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Research article

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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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-neonatally acquired condition caused by infection, traumatic head injury a, 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 neuro-peptides (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.

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Conflict of interest: None.

References

1. Al Mosawi AJ. (2008). Dramatic effect of nandrolone decanoate on motor development in cerebral palsy. Arch Dis Child. 93(11):338-339.
2. Al Mosawi AJ. (2017). A novel therapeutic approach for the treatment of cerebral palsy (Ed). LAP Lambert Academic Publishing, Germany.
3. Al Mosawi AJ. (2017). A novel therapeutic approach for the treatment of brain atrophy (Ed). LAP Lambert Academic Publishing, Germany.
4. Al Mosawi AJ. (2019). New therapies for the treatment of spastic cerebral palsy (Ed). LAP Lambert Academic Publishing, Germany.
5. Al-Mosawi AJ. (2019). New therapies for the treatment of spastic cerebral palsy. Med J Clin Trials Case Stud. 3(2): 000209.
6. Al-Mosawi AJ. (2019). The pattern of cerebral palsy in Iraqi children. Med Life Clin. 1(1):1001.
7. Al-Mosawi AJ. (2019). The pattern of cerebral palsy in Iraqi children. 1st ed., Saarbrücken; LAP Lambert Academic Publishing.
8. Al-Mosawi AJ. (2020). New Therapies for the treatment of ataxic cerebral palsy caused by kernicterus. EC Clinical and Medical Case Reports. 3(4):26-31.
9. Al-Mosawi AJ. (2020). The experience with the use of nandrolone decanoate and pyritinol in children with cerebral palsy. Open Access Journal of Biogeneric Science and Research. 2(3):1-3.
10. Al-Mosawi AJ. (2020). Cerebral palsy: A unique illustrated experience. Medico Research Chronicles. 7(4):217-239.
11. Al-Mosawi AJ. (2021). Congenital Externally Communicating Porencephaly Presenting as Hemiplegic Cerebral Palsy: Imaging Study of a Rare Condition. SunKrist Journal of Neonatology and Pediatrics. 3 (1):1-4.
12. Al-Mosawi AJ. (2021). The early treatment of a boy from Virginia with ataxic cerebral palsy. Journal of Pediatrics and Child Health. 2 (4): 1-5.
13. Al-Mosawi AJ. (2020). Cerebral palsy: An illustrated ground-breaking experience. Scholar's press.
14. Al-Mosawi AJ. (2020). Mózgowe porażenie dziecięce: Ilustrowane przełomowe doświadczenie: Medycyna oparta na dowodach (Polish Edition). Wydawnictwo Nasza Wiedza.
15. Al-Mosawi AJ. (2020). Cerebrale parese: Een geïllustreerde baanbrekende ervaring: Bewijsgerichte geneeskunde (Dutch Edition) .Uitgeverij Onze Kennis.

16. Blair E, Stanley FJ. (1982). An epidemiological study of cerebral palsy in Western Australia, 1956-1975. III: Postnatal aetiology. Dev Med Child Neurol. 24(5):575-585.
17. Arens LJ, Molteno CD. (1989). A comparative study of postnatally-acquired cerebral palsy in Cape Town. Dev Med Child Neurol. 31(2):246-254.
18. Murphy CC, Yeargin-Allsopp M, Decouflé P, Drews CD. (1993). Prevalence of cerebral palsy among ten-year-old children in metropolitan Atlanta, 1985 through 1987. J Pediatr. 123(5):13-20.
19. Cans C, McManus V, Crowley M, Guillem P, Platt MJ, Johnson A, Arnaud C. (2004). Surveillance of Cerebral Palsy in Europe Collaborative Group. Cerebral palsy of post-neonatal origin: characteristics and risk factors. Paediatr Perinat Epidemiol. 18(3):214-220.
20. Reid SM, Lanigan A, Reddihough DS. (2006). Post-neonatally acquired cerebral palsy in Victoria, Australia, 1970-1999. J Paediatr Child Health. 42(10):606-611.
21. Salih K. (2020). Pattern of cerebral palsy among Sudanese children less than 15 years of age. Cureus. 12(3):7232.
22. Al-Mosawi AJ. (2020). Clinical uses of Cerebrolysin in Pediatric Neuropsychiatry. Science World Journal of Pharmaceutical Sciences. 1(1):1-4.
23. Al-Mosawi AJ. (2020). A Unique experience with mental and developmental retardation: Innovative Medical therapies for idiopathic mental retardation. EC Clinical and Medical Case Reports. 3(5):42-54.
24. Al-Mosawi AJ. (2020). Treatment of a Boy with Idiopathic Mental Retardation: From Uneducable to Educable. Progressing Aspects in Pediatrics and Neonatology. 2(5): 197-202.
25. Al-Mosawi AJ. (2019). New Therapies for the treatment of spastic cerebral palsy. Medical Journal of Clinical Trials & Case Studies. 3(2):1-9.
26. Al-Mosawi AJ. (2020). New Therapies for the treatment of ataxic cerebral palsy caused by kernicterus. EC Clinical and Medical Case Reports. 3(4): 26-31.
27. Al-Mosawi AJ. (2019). New medical therapies for the treatment of myelomeningocele. Surgical Medicine Open Access Journal. 2(4):1-4.
28. Al-Mosawi AJ. (2018). A novel therapy for pediatric juvenile spinal muscular atrophy.1st ed., Saarbrücken; LAP Lambert Academic Publishing.
29. Al-Mosawi AJ. (2020). The use of cerebrolysin in pediatric Wohlfart Kugelberg Welander syndrome. MOJ Clinical & Medical Case Reports. 10(1):20-30.
30. Al-Mosawi AJ. (2018). A novel therapy for pediatric Charcot Marie Tooth disease. 1st ed., Saarbrücken; LAP Lambert Academic Publishing.
31. Al-Mosawi AJ. (2020). The use of Cerebrolysin in Pediatric Charcot Marie Tooth Disease. Journal of neurological research and therapy. 3(2):17-21.
32. Al-Mosawi AJ. (2019). The novel use of cerebrolysin and citicoline in the treatment of kernicterus. Online Journal of Neurology and Brain Disorders. 3(1): 208-212.
33. Al-Mosawi AJ. (2019). The Use of Intramuscular Cerebrolysin and Citicoline in the Treatment of Kernicterus. SunKrist Journal of Neonatology and Pediatrics. 1(1):1-5.
34. Al-Mosawi AJ. (2019). Agenesis of corpus callosum with colpocephaly: A novel therapy. 1st ed., Saarbrücken; LAP Lambert Academic Publishing.
35. Al-Mosawi AJ. (2020). The use of piracetam and cerebrolysin in the treatment of agenesis of corpus callosum with colpocephaly. EC clinical and medical case reports. 3(1): 01-05.
36. Al-Mosawi AJ. (2019). Citicoline research progress. 1st ed., Saarbrücken; LAP Lambert Academic Publishing.
37. Al-Mosawi AJ. (2019). The Use of Citicoline in Pediatric Neurology and Pediatric Psychiatry. Austin Pediatrics. 6(1):1071-1072.

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