

A Girl from Qatar with Post-Infantile Acquired Cerebral Palsy Caused By Submersion Injury: A Rare Etiology and a Therapeutic-Challenge.

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Received date: December 11, 2021; **Accepted date:** December 27, 2021; **Published date:** January 08, 2022

Citation: Aamir J. Al-Mosawi. (2022). A Girl from Qatar with Post-Infantile Acquired Cerebral Palsy Caused By Submersion Injury: A Rare Etiology and a Therapeutic-Challenge. *J Clinical Research and Reports*, 10(2); DOI:10.31579/2690-1919/222

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Abstract

Background: Cerebral palsy is a heterogeneous disorder that can cause a lifelong disability that is associated with a non-progressive damage in the brain. It is commonly caused by antenatal, perinatal, early postnatal and neonatal conditions. However, post-neonatal cases of acquired cerebral palsy have also been reported, and were commonly caused by infection.

Patients and Methods: The family of a girl from Qatar, who developed severe cerebral palsy caused by submersion injury, consulted us about the possible therapies for her condition. Clinical picture and brain imaging abnormalities are described, and the relevant literatures were reviewed with the aim of suggesting possible evidence-based therapies.

Results: At the age of 23 months, a previously healthy girl developed anoxic encephalopathy after experiencing submersion injury. MRI showed evidence of significant hypoxic ischemic injury primarily affecting the deep grey matter, hippocami, mid-brain and the posterior cortex. EEG showed diffuse slowness of cerebral activity and diffuse attenuation of the background without no epileptic abnormalities suggesting diffuse encephalopathy resulting from diffuse cortical injury. At the about age of three and half years, her family consulted us about her condition as she was still showing no awareness to the environment, showing no significant spontaneous movements. She had poor head control. Unable to sit or stand alone, and had a flexed posture. She was on levetiracetam (Keppra), diazepam, and baclofen 30 mg daily. She was still having tracheotomy, and was fed through gastrostomy tube.

Conclusion: In this paper, the rare occurrence of severe post-infantile cerebral palsy is described. Emphasis is made on the possibility of using evidence-based multi-factorial therapies in cerebral palsy.

Keywords: acquired post-infantile; cerebral palsy; submersion injury; Qatar; multi-factorial therapies

Introduction

Cerebral palsy is a heterogeneous disorder that can cause a lifelong disability that is associated with a non-progressive damage in the brain. 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 [1-10].

Patients and methods

The family of a girl from Qatar, who developed severe cerebral palsy caused by submersion injury, consulted us about the possible therapies for her condition. Clinical picture and brain imaging abnormalities are

described, and the relevant literatures were reviewed with the aim of suggesting possible evidence-based therapies.

Results

At the age of 23 months (February 8, 2020), a previously healthy girl who was born on the 1st of March, 2018, was admitted to the emergency department at Sidra Medicine Hospital in Doha after experiencing submersion injury in the home garden swimming pool. She had cardiac arrest when she arrived at the emergency unit and required about ten minutes of cardiopulmonary resuscitation to bring her back, and she was admitted to the intensive care unit.

A diagnosis of anoxic encephalopathy was made. Early CT-scan didn't show abnormalities, but she started to experience myoclonic seizures affecting all of her limbs. Seizures were initially treated with i.v Keppra and phenobarbitone. Midazolam drip and thiopental continuous infusion were added on the 9th of February because seizures continued to occur. After few days, it was possible to control her seizures with Keppra alone in a dose of 60 mg/ kg given in 2 divided doses.

MRI performed on the 12th of February showed evidence of significant hypoxic ischemic injury primarily affecting the deep grey matter, hippocami, mid-brain and the posterior cortex.

On the 19th night of hospitalization she started experiencing cough of gag reflex with deep suctioning for the first time.

On the 15th of March, in addition to Keppra 60 mg/ kg. She was also receiving baclofen 12.5 mg three times a day and diazepam 5 mg a day to control the developing spasticity.

On the 22nd of March, tracheostomy was performed and she was off the ventilator and oxygen support, Gastrostomy tube was also inserted on the 22nd of March for feeding.

EEG showed diffuse slowness of cerebral activity and diffuse attenuation of the background without no epileptic abnormalities suggesting diffuse encephalopathy resulting from diffuse cortical injury.

During the last week of September, 2021, her family consulted us about her condition as was she still showing no awareness to the environment, showing no significant spontaneous movements. She had poor head control, unable to sit or stand alone, and had a flexed posture (Figure-1). She was on Keppra, diazepam, and baclofen 30 mg daily. She was still having tracheotomy, and was fed through gastrostomy tube.



Figure 1A: She had poor head control, unable to sit or stand alone, and had a flexed posture



Figure 1B: She had poor head control, unable to sit or stand alone, and had a flexed posture

Literature review suggested the possible usefulness of the use multi-factorial therapies including cerebrolysin, citicoline, piracetam, and pyritinol, and based on our extensive clinical experience [1-15], we suggested an initial one-month therapeutic course.

The initial therapeutic course primarily aimed at repairing brain which can result in improved brain function that can be manifested early by improvement in head control, and sometimes may result an early cognitive improvement which can be associated with initiation of speech development and a better understanding of simple speech.

Our suggested evidence-based treatment included cerebrolysin 2.5 ml given by intramuscular injection every other day during the morning hours (15 doses over one month), and oral citicoline syrup 3 ml (300mg) daily in the morning.

Discussion

Cerebral palsy is a heterogeneous disorder that can cause a lifelong disability that is associated with a non-progressive damage in the brain. It is commonly caused by antenatal, perinatal, early postnatal and neonatal

conditions. However, post-neonatal cases of acquired cerebral palsy have also been reported, and were commonly caused by infection [1-10].

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 [16].

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 [17].

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 [18].

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 [19].

Reid and colleagues (2006) from Australia reported that 10.7% of 339 cases of cerebral palsy had post-neonatal acquired condition caused by infection, traumatic head injury, hypoxia, acute encephalopathies, and cerebrovascular accidents [20].

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 [21].

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 neuropeptides (Brain-derived neurotrophic factor, glial cell line-derived neurotrophic factor, nerve growth factor, ciliary neurotrophic factor [22]. Cerebrolysin has been used safely with benefit in a variety of neuro-psychiatric disorders including idiopathic mental retardation [23, 24], cerebral palsy [25, 26], myelomeningocele [27], pediatric juvenile spinal muscular atrophy [28, 29], pediatric Charcot Marie Tooth disease [30, 31], kernicterus [32, 33], agenesis of corpus callosum with colpocephaly [34, 35].

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 [36-37].

Conclusion

In this paper, the rare occurrence of severe post-infantile cerebral palsy is described. Emphasis is made on the possibility of using evidence-based multi-factorial therapies in cerebral palsy.

Acknowledgement

The author would like to express his gratitude for the family of the patients who willingly accepted publishing the photo of their child.

Conflict Of Interest: None.

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DOI: [10.31579/2690-1919/222](https://doi.org/10.31579/2690-1919/222)

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