
CASE REPORT

An Indian boy with post-infantile acquired cerebral palsy caused by submersion injury: A rare etiology and a therapeutic challenge

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ABSTRACT

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, early postnatal and neonatal conditions. However, post-neonatal cases of acquired cerebral palsy have also been reported, and were commonly caused by infection.

CASE PRESENTATION: The father of an Indian boy, who developed severe cerebral palsy caused by submersion injury before about three months, consulted us during December, 2021 about the possible therapies for his 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 21 months, a previously healthy boy who was living in Houston, Texas, was involved in a near fatal drowning accident. The boy was hospitalized, and severe global brain damage resulted in no vision, hearing, and motor skills. The

doctors in Houston, Texas, USA told the father that his son won't be able to ever get any motor skills back, and will remain in vegetative state factorial therapies including cerebrolysin, citicoline, piracetam.

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.

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.

Key Words: Acquired post-infantile; Cerebral palsy; Submersion injury; 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].

CASE PRESENTATION

The father of an Indian boy, who developed severe cerebral palsy caused by submersion injury before about three months, consulted us during December, 2021 about the possible therapies for his 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 21 months (September, 2021), a previously healthy boy (Figure 1) who was living in Houston, Texas, was involved in a near fatal drowning accident. The boy was hospitalized (Figure 2), and severe global brain damage resulted in no vision, hearing, and motor skills. The doctors in Houston, Texas, USA told the father that his son won't be able to ever get any motor skills back, and will remain in vegetative state.

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Figure 1) The boy was health until the age of 21 months when he experienced a near fatal drowning accident



Figure 2) The boy was hospitalized after a near fatal drowning accident

The boy was hospitalized for one and half month ago and was receiving hyperbaric oxygen therapy at 1.2 atm pressure. He has completed 23 dives out of 40. After discharge from hospital he was responding to painful stimuli.

On the 28th of September, 2021, MRI showed extensive and relatively symmetric regions of gliosis and volume loss especially involving the basal ganglia and thalamus and brainstem and occipital cortex. There was also global atrophy of the cerebral hemispheres and cerebellum, moderately extensive cystic encephalomalacia in the bilateral globus pallidus medial aspect of the putamen bilaterally and with volume loss, and T2 hyperintensity in the bilateral thalamus and seen in the bilateral midbrain and in the bilateral pons in the bilateral cerebellar hemispheres.

In addition, there was T2 hyperintensity and volume loss within cortex especially in the bilateral occipital lobes medially and diffuse volume loss of cerebral cortex throughout some volume loss of cerebral white matter. This is associated with enlargement of lateral ventricles and third ventricle and fourth ventricle. There was no shift of midline. No subdural collections.

Flow-voids in major vessels at base of brain were generally normal. The A1 segment of the right anterior cerebral artery is hypoplastic, a normal variant of the circle of Willis. Optic nerves appear normal. Globes were normal. There was extensive opacification of paranasal sinuses and maxillary sinuses and ethmoid air cells are also opacification mastoid air cells middle ears.

Three months after the accident, at about the age of two years (Figure 3), the boy had poor spontaneous movements and was not producing any voice. He was receiving Keppra 100 mg three times daily, Baclofen 10 mg three times daily, and Clonazepam 0.1 mg three times daily.

The addition of oral citicoline based on the evidence provided by our publications was associated with the appearance of some purposeful movement his hands.

He was still not tolerating feeding with naso-gastric tube, and remained feeding via G/J tube, and needed suction of his secretions every two hours.



Figure 3) Three months after the accident, at about the age of three years, the boy had poor spontaneous movement

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 [1-15].

Blair and Stanley 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 [16]. 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.

Arens and Molteno 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. 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 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 [19]. They suggested that children with cerebral palsy of post-neonatal origin had a more severe functional pattern than non-post-neonatal cerebral palsy children.

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

The work of Salih (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, cerebral palsy, myelomeningocele, pediatric juvenile spinal muscular atrophy, pediatric Charcot Marie Tooth disease, kernicterus, and agenesis of corpus callosum with colpocephaly [23-31].

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.

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.

DECLARATIONS

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