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Original article

A magnetic resonance imaging finding in children with cerebral palsy: Symmetrical central tegmental tract hyperintensity

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Abstract

Background: Central tegmental tract is an extrapyramidal tract between red nucleus and inferior olivary nucleus which is located in the tegmentum pontis bilaterally and symmetrically. The etiology of the presence of central tegmental tract hyperintensity on MRI is unclear.

Purpose: In this study our aim is to evaluate the frequency of central tegmental tract lesions in patients with cerebral palsy and control group, as well as to determine whether there is an association between central tegmental tract lesions and cerebral palsy types.

Materials and methods: Clinical and MRI data of 200 patients with cerebral palsy in study group (87 female, 113 male; mean age, 5.81 years; range, 0–16 years) and 258 patients in control group (114 female, 144 male; mean age, 6.28 years; range, 0–16 years) were independently evaluated by two reader for presence of central tegmental tract hyperintensity and other associated abnormalities.

Results: Central tegmental tract hyperintensities on T2WI were detected in 19% of the study group (38/200) and 3.5% of the control group (9/258) ($p < 0.0001$). Among the total of 38 central tegmental tract lesions in study group, the frequency of central tegmental tract hyperintensity was 16% (24/150) in spastic cerebral palsy and 35% (14/40) in dyskinetic cerebral palsy ($p = 0.0131$).

Conclusion: The prevalence of central tegmental tract hyperintensity is higher in patients with cerebral palsy particularly in dyskinetic type. We suggest that there is an increased association of the tegmental lesions with dyskinetic CP. Patients with cerebral palsy and ischemic changes were more likely to have central tegmental tract lesions. According to our results we advocate that an ischemic process may have a role in the etiopathogenesis.

Keywords: Central tegmental tract; Magnetic resonance imaging; Cerebral palsy

1. Introduction

The central tegmental tract (CTT) is an extrapyramidal tract between red nucleus in mesencephalon and inferior olivary nucleus in bulbus that is a part of dentato-rubro-olivary system, called Guillain–Mollaret triangle. Moreover, this tract includes the ascending

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tracts between reticular nuclei and thalamus. CTT, which is located as an area in the mediocentral tegmentum of the pons bilaterally and symmetrically, usually cannot be seen as a recognizable signal on brain magnetic resonance imaging (MRI) after birth because of the early myelination in fetal life [1–3].

CTT hyperintensity on T2-weighted images (T2WI) is an uncommon neuroimaging finding of uncertain etiology. CTT hyperintense lesions are always bilateral and cannot be identified on T1-weighted images. There are only few articles and case reports describing CTT abnormalities in children which could be seen in various conditions including hypoxic-ischemic encephalopathy (HIE), neurodevelopmental and neurometabolic diseases, etc. [1–4]. Aguilera-Albesa et al. suggested that the occurrence of CTT hyperintensity primarily may be an age-related physiological process and the physiological process may be modified by endogenous–exogenous factors [1]. Furthermore, Shioda et al. reported CTT lesions in 25 among the 120 autopsy cases including patients with congenital brain anomalies, neurodegenerative disorders, metabolic disorders, neuromuscular disorders, and postnatal brain disorders [2]. Yoshida et al. reviewed the MR images of 392 children ranging the age from 1 to 6 years, and found that the frequency of CTT hyperintensity was 5.1% ($n = 20/392$). In addition, they reported that the most frequent clinical diagnosis was cerebral palsy ($n = 6$) in those 20 children with CTT hyperintensities [3].

Cerebral palsy (CP) is the most common physical disability and movement disorder in children. It occurs in about 2.1 per 1000 live births [5]. Cerebral palsy is produced by abnormal development or damage to the portions of the fetal or infantile brain that control body movement, muscle control, muscle coordination, muscle tone, reflex, posture and balance. The problem may occur during pregnancy, childbirth, or shortly after birth. CP prevalence increases with prematurity and lower birth weight. Furthermore, the CP rates rise up with the increase in the numbers of the survivors of preterm birth. Extent and distribution determine the clinical subtype of CP and the severity of associated disabilities [5,6]. Cerebral palsy can be classified according to the type of movement disorder: spastic cerebral palsy accounts for approximately 60% of cases and can be subdivided into hemiplegic, diplegic, quadriplegic, and monoplegic types; whereas other forms include dyskinetic, ataxic, and mixed in descending order. In patients with CP, spasticity is mainly due to the damage to the motor cortex whereas; dyskinesia results from damage to the basal ganglia. Diagnosis is based on the child's clinical development over time [5,6].

According to these observations, in this study, our aim is to evaluate the frequency of CTT hyperintensity in patients with CP and compare these with control group, as well as to investigate whether there is an association between CTT hyperintensity and CP types.

2. Materials and methods

2.1. Case identification and data collection

This retrospective study was approved by the committee of our institutional research ethics board. The patients that included in the study were collected from the imaging data bases of our hospital. All studies were performed for clinical indication in line with the local ethics guidelines and standard departmental imaging protocols. Informed consent was obtained from the parents of all individual participants included in the study.

In our institution the pediatric radiologist and the pediatric neurologist worked together collaboratively. Previously, the pediatric neurologists checked and examined the patients. Developmental delay, abnormal growth charts, impaired muscle tone, and abnormal reflexes are the indications of CP. Therefore, there was not any test that definitely confirms CP; we did brain MRI according to the suspicious of the neurologist for the exclusion of the other possible causes. The diagnosis of CP was made by a pediatric neurologist after all the tests were done and ruled out of other similar conditions. Thus, the determination of the subtype of CP in young children was generally difficult and the diagnosis was based on the child's clinical development over time. For this reason, in this study most of the patients got the exact diagnosis of CP and the subtypes after the MRI exams. The period between the MRI exams and the exact diagnosis were shorter than twelve months.

The study director, with 7 years' experience in neuroradiology, retrospectively evaluated the clinical and MRI data of 265 patients who had been followed up with the diagnosis of CP between August 2011 and August 2015, collected from the imaging data bases of our hospital. Sixty-five patients with CP were excluded. The exclusion criteria's were poor imaging due to motion artifacts, operation history and unavailability of clinical and radiological data. Consequently, 200 patients with CP on MRI were included in the study group, in which, 150 (75%) were diagnosed as spastic type CP, whereas other 40 (20%) were dyskinetic, 8 (4%) were ataxic, and 2 (1%) were mixed type CP.

The director also retrospectively evaluated images of randomly selected 320 age and sex matched patients who had undergone brain MRI examination for various reasons without any clinical history for CP in the same period from August 2011 to August 2015 at the same institution. Poor imaging due to motion artifacts were excluded. Among those patients who didn't have any clinical evidence for CP, 258 were included in the control group. The director matched control ($n = 258$) and