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Original Article

Central Tegmental Tract Hyperintensity on Cranial MRI in Pediatric Patients

Pediatrik Hastalarda Kraniyal MRG'de Santral Tegmental Trakt Hiperintensitesi

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ABSTRACT

Objective: The central tegmental tract (CTT) is an extrapyramidal pathway located in the brainstem. CTT hyperintensity (CTTH) is a neuroimaging finding of uncertain etiology, considered either part of the normal maturation process or a finding that can be seen in various neurological conditions. This study aims to investigate its relationship with age and the associated disorders of CTTH observed in the pediatric population.

Method: All brain magnetic resonance imagings (MRIs) performed between July 2023 and November 2025 at a tertiary pediatric hospital were retrospectively reviewed. Eighty-one pediatric patients with CTTH were included in the study. Clinical data, additional MRI findings, and available follow-up scans were examined.

Results: CTTH was detected in 1.4% of all cranial MRIs, while the median age of the cases was 17 months. The most common complaints at admission were seizures (35.8%) and developmental delay (28.4%). Additional MRI findings (most commonly periventricular leukomalacia) were observed in 51.9% of the cases. The age of patients in this group with additional MRI findings was significantly lower than those without (median ages: 15 vs. 23 months). CTTH resolved in 36.7%, and persisted in 63.3% of the patients who underwent follow-up MRI. It was determined that CTTH resolved significantly in older children compared to the persistent cases among younger patients (median: 26 vs. 14 months).

Conclusion: CTTH is a finding that can be seen in pediatric cranial MRI examinations, and its clinical significance should be interpreted by considering the patients' age and associated neurological conditions.

Keywords: Central tegmental tract hyperintensity, cranial MRI, pediatric neuroimaging, cerebral palsy, myelination

ÖZ

Amaç: Santral tegmental trakt (STT), beyin sapında yer alan ekstrapiramidal bir sinir yoludur. STT hiperintensitesi (STTH), etiyolojisi belirsiz bir nörogörüntüleme bulgusu olup, normal matürasyonun bir parçası veya nörolojik hastalıklarda görülebilen bir bulgu olarak değerlendirilmektedir. Bu çalışma, pediatrik popülasyonda görülen STTH'nin yaş ile ilişkisini ve eşlik eden hastalıkları araştırmayı amaçlamaktadır.

Yöntem: Temmuz 2023-Kasım 2025 tarihleri arasında bir üçüncü basamak çocuk hastanesinde çekilen tüm beyin MRG'ler retrospektif olarak tarandı. STTH saptanan 81 pediatrik olgu çalışmaya dahil edildi. Olguların klinik verileri, ek MRG bulguları ve mevcut takip görüntülemeleri analiz edildi.

Bulgular: Kraniyal MRG'lerin %1,4'ünde STTH tespit edildi; olguların ortanca yaşı 17 aydı. En sık başvuru şikayetleri nöbet (%35,8) ve gelişim geriliği (%28,4) olarak saptandı. Olguların %51,9'unda ek MRG bulgusu (en sık periventriküler lökomalazi/asfiksi sekeli) görüldü; bu gruptaki hastaların yaşı ek bulgusu olmayanlara göre anlamlı düzeyde daha düşüktü (medyan: 15 vs. 23 ay). Takip MRG'si yapılanların %36,7'sinde STTH'nin kaybolduğu, %63,3'ünde sebat ettiği görüldü. Bulgunun kaybolduğu olguların, sebat edenlere göre anlamlı şekilde daha büyük yaşta olduğu (medyan: 26 vs. 14 ay) belirlendi.

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Sonuç: STTH, pediatrik kraniyal MRG incelemelerinde görülebilen ve klinik önemi hasta yaşı ile eşlik eden nörolojik durumlar dikkate alınarak yorumlanması gereken bir bulgudur.

Anahtar kelimeler: Santral tegmental trakt hiperintensitesi, kraniyal MRG, pediatrik nörogörüntüleme, serebral palsi, myelinizasyon

INTRODUCTION

Central tegmental tract (CTT) is an extrapyramidal pathway that runs along the posterior brainstem and connects the red nucleus in the mesencephalon to the inferior olivary nucleus in the medulla. On routine brain magnetic resonance imaging (MRI), the CTT is usually not identified as a distinguishable signal. However, in certain cases, it may appear as a symmetrical area of hyperintensity within the pontine tegmentum, particularly on T2-weighted and diffusion-weighted sequences[1,2].

CTT hyperintensity (CTTH) is a relatively rare neuroimaging finding with an unclear etiology. Some studies have suggested that CTTH observed in early childhood may represent a component of the normal myelination process[3]. Kesimal et al.[4] proposed that CTTH may be related to physiological maturation, while also potentially occurring secondary to a transient response of brain tissue to toxic, metabolic, or ischemic injury. In addition, CTTH has been reported in association with various neurological conditions, including cerebral palsy, epilepsy, developmental delay, and intracranial tumors[1,5-7]. Conversely, a more recent study suggested that CTTH may reflect irreversible myelin degeneration[8].

The conflicting literature data indicate that the pathophysiology of CTTH remains incompletely understood. The objectives of this study are to determine the prevalence of CTTH and its relationship with age, to identify associated neurological conditions, and to evaluate the presence of concomitant cranial abnormalities. In addition, we investigated whether CTTH persists on follow-up imaging and examined its association with patient's age.

MATERIALS and METHODS

Patient Selection

This retrospective and descriptive study included a review of all brain scans performed between July 2023 and November 2025 at a tertiary referral pediatric hospital ie. Ethical approval was obtained from the University of Health Sciences Türkiye, Dr. Behçet Uz Children's Diseases and Surgery Training and Research Hospital (decision no.:2026/01-07, date: 15.01.2026). All brain MRI reports were retrospectively screened, and all pediatric patients (0-18 years) in whom CTTH was identified were included in the study. The prevalence of CTTH was calculated. In patients with follow-up MRI scans, the persistence or resolution of CTTH on control MRIs was recorded. Patients in whom CTTH could not be reliably evaluated due to motion artifacts or insufficient image quality (n=3), as well as those whose clinical data could not be accessed through hospital medical records (n=1), were excluded from the study.

Clinical and Demographic Data

Sex and age of the patients at the time of MRI examination were recorded for all patients. Perinatal data were obtained from the hospital information system. Patients with a gestational age of ≥37 weeks were classified as term, and those born at <37 weeks as preterm infants. A history of neonatal intensive care unit (NICU) admission and, if present, the duration of hospitalizastion were recorded. The presence of motor deficits at presentation, Denver Developmental Screening Test results, and electroencephalography (EEG) findings were retrieved from hospital records. EEG findings showing slowing, dysmaturation, epileptiform activity, or other pathological patterns were categorized as "pathological EEG".

The primary presenting complaints and pre-MRI preliminary diagnoses were recorded. In addition, information regarding final diagnoses and confirmed genetic or metabolic disorders, if present, was obtained from hospital records and review of the archival data.

MRI Protocol and Image Analysis

Cranial MRI examinations were performed using a 1.5 Tesla scanner (Philips Ingenia, Philips Healthcare, Best, The Netherlands). CTTH was assessed on diffusion-weighted images (b=0 and 1000 s/mm^2), axial T2-weighted images, and T2-weighted fluid-attenuated inversion recovery sequences. All images were evaluated by a single neuroradiologist with 10 years of experience in pediatric neuroimaging. Imaging parameters of the three sequences used for the assessment of CTTH are provided in Table 1. Additional findings potentially associated with CTTH were evaluated considering all MRI sequences. The routine brain MRI protocol consisted of axial T1W, T2W, T2-FLAIR, diffusion weighted imaging (DWI), sagittal T1W, and coronal T2W sequences.

Hyperintensity along the course of the CTT within the pontine tegmentum on T2-weighted and/or DWI sequences was defined as CTTH (Figure 1). Signal abnormalities and pathological findings in other neural pathways and anatomical regions of the brain were also recorded. For patients with available follow-up MRI scans, sex, age at follow-up MRI, and the interval between the initial and follow-up MRI examinations were calculated.

Statistical Analysis

Statistical analyses were performed using SPSS version 20 software. A two-tailed p value of <0.05 was considered statistically significant.

Statistical comparisons of sex and age of the patients with and without additional MRI findings were conducted using Fisher's exact test for categorical and the Mann-Whitney U test for continuous variables. The interval between the initial and follow-up MRI examination results were compared between patients with persistent and resolved CTTH using the Mann-Whitney U test. Comparisons of sex and age between patients with and without signs of CTTH detected on follow-up MRI scans were performed using Fisher's exact test and the Mann-Whitney U test, respectively.

RESULTS

The study population (n=81) consisted of 34 (42%) female, and 47 (58%) male patients. Patients' ages ranged from 2 to 131 months, with a mean age of 22.3±18.3 months and a median age of 17 months (25th percentile, 12 months; 75th percentile, 28.5 months). The estimated prevalence of CTTH was 1.4% (81/5,810) considering all cranial MRI examinations performed during the study period. Based on gestational age, 13 (16%) patients were classified as preterm and 68 patients (84%) as term infants. Twenty-three patients (28.4%) had a history of NICU admission, with a mean duration of 24±24 days. Motor deficits were observed in 11 (13.6%) patients at presentation. Developmental delay was detected in 29 (58%) out of 50 patients who underwent the Denver Developmental Screening Test, and pathological findings were detected on EEGs of 17 (43.6%) out of 39 patients (Table 2).

Table 1. Imaging parameters of the MRI sequences

Parameters	T2 axial	DWI	FLAIR
TR (ms)	4000	4698	11000
TE (ms)	110	97	140
Slice thickness (mm)	4	4	4.2
FOV (mm)	230	194	230
NEX	1	2	1
Acquisition time (s)	136	98	176

TR (ms): Repetition time in milliseconds, TE: Echo time in milliseconds, MRI: Magnetic resonance imaging, FOV: Field of view in millimeters, NEX: Number of excitations

The primary presenting complaints were detected in indicated number of patients as follows: seizures (n=29), developmental delay (n=23), fever (n=4) (suspected central nervous system infection), hypotonia (n=4), movement disorders (n=4), visual symptoms (n=4), behavioral changes (n=4), macrocephaly (n=4), microcephaly (n=1), facial paralysis (n=1), trauma (n=1), suspected diagnoses of Langerhans cell histiocytosis (n=1), and neurofibromatosis (n=1).

Pre-MRI preliminary diagnoses included epilepsy (n=21), asphyxia (n=18), genetic or metabolic disorders (n=14), encephalitis/encephalopathy/infection (n=13), suspect cases of hemorrhage or mass lesion (n=9), and conditions potentially associated with neurodevelopmental disorders in 6 patients. Definitive genetic and metabolic diagnoses are summarized in Table 3.

While no additional MRI findings were observed in 39 patients, 42 (51.9%) patients demonstrated additional findings accompanying CTTH (Figure 2). In 16 patients, symmetrical and bilateral signal hyperintensities were observed in other cerebral regions on diffusion-weighted images including: isolated inferior cerebellar peduncles in 5, isolated fornix in 5, fornix + globus pallidus + hippocampus in 2, fornix + mammillary body + hippocampus in 2, isolated hippocampus in 1, and fornix + hypothalamus in 1 patient, respectively. Additional findings included periventricular leukomalacia/asphyxia-related changes in 12, thinning of the corpus callosum in 3, arachnoid cyst in 3, hydrocephalus in 2, septo-optic dysplasia in 2, subependymal heterotopia in 1, cortical tuber in 1, quadrigeminal lipoma in 1, and subarachnoid hemorrhage in 1 patient, respectively.

There were no statistically significant gender differences between patients with and without additional MRI findings (p=0.368). The median age of the patients without and with additional MRI findings was 23 months (25th percentile, 13 months; 75th percentile, 33 months) and 15 months (25th percentile, 10.8 months; 75th percentile, 22 months), respectively, with a statistically significant difference between the groups (p=0.027).

Follow-up MRI scans were available for 30 (37%) patients. The mean interval between the initial and follow-up MRI examinations was 13.5±20.3 months. When follow-

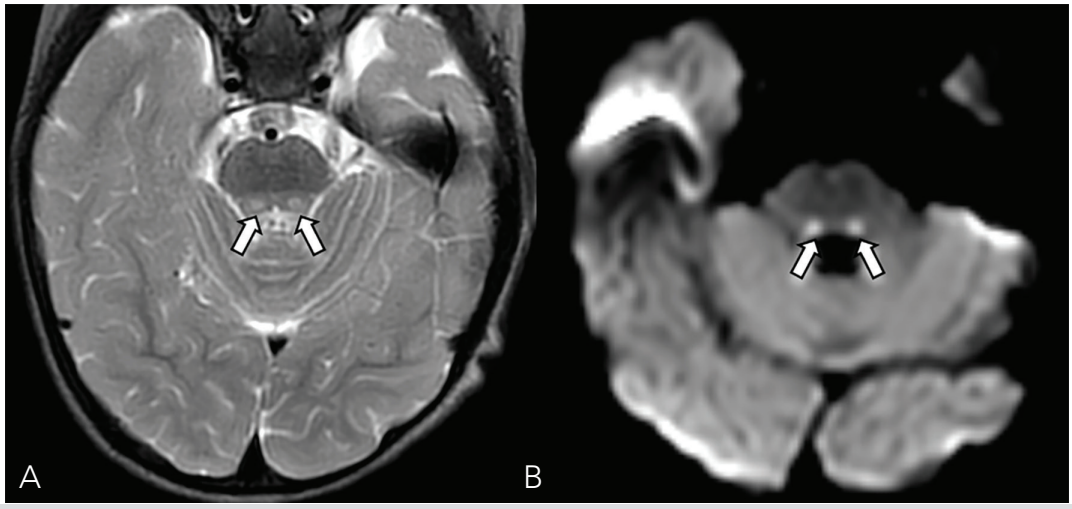

Figure 1. In a 16-month-old male patient followed for mitochondrial disease, central tegmental tract hyperintensity is demonstrated on (A) T2-weighted imaging and (B) diffusion-weighted MRI (arrows)

MRI: Magnetic resonance imaging

Table 2. Demographic and clinical characteristics of the cases	
Total number of cases	**81**
Age (months), median	17 (25p 12; 75p 28.5) months
Age (months), mean (minimum-maximum)	22.3 (2-131) months
n (%)	
Female patients	34 (42)
Male patients	47 (58)
Term infants	68 (84)
Preterm infants	13 (16)
No NICU admissions	58 (71.6)
NICU admissions present	23 (28.4)
Past history of seizure	50 (64.2)
Patients with seizure	29 (35.8)
Normal development	21 (42)
Isolated motor delay	4 (8)
Isolated cognitive/language delay	8 (16)
Global developmental delay	17 (34)
Developmental assessment was not performed	31 (38)
Normal EEG	22 (56.4)
Pathological EEG	17 (43.6)
EEG not performed	42 (51)
Patients without genetic disorder	61 (75.3)
Patients with genetic disorder	20 (24.7)

EEG: Electroencephalography, NICU: Neonatal intensive care unit

up MRI scans were examined, it was seen that CTTH had resolved in 11 (36.7%) and persisted in 19 (63.3%) patients. There was no significant difference in terms of the follow-up intervals between patients with resolved and persistent CTTH (p=0.11). No significant difference was observed on follow-up MRI scans regarding the distribution of male and female patients with and without CTTH (p=0.1). The median age of patients without and with CTTH on follow-up MRI scans was 26 months (25th percentile, 21 months; 75th percentile, 35 months) and 14 months (25th percentile, 5 months; 75th percentile, 20 months), respectively, with a statistically significant difference between the groups in terms of median ages of the patient groups (p=0.003).

DISCUSSION

The results of our study performed in the pediatric population have demonstrated that CTTH is associated with a heterogeneous clinical spectrum that shows variability with age. The median age of the 81 patients included in the study was 17 months. One of the most notable findings was that patients with additional MRI abnormalities were significantly younger (median age, 15 months) than those without (median age, 23 months) (p=0.027). Furthermore, the finding that patients in whom CTTH resolved on follow-up MRI scans were older (median age, 26 months) than those in whom CTTH persisted (median age, 14 months) (p=0.003), which suggests that CTTH may be a maturation-related and potentially transient phenomenon.

Previous studies have reported a wide spectrum of clinical presentations and indications for imaging in

Table 3. Patients with CTTH and confirmed genetic and metabolic diagnoses	
Diagnosis	**n**
Glutaric aciduria type 1	2
Mitochondrial disease	2
Septo-optic dysplasia	2
FOXC1 mutation	1
L-2-hydroxyglutaric aciduria	1
Neurofibromatosis type 1	1
Greig cephalopolysyndactyly syndrome	1
Achondroplasia	1
Poretti-Boltshauser syndrome	1
Thiamine-biotin-responsive basal ganglia disease	1
Tuberous sclerosis	1
Total**	14

**Six patients whose clinical and/or imaging findings were compatible with a neurometabolic disorder but in whom a definitive genetic or metabolic diagnosis had not been established at the time of the study were not included in this table.
FOXC1 mutation: a genetic defect in the *FOXC1* gene on chromosome 6
CTHH: Central tegmental tract hyperintensity

patients with CTTH[1,3,9]. Dablan et al.[10] reported seizures (40.3%) and developmental delay (15.5%) as the most common presenting complaints in patients with CTTH. In addition, hypotonia, macrocephaly, and movement disorders have also been described as frequent indications for imaging[1,4,9]. In our study, in line with the existing literature data, seizures (35.8%) and developmental delay (28.4%) were the most common presenting complaints of patients with CTTH.

One widely accepted hypothesis is that CTTH occurs in the context of hypoxic-ischemic encephalopathy, in patients receiving vigabatrin for the treatment of West syndrome, and in metabolic conditions such as glutaric aciduria type 1 or mitochondrial disorders[3,7,11,12]. CTTH has also been reported in rare metabolic conditions, including Tay-Sachs disease, Krabbe disease, and *CLCN2*-related leukoencephalopathy[1,13,14]. In our cohort, 22% of patients had a diagnosis of asphyxia. The most frequently confirmed genetic–metabolic diagnoses were glutaric aciduria type 1 and mitochondrial disorders. The coexistence of rare genetic or syndromic conditions, such as *FOXC1* mutations, Greig cephalopolysyndactyly syndrome, and Poretti-Boltshauser syndrome, further suggests that CTTH may be observed across a broad spectrum of neurogenetic disorders.

Common MRI findings reported in association with CTTH include periventricular leukomalacia, thinning

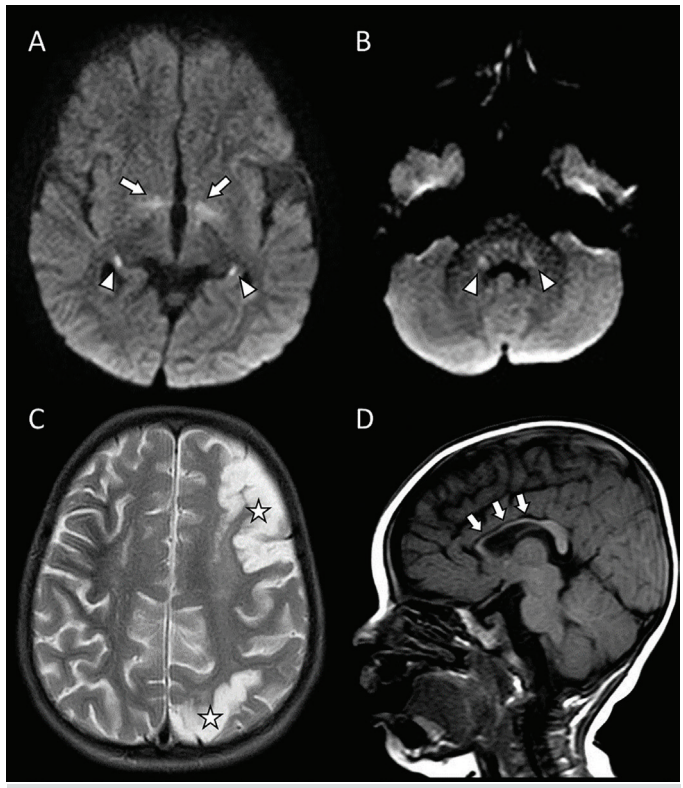

Figure 2. Examples of MRI findings accompanying CTTH. **A)** Bilateral signal increase in the posterior crus of the fornix-hippocampus (arrowheads) and hypothalamus (arrows) on diffusion MRI, **B)** Symmetrical signal increase in the inferior cerebellar peduncles (arrowheads) on diffusion MRI, **C)** Areas of gliotic encephalomalacia (stars) secondary to an old left frontoparietal infarct in a case of cerebral palsy on T2W imaging, and **D)** Thinning of the genu and body of the corpus callosum (arrows) on sagittal T1W imaging

MRI: Magnetic resonance imaging

of the corpus callosum, and signal abnormalities in the basal ganglia[1,5,15]. Işık and Dinçer[9] described diffuse T2 hyperintensity in the fornix, optic nerves, and basal ganglia accompanying CTTH in a patient with mitochondrial disease. In our study, additional MRI findings were detected in 51.9% of patients, while periventricular leukomalacia/asphyxia-related changes were the most frequently detected comorbidities in 14.8% of the patients. Thinning of the corpus callosum was observed in 3.7% of patients. Notably, 19.8% of patients demonstrated symmetrical and bilateral hyperintensities on diffusion-weighted images involving one or more regions, including the fornix, inferior cerebellar peduncle, hippocampus, hypothalamus, mammillary bodies, and globus pallidus. These accompanying pathologies suggest that CTTH may not represent an isolated brainstem abnormality, but rather it accompanies a more

widespread involvement affecting the limbic system and other extrapyramidal pathways.

In their large series of 206 patients, Dablan et al.[10], reported regression of CTTH at a median age of 52 months in 28.6% of their patients as detected on follow-up imaging. Buyukceran et al.[8] reported resolution of CTTH in 58% of patients on follow-up imaging, interpreting resolution as a transient developmental phenomenon and persistence as irreversible myelin degeneration. However, no specific age for the onset of resolution was reported. It has been suggested that endogenous factors, such as underlying mitochondrial disease or severe metabolic disorders may contribute to the persistence of CTTH. In our study, CTTH resolved in 36.7% and persisted in 63.3% of the patients as revealed on follow-up MRI scans. Although previous studies suggested that resolution of CTTH may parallel clinical improvement, our findings indicate that age appears to be a key determinant, supporting the hypothesis that CTTH resolves or disappears as the maturation of the rubro-olivary pathways is completed[4].

Study Limitations

This study has several limitations. The single-center design may limit the generalizability of the findings. Due to its retrospective design, clinical and laboratory data were restricted to information available in hospital records, and advanced genetic testing, such as whole-exome sequencing, could not be performed in all patients with developmental delay, precluding definitive etiological diagnoses in some cases. In addition, the lack of follow-up MRI scans in all patients and the non-standardized follow-up intervals limited the ability to draw stronger conclusions regarding the natural history of CTTH. Finally, the absence of histopathological correlation, similar to other studies in the literature, prevents precise elucidation of the cellular correlates of the observed radiological findings.

CONCLUSION

In conclusion, CTTH is an imaging finding that can be detected on pediatric cranial MRI examinations, but its clinical significance remains uncertain. The clinical relevance of CTTH should be interpreted in the context of patient age and accompanying neurological conditions.

Ethics

Ethics Committee Approval: Ethical approval was obtained from the University of Health Sciences Türkiye, Dr. Behçet Uz Children's Diseases and Surgery Training and Research Hospital (decision no.:2026/01-07, date: 15.01.2026).

Informed Consent: This is a retrospective study.

Footnotes

Author Contributions

Concept: Y.K.Ç., B.M., M.C., Design: Y.K.Ç., B.S.B., M.C., Data Collection or Processing: Y.K.Ç., S.P., B.S.B., D.K.A., B.M., Analysis or Interpretation: Y.K.Ç., S.P., M.C., Literature Search: Y.K.Ç., S.P., D.K.A., B.M., Writing: Y.K.Ç., M.C.

Conflict of Interest: The authors disclose no potential conflicts of interest.

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