

MRI Brain Study of Lateral Ventricles in Children with Cerebral Palsy

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ABSTRACT

Introduction: Cerebral Palsy (CP) is not a disease but a group of conditions characterised by spasticity, seizures and paralysis along with disorganised motor skills. It occurs following brain injuries during foetal development or during childbirth or even shortly thereafter. Motor deficit is the usual presentation in a CP patient along with delayed developmental milestones that make medical evaluation a must for the child. By proper history, progressive or degenerative disease can be excluded. This combined with a neurological examination, the diagnosis of CP can be made by establishing that the motor deficit of the patient is due to a cerebral cause.

Aim: To evaluate any connection between lateral ventricular

volume and severity of motor deficit, presence of seizures along with cognitive impairment

Materials and Methods: MRI Brain images of 20 patients with cerebral palsy were reviewed for lateral ventricular volume and compared with the degree of motor deficit, cognitive impairment and presence of seizures.

Results: There was a direct correlation between the degree of motor deficit and cognitive impairment with the lateral ventricular volume. No such relationship was found between the presence or absence of seizures with the lateral ventricular volumes.

Conclusion: Lateral ventricular volume measurements can be used as quantitative markers of the severity of clinical impairment.

Keywords: Disability, Lateral ventricular volume, Neurological disorder, Seizures

INTRODUCTION

Cerebral palsy or CP is not a disease but a group of conditions characterized by spasticity, seizures and paralysis along with disorganized motor skills. It occurs following brain injuries during fetal development or during childbirth or even shortly thereafter. CP is a permanent disability which remains static and presents to the clinician as a chronic non-progressive neurological disorder. Till date, there is no cure for this condition but with constant physiotherapy, some motor functions can be maintained. The incidence of CP is about 1-4 in 1000 live births [1].

CP develops during brain development. Most cases (80%) occur during the first month of life. It is a non-progressive disorder; once damage to the brain occurs, no additional damage occurs as a result of this condition.

Motor deficit is the usual presentation in a cerebral palsy patient along with delayed developmental milestones that make medical evaluation a must for the child. By proper history, progressive or degenerative disease can be excluded. This combined with a neurological examination, the diagnosis

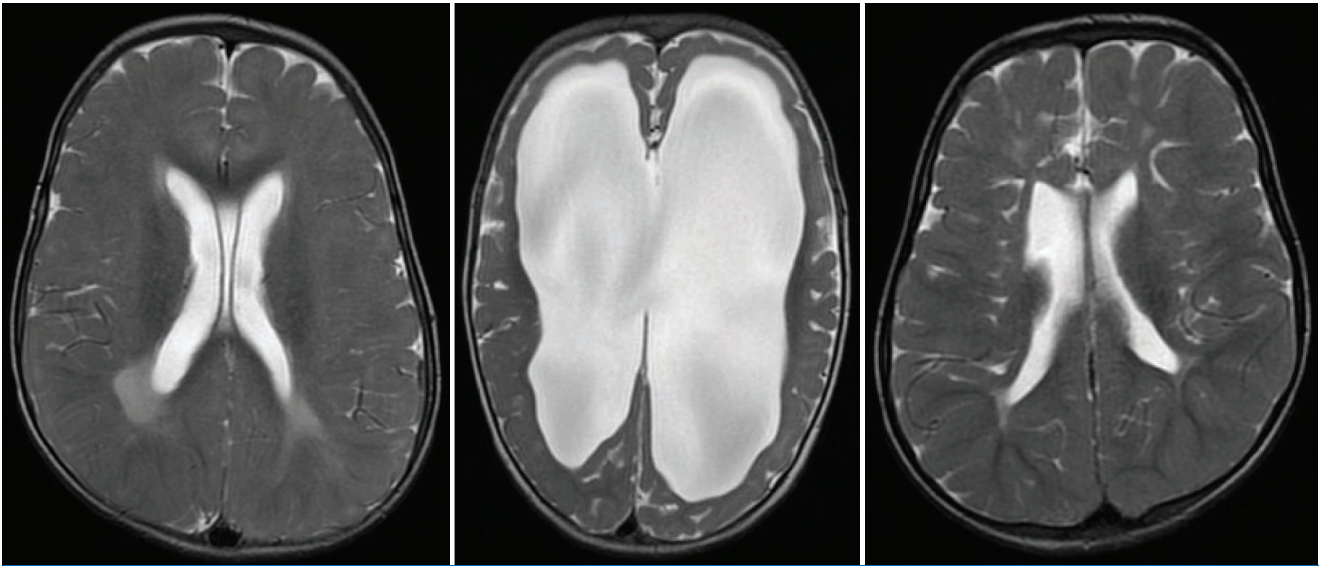
of cerebral palsy can be made by establishing that the motor deficit of the patient is due to a cerebral cause. For example MRI brain in a 5 years old child with spasticity and mild clumsiness, no cognitive impairment and no seizure disorder revealed periventricular leukomalacia [Table/Fig-1].

According to Nelson K et al., CP can be attributed to a lesion or a defect in developing brain, which may occur in either prenatal, perinatal or postnatal period [2].

Many MRI brain studies have been reported in children with cerebral palsy. Truwit C et al., did a case study on 40 patients and reported that cerebral palsy was often the result of variable prenatal factors on 29 term infants [3].

Krageloh-Mann I et al., found a predominantly prenatal aetiology in a series of 56 patients who had bilateral spastic cerebral palsy [4]. For example, T2 weighted MR image in a 7-month-old boy with spastic quadriplegia, marked motor impairment and seizure disorder reveals dilated lateral ventricles with irregular outline [Table/Fig-2].

Steinlin M et al., found prenatal cause in 20-40% of cases out of 33 children with congenital hemiplegia [5].



[Table/Fig-1]: Periventricular leukomalacia in a 5-year-old boy with spasticity and mild clumsiness, no cognitive impairment and no seizure disorder. **[Table/Fig-2]:** T2 weighted image in a 7-month-old boy with spastic quadriplegia, marked motor impairment and seizure disorder reveals dilated lateral ventricles with irregular outline. **[Table/Fig-3]:** T2 weighted MR image in a 16-month boy with spastic quadriplegia, moderate cognitive impairment and no seizure disorder reveals irregular lateral ventricles.

This study would evaluate whether there is any connection between lateral ventricular volume measurement and severity of motor deficit, presence of seizures along with cognitive impairment.

Our null hypothesis were as follows –

- There is no change in lateral ventricular volumes with differences in the severity of motor deficit and cognitive impairment.
- There is no difference between lateral ventricular volumes in affected children with or without seizure disorder.

MATERIALS AND METHODS

This type of ethical committee approved analytical study was conducted in the Department of Radiodiagnosis at AVBRH, Jawaharlal Nehru Medical College, Sawangi, Wardha, Maharashtra, India for a duration of one year from October 2015 to September 2016. Total 20 children with ages ranging from 6 months to 12 years, admitted with the clinical diagnosis of cerebral palsy in Department of Pediatrics were reviewed for sex, gestational age at birth, severity and type of motor and cognitive impairments, and presence of a seizure disorder.

Children with cerebral palsy were divided into

- Three groups depending on the severity of motor impairment – mild (clumsiness with spasticity), moderate (diplegia) or marked (quadriplegia);
- Four groups depending upon the degree of cognitive impairment (score ranges for the Bayley scale of infant development, Stanford-Binet Intelligence scale [1] – no

cognitive impairment, mild cognitive impairment, moderate cognitive impairment and marked cognitive impairment and

- Two groups based on the presence or absence of seizure disorder.

All children included in the study underwent a standard paediatric brain MRI examination performed with a 1.5T super conducting magnet with the following parameters – axial, sagittal and transverse spin echo [550/20 (repetition time msec/echo time msec)] and transverse T2-weighted double-echo SE imaging. Sections were of 5 mm thickness.

Lateral ventricles of each patient were studied and compared with the degree of motor and cognitive impairments and seizure disorder. For example, T2 weighted MR image in a 16-month boy with spastic quadriplegia, moderate cognitive impairment and no seizure disorder reveals irregular lateral ventricles [Table/Fig-3].

Ventricular abnormalities included irregular ventricular contour, rounded trigones and ventricular enlargement and hydrocephaly.

STATISTICAL ANALYSIS

SPSS Version 17 was used for both database entry and statistical analysis. The baseline characteristics re-recorded. Simple descriptive statistics was used throughout. Possible statistical associations between categorical variables were evaluated using Pearson's Chi-square analysis. When statistical significance was attained (p -value <0.05), regression and correlation analysis was performed.

RESULTS

MRI Findings in Motor Impairment Group

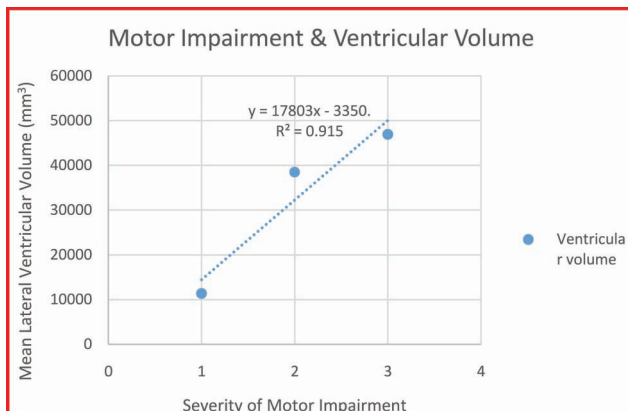
Seven children had mild motor impairment, 6 had moderate motor impairment and 7 had marked motor impairment. The means and SDs of lateral ventricular volumes for this group is shown in the [Table/Fig-4].

Severity of Motor Impairment Group	Mean Lateral Ventricular Volume (mm ³ ± SD)
Mild	11,336.43 ± 3,210.14
Moderate	38,487.26 ± 14,700.28
Marked	46,942.11 ± 19,774.55

[Table/Fig-4]: Observed results of mean lateral ventricular volume for different severities of motor impairment. Stereological volume measurements were made by the Cavalier principle. To accurately calculate the volume, different sections were taken equidistant, parallel and serially.

The two-tailed p-value for this data is less than 0.0001.

Under the hypothesis, there is a direct relationship between the severity of motor impairment and the mean lateral ventricular volume. To test this hypothesis, the data was compared with a model which considered mean ventricular volume to be directly proportional to severity of motor impairment. The observed values were then compared to the expected values and the Chi-square values were calculated. The Chi-square valued help us understand the statistical significance of the results and figure out the two tailed p-value [Table/Fig-5].



[Table/Fig-5]: Graph of mean lateral ventricular volume with respect to the level of motor impairment in patients. The dotted line indicates the expected results whereas the points indicate the actual results. This graph was used to calculate the significance value and the two-tailed p-value (less than 0.0001).

For the above data, the Chi-square value equals 2069.89 with 2 degrees of freedom. The corresponding two-tailed p-value is less than 0.0001. This signifies that the results are very highly statistically significant. One can therefore draw a direct correlation between the severity of motor impairment and the mean lateral ventricular volume.

Cognitive Impairment Group

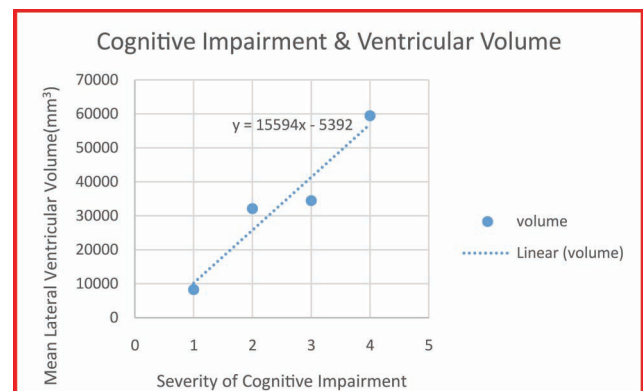
Four children had no cognitive impairment, 5 had mild impairment, 4 had moderate impairment and 7 had marked impairment. The means and SDs of lateral ventricular volumes for this group is shown in the [Table/Fig-6].

Using similar methods as described above for the motor impairment group, the observed data was compared to the expected data.

For the above data, the Chi-square value equals 3167.50 with 3 degrees of freedom. The corresponding two-tailed p-value is less than 0.0001. This signifies that the results are very highly statistically significant. One can therefore draw a direct correlation between the severity of cognitive impairment and the mean lateral ventricular volume [Table/Fig-7].

Severity of Cognitive Impairment Group	Mean Lateral Ventricular Volume (mm ³ ± SD)
None	8,293.76 ± 697.32
Mild	32,116.24 ± 12,558.45
Moderate	34,483.66 ± 12,962.53
Marked	59,485.82 ± 24,592.27

[Table/Fig-6]: Observed results of mean lateral ventricular volume for different severities of cognitive impairment. Stereological volume measurements were made by the Cavalier principle. To accurately calculate the volume, different sections were taken equidistant, parallel and serially. The two-tailed p-value for this data is less than 0.0001.



[Table/Fig-7]: Graph of mean lateral ventricular volume with respect to the level of cognitive impairment in patients. The dotted line indicates the expected results whereas the points indicate the actual results. This graph was used to calculate the significance value and the two-tailed p-value (less than 0.0001).

Significant difference between the lateral ventricular volumes was found between the mild and no cognitive impairment group (p-value less than 0.0001), moderate and no cognitive impairment group (p-value less than 0.0001), marked and no cognitive impairment group (p-value less than 0.0001), mild group and the marked group (p-value less than 0.0001), moderate group and the marked group (p-value less than 0.0001).

Seizure Disorder Group

Out of 20 children, 14 had no history of seizure disorder and 6 had a history of seizure disorder.

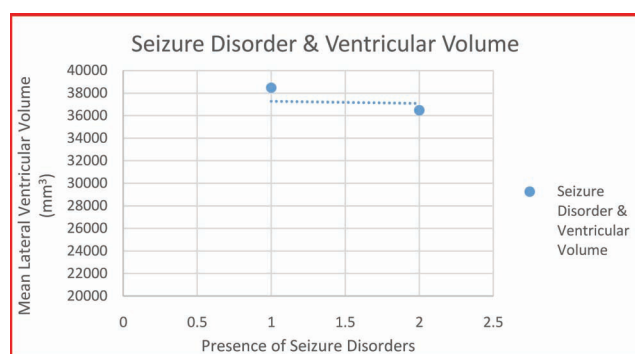
The means and SDs of lateral ventricular volumes for this group is shown in [Table/Fig-8].

To test the hypothesis that presence of seizure disorder does not affect the mean lateral ventricular volume, the observed data was compared to a model where both groups have equal mean lateral ventricular volume.

For the above data, the Chi-square value equals 54.22 with 1 degree of freedom. The corresponding two-tailed p-value is less than 0.0001. This signifies that the results are very highly statistically significant. Therefore, one can safely conclude that there is no link between the presence of seizure disorders and mean lateral ventricular volume [Table/Fig-9].

Seizure Disorder Group	Mean Lateral Ventricular Volume ($\text{mm}^3 \pm \text{SD}$)
Absent	38,485.23 \pm 15,682.58
Present	36,649.74 \pm 14,953.62

[Table/Fig-8]: Observed results of mean lateral ventricular volume based on presence of seizure disorders. Stereological volume measurements were made by the Cavalier principle. To accurately calculate the volume, different sections were taken equidistant, parallel and serially. The two-tailed p-value for this data is less than 0.0001.



[Table/Fig-9]: Graph of mean lateral ventricular volume with respect to the presence of seizure disorders. The dotted line indicates the expected results whereas, the points indicate the actual results. This graph was used to calculate the significance value and the two-tailed p-value (less than 0.0001).

DISCUSSION

Though recent times have seen lot of improvements in the foetal and neonatal care, unfortunately, the prevalence of CP could not be reduced and it remains the most common form of chronic motor disability in children. The most common form of brain injury is PVL which gives rise to various degrees of deficient motor skills, cognitive impairment and seizures in severe cases [6].

In preterm babies, whenever there is oxygen deprivation, it results in decrease in cerebral perfusion. Immature cerebral blood vessels are highly susceptible to lack of oxygen and irreversible damage to these vessels is the final outcome. Also, there is damage to oligodendroglia in the periventricular white matter. Brain injury manifests itself in various degrees of cognitive impairment, spasticity and seizures. Thus, PVL is the most common form of brain injuries in preterm infants [7-9].

In case of PVL, the MRI findings show white matter cavitations and loss along with gliosis but overlying cortex is somewhat unaffected. Secondary manifestations include corpus callosum thinning along with lateral ventricular enlargement and irregularities. In this study, we measured periventricular volume loss by taking lateral ventricular volume, the reason being its anatomic and image post processing issues [10].

Our results revealed that the lateral ventricular volumes increased as the severity of motor or cognitive impairment in children with cerebral palsy increased.

Spastic cerebral palsy in children with PVL is the result of injury to the corticospinal tract that traverses dorso-lateral to the angle of lateral ventricle that preferentially innervates the lower extremities [11]. In severe cases of PVL, involvement of both upper and lower extremities is seen as a result of extensive white matter loss. Our results showed that the quantification of lateral ventricular volumes reasonably reflected the severity of motor impairment and is a good marker for the degree of periventricular white matter loss.

The pattern of cognitive impairment in children with PVL is characterised by greater involvement of visuomotor and perceptual abilities than of verbal abilities [12]. Using quantitative scoring systems Yokoshi K et al., found no correlation between the severity of mental impairment, expressed in terms of full-scale IQ, and the degree of periventricular white matter loss [13]. But in our study, we found a significant correlation between the degree of periventricular white matter loss and the severity of mental impairment. There was an increase in lateral ventricular volumes with the increasing severity of cognitive impairment.

Subtle cortical malformations secondarily cause seizures in children with PVL [14].

Our study showed no significant difference in the ventricular volumes between children with history of seizure disorder to those with no history of seizure disorder.

LIMITATIONS

Potential limitations of our study, were in part, related to limited clinical information available in patient's charts, especially with regard to the type and severity of cognitive impairment. Also, due to non-availability of MR images with a high grey-to-

white matter contrast, we were unable to quantify directly the volume of periventricular white matter.

CONCLUSION

Cerebral palsy is not a disease but a group of conditions characterised by spasticity, seizures and paralysis along with disorganised motor skills. Till date no cure for this condition has been found. Comparative study of the ventricular volume with the severity of motor deficit, presence of seizures along with cognitive impairment allows us to predict the degree of clinical impairment and thus give the required treatment and counselling to the family.

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