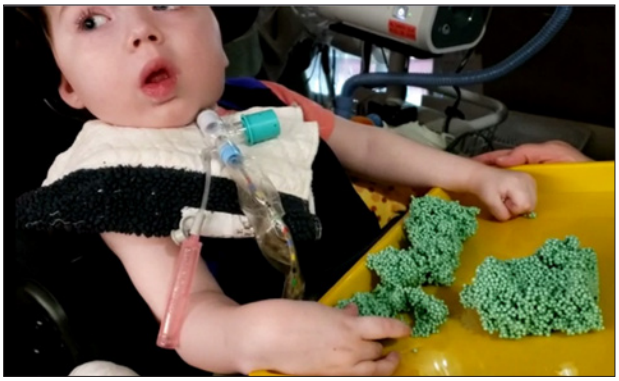

Figure-1B: A reduction in tone and improved alertness were observed and spontaneous movements with movement of her fingers appeared after the initial treatment with cerebrolysin and citicoline

Intensive multi-factorial therapies were prescribed based on our extensive published experiences and the one- month of intensive multi-factorial therapies include (Al Mosawi, 2008 ; Al Mosawi, 2017; Al Mosawi, 2017; Al Mosawi, 2019; Al-Mosawi, 2019; Al-Mosawi, 2019; Al-Mosawi, 2019; Al-Mosawi, 2020; Al-Mosawi, 2020; Al-Mosawi, 2022; Al-Mosawi, 2021; Al-Mosawi, 2021; Al-Mosawi, 2020; Al-Mosawi, 2020; Al-Mosawi, 2020).

1. Cereblolsin 3 ml intramuscularly every other day during the morning hours (15 doses over one month).
2. Oral citicoline syrup 3 ml (300 mg) daily in the morning.
3. Nootropil (Piracetam) 2 ml (200mg) intramuscularly every other day during the morning hours (15 doses over one month) [Not on the same day of cerebrolysin].
4. Oral pyritinol 50 mg daily at 5 pm.

Results

After the initial one-month intensive multi-factorial therapies, improvements in alertness, posture, and head control were observed (Figure-2).

Figure-2: After the initial one-month intensive multi-factorial therapies, improvements in alertness, posture, and head control were observed

Discussion

Cerebral palsy is a heterogeneous condition that can cause a lifelong neurological impairments resulting from a non-progressive brain injury. It is commonly caused by antenatal, perinatal, and early postnatal and neonatal conditions. However, post-neonatal cases of acquired cerebral palsy have also been reported, and were commonly caused by infection (Al Mosawi, 2008; Al Mosawi, 2017; Al Mosawi, 2017; Al Mosawi, 2019; Al-Mosawi, 2019; Al-Mosawi, 2019; Al-Mosawi, 2019; Al-Mosawi, 2020; Al-Mosawi, 2020; Al-Mosawi, 2022). We have previously reported the uncommon situation of the occurrence of post-infantile acquired cerebral palsy caused by submersion injury in a girl from Qatar (Al-Mosawi, 2022).

Blair and Stanley (1982) reported that 11% of cases of cerebral palsy in Western Australia were postnatally-acquired condition, and males under one year of age, were particularly vulnerable. Infections such as meningitis and encephalitis accounted for more than 50% of the cases, and accidents accounted for about 25% of the cases. Other causes included epileptic fits and cerebrovascular accidents (Blair & Stanley, 1982).

Arens and Molteno (1989) from South Africa reported that the chief causes of postnatal acquired cerebral palsy were cerebral infections (particularly meningitis), cerebral trauma and cerebrovascular accidents (Arens & Molteno, 1989).

Murphy et al (1993) from the USA reported that the Metropolitan Atlanta Developmental Disabilities (A population-based study, 1985-1987) found that 16% of children with cerebral palsy had a postnatally-acquired condition (Murphy et al., 1985).

Cans et al (2004) reported that 50% of cases of cerebral palsy with post-neonatal origin (arising more than 28 days after birth, and before the age of 25 months) were caused by infection;20% caused by vascular episodes, 18% caused by head injury. They suggested that children with cerebral palsy of post-neonatal origin had a more severe functional pattern than non-post-neonatal cerebral palsy children (Cans et al., 2004).

Reid and colleagues (2006) from Australia reported that 10.7% of 339 cases of cerebral palsy had post-neonatally acquired condition caused by infection, traumatic head injury a, hypoxia, acute encephalopathies, and cerebrovascular accidents (Reid et al., 2006).

The work of Salih (2020) from Sudan emphasized that prenatal causes were the most common causes of cerebral palsy, but post natal causes included neonatal jaundice and acquired sepsis (Salih, 2020).

For the patient in this report, we suggested the use of evidence-based treatments which included intramuscular cerebrolysin and oral citicoline.

Cerebrolysin solution contains free amino acids (85%) and 15% biologically active low molecular weight amino acids including neuro-peptides (Brain-derived neurotrophic factor, glial cell line-derived neurotrophic factor, nerve growth factor, ciliary neurotrophic factor (Al-Mosawi, 2020). Cerebrolysin has been used safely with benefit in a variety of neuro-psychiatric disorders including idiopathic mental retardation (Al-Mosawi, 2020; Al-Mosawi, 2020), cerebral palsy (Al-Mosawi, 2019; Al-Mosawi, 2020), myelomeningocele (Al-Mosawi, 2019), pediatric juvenile spinal muscular atrophy (Al-Mosawi, 2018; Al-Mosawi, 2020), pediatric Charcot Marie Tooth disease (Al-Mosawi, 2018; Al-Mosawi, 2020), kernicterus (Al-Mosawi, 2019; Al-Mosawi, 2019), agenesis of corpus callosum with colpocephaly (Al-Mosawi, 2019; Al-Mosawi, 2020).

Citicoline, which has been increasingly grouped with the water soluble B vitamins, and is regarded as a form of the essential nutrient choline. It has been increasingly used with noticeable benefits in the treatment of several pediatric neuro-psychiatric disorders including, pervasive developmental disorders including Rett syndrome, and kernicterus (Al-Mosawi, 2019; Al-Mosawi, 2019).

Pyritinol which is also called pyridoxine disulfide is a semi-synthetic water soluble analog of pyridoxine. It has been

reported to be of benefits in patients with cerebral palsy, and the benefit has been attributed to cerebral blood supply improvement which causes an improvement of nerve cell metabolism (Al Mosawi, 2017; Al Mosawi, 2017; Al-Mosawi, 2020).

Conclusion

The management of severe brain damage remains challenging, and without the judicious use of evidence-based multi-factorial therapies, management is generally not expected to be rewarding.

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Conflict of interest

None.

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