Introduction

Cerebral palsy (CP) is a non-progressive neurological disorder that results from brain damage caused before birth or during the first two or three years of life. CP is the most common cause of motor impairment in children. It is a common disorder, with an estimated prevalence of two in 1000 population. ***(Johnson, 2002; Sellier et al., 2010).***

Acquired cases in the postnatal period are usually related to central nervous system infection, trauma, strokes, and severe hypoxic events such as near drowning. Genetic disorders and acquired insults follow a pattern of selective vulnerability during early brain development. For example, the neonatal neuro-pathological correlates of hypoxic–ischemic encephalopathy include specific and well-known patterns of brain injury ***(Fatemi et al., 2009)***.

The incidence of CP has varied in different series according to criteria of selection, time and community studied. A figure of between 1 and 3 cases per 1,000 live births has been *quoted* ***(Nettina, 2001; Reddihough and Collins, 2003).***

Upper motor lesion produces muscle spasticity which increases the resistance against passive movements. Spasticity disturbs walking and functional abilities of patients ***(Feng and Mak, 1997).***

Spasticity is characterized by the increase in tendon reflex and tonic stretch reflex because of the hyper-excitability of the stretch reflex after upper motor neuron lesion ***(Brunstrom et al., 2001).***

Different methods are used to evaluate spasticity. These include subjective methods such as passive goniometric measurement and clinical ratio scales as Modified Ashworth Scale and objective methods such as electro-physiologic tests Hoffman (H) reflex and H/M ratios ***(Moore et al., 1998).***

There is a general agreement that spasticity treatment is important ***(Dones et al., 2006).***Various treatments have been recommended to reduce spasticity, including surgical, medical and physiotherapy techniques ***(Albright et al, 2003).***

Methods such as drug therapy, chemical nerve block or neurosurgical treatments may reduce spasticity but may cause muscle weakness or paralysis ***(Carmick et al, 1993).***

Constipation, a common dysmotility disorder of the gut in children with CP, is often overlooked. More than half of the children with severe generalized CP are constipated ***(Ravelli. and Milla. 1998).*** The high incidence of the dysmotility disorders emphasizes the defective integration and modulation of information in the brain–gut axis in CP ***(Veugelers et al., 2010)***.

Cerebral palsy has been shown to be associated with a high rate of chronic constipation. An article by ***Veugelers et al.,*** estimates an outpatient incidence as high as 74% in patients with

CP, and there appears to be a neural component to the observed colonic dysmotility ***(Johanson et al., 1992)***. In a study by ***Johanson et al.,*** neurological disease causing damage to the central nervous system was identified as an important independent risk factor ***(Bongers et al., 2009)***.

Aim of the Work

To investigate the therapeutic and adverse effects of oral magnesium sulfate therapy on spasticity and constipation in infants and children with spastic cerebral palsy.

Cerebral Palsy

Cerebral palsy (CP) is the most common chronic disability of childhood today. It is universal and it occurs all around the world. In developed nations, the incidence is about 1 to 2 per 1000 births ***(Nadire et al., 2011).***

Cerebral palsy (CP) describes a group of disorders of the development of movement and posture, causing activity limitations, which are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of CP are often accompanied by disturbances of sensation, cognition, perception, behavior and/or seizure disorders and by secondary musculoskeletal problems ***(Rosenbaum et al., 2011).***

Scientific consensus still holds that CP is neither genetic nor a 'condition', and it is also understood that the vast majority of cases are congenital, coming at or about the time of birth, and/or are diagnosed at a very young age rather than during adolescence or adulthood ***(Beukelman and Mirenda, 2009)***.

It can be defined as a central motor dysfunction affecting muscle tone, posture and movement resulting from permanent, non-progressive defect or lesion of the immature brain ***(Beukelman and Mirenda, 2009)***.

Cerebral palsy's nature as a broad category means it is defined mostly via several different subtypes, especially the type

featuring spasticity, and also mixtures of those subtypes. It is caused by damage to the motor control centers of the developing brain and can occur during pregnancy, during childbirth or after birth up to about age three ***(Rosenbaum et al., 2007)***.

Resulting limits in movement and posture cause activity limitation and are often accompanied by disturbances of sensation, depth perception, and other sight-based perceptual problems, communication ability; impairments can also be found in cognition, and epilepsy is found in about one-third of cases. CP, no matter what the type, is often accompanied by secondary musculoskeletal problems that arise as a result of the underlying disorder ***(Rosenbaum et al., 2007)***.

Historical Background:

CP, formerly known as "Cerebral Paralysis," was first identified by English surgeon *William little in 1860*. Little raised the possibility of asphyxia during birth as a chief cause of the disorder. It was not until 1897 that *Sigmund Freud*, then a neurologist, suggested that a difficult birth was not the cause but rather only a symptom of other effects on fetal development ***(Brindle, 2002)***.

Research conducted during the 1980s by the National Institute of Neurological Disorders and Stroke (NINDS) suggested that only a small number of cases of CP are caused by lack of oxygen during birth ***(Brindle, 2002)***.

Epidemiology

In the industrialized world, the prevalence of cerebral palsy is about 2 per 1000 live births. The incidence is higher in males than in females; the Surveillance of Cerebral Palsy in Europe (SCPE) reports M: F ratio of 1.33:1. Variances in reported rates of incidence or prevalence across different geographical areas in industrialized countries are thought to be caused primarily by discrepancies in the criteria used for inclusion and exclusion. When such discrepancies are taken into account in comparing two or more registers of patients with cerebral palsy (for example, the extent to which children with mild cerebral palsy are included), the prevalence rates converge toward the average rate of 2:1000 ***(Smith et al., 2009)***.

In the United States, approximately 10,000 infants and babies are diagnosed with CP each year, and 1200-1500 are diagnosed at preschool age ***(United Cerebral Palsy Research and Education Foundation, 2007)***.

Overall, advances in care of pregnant mothers and their babies have not resulted in a noticeable decrease in CP. This is generally attributed to medical advances in areas related to the care of premature babies (which results in a greater survival rate) ***(Hirtz et al., 2007)***.

Only the introduction of quality medical care to locations with less-than-adequate medical care has shown any decreases. The incidence of CP increases with premature or very low-weight

babies regardless of the quality of care. Prevalence of cerebral palsy is best calculated around the school entry age of about six years, the prevalence in the US is estimated to be 2.4 out of 1000 children ***(Hirtz et al., 2007)***.

Risk Factors

A multitude of risk factors both environmental and genetic has been associated with the development of CP.

Table (1): Cerebral palsy risk factors:

Prenatal risk factors:

Prenatal risk factors: include hyperemesis gravidarum, pregnancy-induced hypertension, threatened fetal loss, placenta previa, abruption placentae, teratogenic drugs, intrauterine bacterial and viral infections, maternal malnutrition and family history of CP.

Natal risk factors:

Natal risk factors: include breech delivery, multiple gestation, asphyxia, low Apgar score and especially prematurity and low birth weight.

Postnatal risk factors:

Postnatal risk factors: include head trauma, seizures, hyper-bilirubinemia, intracranial infections, toxic encephalopathies and cerebral and interventricular hemorrhages.

Despite this extensive list, 17-60% of infants with CP have experienced no recognizable adverse event. Although risk factors

analysis is not a very specific or sensitive predictor of CP, risk factors should not be ignored. It is important to take a careful history of prenatal, natal and postnatal events ***(Jacobsson and Hagberg, 2004)***.

Other risk factors associated with an increased risk of CP include patent ductus arteriosus, hypotension, blood transfusion, prolonged ventilation, pneumothorax, sepsis, hyponatremia and total parenteral nutrition. Seizures were associated with an increased risk of CP as were parenchymal damage and an appreciable ventricular dilatation ***(Stelmach et al., 2005)***.

Pathology

The site of lesion and the type of disability varies with the gestational age. In preterm infants, the injury usually involves the white matter and the motor fibers of the lower extremities; thus injured preterm infants generally have a spastic diplegia, primarily of the legs, with normal or near-normal cognitive development because the gray matter of the cortex is not injured. This injury in term infants, usually from a hypoxic ischemic insult such as perinatal asphyxia, usually results in a parasagittal cortical lesions and leads to involvement of the upper extremities, face and tongue with impairment of speech ***(Stelmach et al., 2005)***.

Classification

Table (2): Cerebral palsy types and subtypes.

Type	Subtype
Spastic	***Diplegia:*** 30% - 40% of spastic CP; 50% were born preterm
	Hemiplegia: 20% - 30% of spastic CP; associated with strokes, vascular malformations.
	Quadriplegia: 10% - 15% of spastic CP; associated with severe asphyxia in all infants
	Monoplegia/Triplegia
Nonspastic	***Dyskinetic:*** Damage to basal ganglia or thalamus (deep motor neurons)
	Ataxic: Damage to neurons in cerebellum**.**

(Pueyo et al., 2003).

Cerebral Palsy (CP) is divided into four major classifications to describe different movement impairments. These classifications also reflect the areas of the brain that are damaged. The four major classifications are: spastic, ataxic, athetoid/dyskinetic and mixed ***(Pennington et al., 2004)***.

Cerebral palsy has a complex and multifactorial etiology. Approximately 5% - 10% of cases can be ascribed to perinatal hypoxia ***(Stevenson et al., 2010)***.But the vast majority of cases are caused by the interplay of several risk factors and antenatal, perinatal and neonatal events. The strongest risk factors include prematurity and low birth weight.

Clinical characteristics:

Clinical observations that rise suspicious to CP diagnosis

1. Delayed motor milestones:

• Not rolling by 6 months.

• Fisting after 5 months of age.

• Not sitting with support by 8 months.

• Not walking by 15-18 months.

• Discrepancies between intellectual and motor development.

2. Persistent or evolving increased or decreased muscle tone:

• Head lag beyond 6 months of age.

• Poor trunk control and balance.

• Opisthotonic posturing and extensor thrusting.

• Dystonia.

• Early rolling or standing (especially in high risk infant: rolling is achieved involuntarily by a reflexive log roll or arching).

• Toe walking/scissoring.

• Abnormal motor or gait patterns.

3. Focal abnormalities of movement posture and tone:

• Declaring handedness prior to 18 months.

• Differences in functional ability of left/right extremities.

• Clonus persisting past 12 months.

4. Persistence of primitive reflexes.

Behavioral:

• Irritability.

• Easily startle with exaggerated Moro reflex.

• Excessive crying.

• Jittery.

• Sleeping difficulties.

Physical:

• Decreased rate of head growth.

• Poor suck.

• Delayed feeding milestones.

• Poor weight gain/failure to thrive.

(Pueyo et al., 2003).

Data

The physician should always check the head circumference and look for minor dysplasias of the skull, eyes, mouth, ears, genitals etc. Although history and clinical examination is an essential aid not only in diagnostic approach of the child with CP but in every step while practicing medicine ***(Panteliadis and Strassburg et al., 2009)***.

The physical signs of CP may include poor head support after 3 months of age, stiff or rigid arms or legs, pushing away or

arching of the back, floppy body posture, inability to sit-up without support by 8 months of age, delayed or absent motor milestones and tongue protrusion after 6 months of age ***(Palisano et al., 2003)***.

Behavioral signs may include irritability, excessive weak cry, failure to smile by 3 months of age, feeding difficulties, poor sucking, persistent gagging or choking when being fed, lack of interest in environment and prolonged sleeping patterns ***(Rollant et al., 2011)***.

Diagnosis:

The diagnosis of cerebral palsy has historically rested on the patient's history and physical examination. Once diagnosed with cerebral palsy, further diagnostic tests are optional. The American Academy of Neurology published an article in 2004 reviewing the literature and evidence available on CT and MRI imaging. They suggested that neuroimaging with CT or MRI is warranted when the etiology of a patient's cerebral palsy has not been established; an MRI is preferred over CT due to diagnostic yield and safety ***(Ashwal et al., 2004)***.

When abnormal, the neuroimaging study can suggest the timing of the initial damage. The CT or MRI is also capable of revealing treatable conditions, such as hydrocephalus, porencephaly, arteriovenous malformation, subdural hematomas and hygromas, and a vermin tumor (which a few studies suggest are present 5 to 22%). Furthermore, an abnormal neuroimaging study indicates a high likelihood of associated conditions, such as epilepsy and mental retardation ***(Ashwal et al., 2004)***.

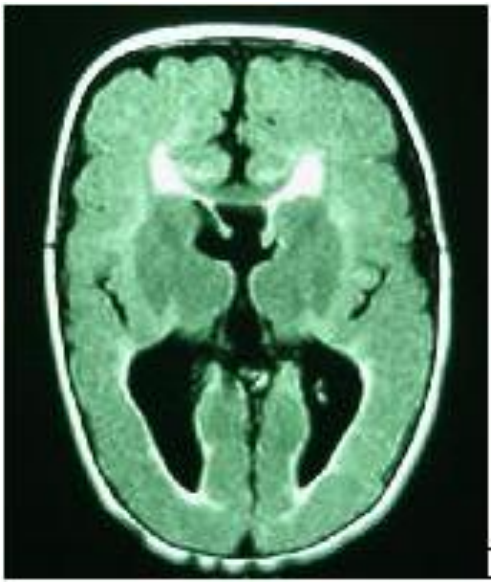

Figure (1): Magnetic Resonance Image (MRI) of a one-year-old boy who was born at gestational age 27 week.

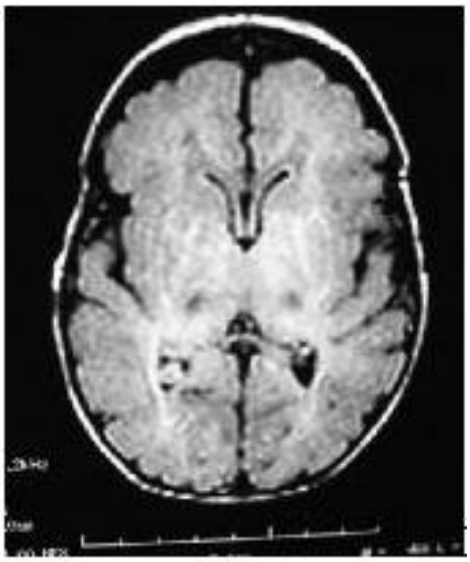

Figure (2): Magnetic Resonance Image (MRI) of a 9-day-old girl who was born at full term and had a perinatal hypoxic-ischemic event

The diagnosis of cerebral palsy can sometimes be made shortly after birth, but is often postponed until the child is 18-24 months of age, in order to evaluate the functional status and the progression or regression of the symptoms ***(Taub et al., 2011)***.

Management:

Treatment for cerebral palsy is a lifelong multi-dimensional process focused on the maintenance of associated conditions. In order to be diagnosed with cerebral palsy the damage that occurred to the brain must be non-progressive and not disease-like in nature. The manifestation of that damage will change as the brain and body develop, but the actual damage to the brain will not increase ***(Pennington et al., 2004)***.

Treatment in the life of cerebral palsy is the constant focus on preventing the damage in the brain from prohibiting healthy development on all levels. ***(Pennington et al., 2004)***.

Various forms of therapy are available to people living with cerebral palsy as well as caregivers and parents caring for someone with this disability. They can all be useful at all stages of this disability and are vital in a person with cerebral palsy's ability to function and live more effectively ***(Pennington et al., 2004)***.

In general, the earlier treatment begins the better chance children have of overcoming developmental disabilities or learning new ways to accomplish the tasks that challenge them. The earliest proven intervention occurs during the infant's