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Review / Derleme

Neuroimaging in Developmental Coordination Disorder

Gelişimsel Koordinasyon Bozukluğunda Nörogörüntüleme

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Abstract

Developmental coordination disorder (DCD) is a neuromotor disorder of unknown etiology characterized by motor skill disorder, which significantly inhibits the child's ability to perform daily living activities and affects psychosocial well-being. DCD is one of the least understood and studied neuromotor disorders, and little is known about the neural mechanisms underlying motor impairment. This makes it difficult to understand why children with DCD have difficulty in learning motor skills and what is the best intervention to optimize motor functions. With the further development of neuroimaging techniques, the number of neuroimaging studies to understand the underlying mechanisms in children with DCD has increased in recent years. Results from these studies suggest that children with DCD activate different regions of the brain during functional tasks and show differences in white matter microstructure, cerebellum, basal ganglia, parietal lobe, and parts of the frontal lobe (medial orbitofrontal cortex and dorsolateral prefrontal cortex) compared with typically developing children. We believe that these neuroimaging data obtained from numerous studies will contribute to the explanation of neural mechanisms related to impaired motor function in children with DCD.

Keywords: Developmental coordination disorder, diffusion tensor imaging, EEG, fMRI, MRI, neuroimaging

Öz

Gelişimsel koordinasyon bozukluğu (GKB), motor beceri bozukluğu ile karakterize, çocuğun günlük yaşam aktivitelerini gerçekleştirme yeteneğini önemli ölçüde bozan ve psikososyal iyilik halini olumsuz etkileyen, etiyolojisi bilinmeyen bir nöromotor bozukluktur. GKB'nin etiyolojisini ve GKB'de görülen motor bozukluğun altında yatan nöral mekanizmaları aydınlatan yeterli bilgi ve kanıt mevcut değildir. GKB'li çocuklarda yapılan nörogörüntüleme çalışmalarının sayısı son yıllarda nörogörüntüleme tekniklerinin gelişmesiyle birlikte artmaktadır. Bu çalışmalar, GKB'li çocukların fonksiyonel görevler sırasında beynin farklı bölgelerini harekete geçirdiğini, tipik gelişen çocuklara kıyasla ak madde mikroyapısında, serebellum, bazal ganglionlar, paryetal lob ve frontal lobun bazı bölümlerinde (medial orbitofrontal korteks ve dorsolateral prefrontal korteks) farklılıklar gösterdiğini ortaya koymaktadır. Ayrıca, GKB'nin etiyolojik kökeninin ve patofizyolojisinin aydınlatılmasının uygun tedavi ve müdahale planına karar verilmesinde de kritik öneme sahip olacağı açıktır. Çok sayıda çalışmadan elde edilen bu nörogörüntüleme verilerinin, GKB'li çocuklarda bozulmuş motor fonksiyon ile ilgili nöral mekanizmalarının açıklanmasına katkısı olacağını düşünmekteyiz.

Anahtar Kelimeler: Gelişimsel koordinasyon bozuklukları, difüzyon tensör görüntüleme, EEG, fMRG, MRG, nörogörüntüleme

Introduction

Developmental coordination disorder (DCD) is a neurodevelopmental condition characterized by an inability to perform motor skills at the appropriate age, motor learning difficulties, and coordination disorders. DCD is one of the most common developmental disorders affecting about 6% of school-age children. Children with DCD have difficulty in acquiring and performing motor skills related to daily life, school, work, entertainment and play activities (1). Many emotional and psychosocial problems such as anxiety, lack of self-confidence,

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anxiety, and depression accompany motor difficulties in children with DCD (2). It is estimated that 75% of children with DCD continue to experience these difficulties and problems in adulthood in the absence of any intervention (3).

Despite its high prevalence in the population, little is known about the etiology of DCD and how it develops. The developments in neuroimaging techniques in the last 20 years include studies to explain the etiology of DCD. Studies investigating the relationship between motor dysfunction in DCD and neuroanatomic structures are remarkable. In addition, neuroimaging studies are critical for the selection of therapeutic interventions for children with DCD and for evaluating the effectiveness of these interventions.

The purpose of this review was to systematically review the data obtained from existing neuroimaging studies in children with DCD. We believe that a common perspective on DCD will be obtained by compiling studies with different research methods and different results.

Method

For this review article, the EMBASE, PubMed, PsycINFO, Science of Web, Ebscohost, CINAHL, and ScienceDirect databases were scanned between January 1st, 2000, and May 30th, 2019. In our literature review, the keywords "developmental coordination disorders" and "neuroimaging" and their equivalents as "DCD" and "imaging" were used. Studies with a diagnosis of DCD according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV or DSM-V) criteria and using the Movement Assessment Battery for Children (MABC) for motor evaluation were included. Studies with participants aged 5 years or over were included because the minimum age at which DCD can be diagnosed according to the European Academy of Childhood Disability (EACD) guidelines is 5 years. Studies involving serious neurologic disorders such as cerebral palsy were excluded. Pilot studies were included in the review, but case reports and articles that were not published in English were excluded.

In this review, a total of 29 studies, including 12 functional magnetic resonance imaging (fMRI) studies, 4 MRI studies, 5 diffusion tensor imaging (DTI) studies, 6 electroencephalogram (EEG) studies, and 2 functional near-infrared spectroscopy (fNIRS) studies were examined. All of the participants in the studies met the diagnostic criteria for DCD.

Neuroimaging Studies in Children with Developmental Coordination Disorders

1- Functional Magnetic Resonance Imaging

fMRI is an indirect measure of neural activity that measures blood oxygenation and local blood flow due to changes in the metabolic activity of active neurons. As the amount of oxyhemoglobin increases in the capillaries as a response to neural activity, the amount of deoxyhemoglobin decreases. This change in the ratio of oxyhemoglobin and deoxyhemoglobin can be detected using fMRI (4).

Considering the structural changes in the brain in children with DCD, an increase in activity is observed in the pathway between the inferior parietal cortex in the left hemisphere and both the middle frontal cortex and the anterior cingulate nucleus. In addition, increased activity was found in the pathway between the striatum and parietal cortex in the right hemisphere. It is thought that the reason for this increase in activity is due to weak inhibition mechanisms. Typically, there is a right lateralization tendency in the typically developing children and a left lateralization tendency in DCD. Querne et al. (5) reported that DCD was a condition characterized by the developmental differences of the brain hemispheres in the neurodevelopmental process.

Clumsiness is a common clinical problem in children with DCD. Kashiwagi et al. (6) examined activation areas of the brain with fMRI by giving a visual-motor task to understand the neural mechanism underlying the clumsiness seen with DCD. As a result of this study, it was found that there were also losses in behavioral performance in DCD, in addition to the finding of the study conducted by Querne et al. (5); brain activity was less in the superior and inferior parietal lobes, left posterior parietal cortex, and left post-central gyrus in children with DCD compared with typically developing children. According to the authors, there may be a relationship between the posterior parietal cortex and clumsiness (6). This relationship between the posterior parietal cortex and clumsiness suggests that the same connectivity in the left lateralization hypothesis of Querne et al. (5) is impaired.

Another problem experienced by children with DCD is fatigue in motor activities. The first neuroimaging evidence to support clinical observations on fatigue was provided in the study of Zwicker et al. (7) published in 2010. In this study, it was investigated whether there was a difference in brain activation patterns during fine motor tasks in children with and without DCD. It was found that children with DCD compensated for poor sensory feedback from joints and muscles with visual information and relied more on visual systems for motor tasks. It was also shown that they used the cerebellum lobule VI area more, which was related to visual-spatial processing, and that twice as many areas of the brain were activated to learn a motor task in patients with DCD. For this reason, it is thought that children with DCD also experience cognitive fatigue related to planning, executing, and learning efforts (7).

The study conducted by Zwicker et al. (8) in 2011 was important because it was the first to examine the effects of motor training and changes in brain activation patterns using fMRI scanning in children with DCD. It was shown that it showed lower signaling in all parts of the brain during the learning process. Children with DCD did not show improvement in performing the motor task correctly after 3 days of task training. Less activation was found in the cerebellar-parietal and cerebellar-prefrontal pathways and brain regions associated with visual-spatial learning. The reason for the insufficient learning of motor skills in children with DCD may be due to a neurobiologic disorder.

Children with DCD have longer reaction times than typically developing ones. In a study by Debrabant et al. (9), using fMRI to investigate the neural relationships of motor timing and reaction time in children with and without DCD, it was shown that children with DCD showed decreased signalization in the right dorsolateral prefrontal cortex, the left posterior cerebellum, and the right temporo-parietal junction. Temporal errors increase when visual or auditory stimuli are given simultaneously.

In addition, the provision of visual stimuli and temporal adjustments do not improve reaction times. It is thought that the failure of children with DCD to respond in the expected time is due to a developmental delay (9). These findings suggest that children with DCD require more effort to perform visual-motor tasks. In a study comparing motor movement patterns that increased cortical activation using fMRI during finger overlap and handshaking tasks, motor movement was found to be weaker in children with DCD compared with typically developing children. According to the analyses performed, no difference was found in handshaking activity in children with DCD, and increases in the activities of the left and right superior frontal gyrus and left inferior frontal cortex were found in the activity of finger overlap task (10). Biotteau et al. (11) reported that only the brain activation patterns of patients with DCD differed from children with developmental dyspraxia and children with both developmental dyspraxia and DCD. These findings suggest that DCD is caused by a neurobiologic mechanism independent of other accompanying clinical conditions.

2- Magnetic Resonance Imaging

MRI is a non-invasive neuroimaging tool for obtaining detailed images of biologic structures with strong magnetic fields and radio waves without using radiation. An MRI scanner arranges hydrogen atoms in the body with the magnetic field it creates. Radio waves enable these arranged atoms to generate signals that are used to form images. Thus, different images can be obtained to characterize the structure and function of the brain. T1-weighted imaging generally allows differentiation of brain anatomy, white matter and gray matter. T2-weighted imaging, on the other hand, distinguishes brain tissue, and is particularly suitable for diagnosis of inflammation, demyelination, and white matter changes (12).

According to MRI studies performed in children with DCD, abnormal activations in brain areas were found (8,9,10,13). In 2014, Langevin et al. (14) concluded that the orbitofrontal cortex was thinner in people with DCD and this was related to lower scores in motor function tests. In addition, it was found that separation and integration and visual-motor pathway in structural connections were weaker in children with DCD (15).

Although the negative correlation between the left posterior cerebellar lobe gray matter volume and the motor skill score in fine motor activity of drawing curved lines in children with DCD was striking, a positive correlation was found between the gray matter volume of the medial frontal cortex and the error in movements. This study, which was the first to show the relationship between gray matter volume and DCD, showed that there were changes in the cerebellum and parietal lobe associated with DCD (16).

It has been demonstrated that the same neuron network is used in typically developing children and children with DCD, and that the global neuronal connection in children with autism spectrum disorder (ASD) differs from typically developing children and children with DCD. Children with both DCD and ASD differ in terms of both structural and functional characteristics in brain regions related to motor function compared with typically developing children. It has been determined that these differences are caused by the white matter organization in the sensorimotor pathways in children with DCD and microstructural losses in the motor, sensory and cerebellar pathways in ASD. In the same studies, it was found that there was a correlation between these differences and motor function (17).

A decrease in cortical thickness was found in the parietal, frontal, and temporal lobes in children with DCD and attention-deficit/hyperactivity disorder (ADHD). This study by Langevin et al. (18) in 2015 supported the fact that ADHD and DCD originated from the same neurobiologic markers. This study by Langevin et al. (18) was the first to show that the cortical area of children with motor and attention problems was thinner.

Low birth weight and preterm delivery are thought to be risk factors for DCD. In an MRI study of 7-year-old children born preterm or with low birth weight, white matters of the right and left hemispheres, caudate nucleus, activation of the right and left hippocampus, and motor performance ($<5^{th}$ percentile) using the MABC were evaluated. This study showed that children with lower motor performance had lower right and left hemisphere white matter activation times. However, no relationship was found between right and left hippocampal activations and caudate nucleus volume difference (19). In an MRI study performed on adolescents with low birth weight, with a mean age of 15 years, moderate-to-advanced ventricular dilatation, focal or diffuse corpus callosum thinness, unilateral or bilateral decreased ventricular white matter were found to be associated with lower motor performance (20).

In a study investigating the relationship between congenital stress and childhood DCD, newborns were classified according to congenital MRI abnormalities. MRI abnormalities in newborns with encephalopathy after perinatal asphyxia were classified as class 1: No damage, class 2: Moderate damage, white matter lesions, and class 3: Moderate-severe damage, basal ganglia/thalamus injury, and focal ischemia. MABC scores were not found $>15^{th}$ percentile in all children with moderate/severe lesions in neonatal MRI, and in 62% of children with normal MRI or mild lesions. It was shown that children with moderate/severe lesions, childhood MRI findings and MABC score not more than the 15^{th} percentile significantly overlapped each other (100%), and that MABC scores were not found $>15^{th}$ percentile in 47% of children with mild lesions or without lesions (21).

3- Diffusion Tensor Imaging

DTI is a neuroimaging method used to measure the amount of displacement (mean diffusion) and relative direction (fractional anisotropy) of the water molecule in axons. Currently, DTI is the only approach that can view the white matter of the brain non-invasively (22). The diffusion parameters used in the DTI method are as follows: Axial diffusion (diffusion of water along the length of the axons) and radial diffusion (water diffusion perpendicular to the axons). An increase in these parameters reflects a better white matter structure for efficient and rapid transfer of information between different brain regions, which supports the development of well-functioning and synchronized neural pathways (23).

It is very important to examine the structure of white matter to understand the neurodevelopmental problems and the underlying causes of these problems in children with DCD. The DTI method has been used in many studies to examine the white matter of the brain. Zwicker et al. (24) compared axial diffusion in the corticospinal tract and thalamic connections in children with DCD and children with normal development and found that axial diffusion was lower in children with DCD. In addition, it was shown by Zwicker et al. (24) that there was a correlation between axial diffusion and MABC.

According to neuroimaging studies conducted in recent years, permanent neurobiologic changes in the white matter in corticospinal tracts associated with motor planning and cognition have been found in children with DCD. Williams et al. (25) investigated the diffusion of white matter in the corticospinal tract, superior longitudinal fasciculus (SLF) and internal capsule using DTI in their neuroimaging study in children with DCD and found that white matter diffusion in children with DCD was lower than in healthy children. SLF is a structure that provides the connection between the anterior and posterior cortical areas. Any defect in this structure causes problems in functions such as attention control, motor movement memory, and movement planning. In addition, the internal capsule is also related to motor skills. Williams et al. (25) suggested that the reduced axonal diffusion seen in these brain structures might be related to the motor problems seen in children with DCD. In the same study, white matter diffusion in the inferior longitudinal longitudinal fasciculus (ILF) was also examined (25). The ILF provides the connection between the occipital and temporal lobes and also represents the visual ventral pathway. It is known that there are problems in the processing of visual-perceptual information in children with DCD (15). It is thought that visual problems seen in children with DCD may be related to low axonal diffusion in ILF (15,25). Another study examining the cause of low axonal diffusion detected in the brain structures of children with DCDs is the study of Hyde et al. (26). It has been determined that children with DCD have lower axon diameters and numbers in SLF than normal children and it has been suggested that this may be related to the decrease in motor abilities (26).

DCD can be seen together with other developmental disorders and attention disorder (27). In a neuroimaging study using DTI, Langevin et al. (14) reported that axial diffusion decreased in the parietal lobe above the corpus callosum, as well as the white matter in the SLF in children with diagnoses of DCD and attention disorder. In the same study, they found that there were abnormalities in the connections between the corpus callosum and the primary and somatosensory motor areas of the parietal lobe. The findings in this study supported that DCD and attention disorder shared the same etiology (14).

Comorbidity is also important because it affects research findings in DCD. Children with DCD may have one or more disorders (such as ADHD) and depending on the degree of dysfunction of brain development, DCD may result from different neural mechanisms (28). Therefore, it is important to identify DCD and perhaps distinguish it from other comorbidities when investigating neural correlations. However, there are very few studies on this subject in the literature. More neuroimaging studies are needed on DCD and its comorbidities to fully illuminate this issue (29).

4- Electroencephalogram

EEG is the oldest functional brain imaging technique and measures the electrical activity of neurons with the placement of electrodes on the scalp. EEG allows visualization of neuronal events occurring at the millisecond level, as well as measuring the current flow emanating from synaptic changes produced by the dendrites of pyramidal neurons. Studies reveal that EEG can directly monitor electrical activities caused by disorders in various structures of the brain. This technique is more preferred in children because it is easier to perform than MRI (30).

According to EEG studies conducted on children with DCD, there is a correlation between EEG signals detected from cortical areas and motor functions. These cortical areas are expressed as follows: Frontal lobe, parietal lobe, and lateral areas of the right and left hemispheres. Overall, these studies compared typically developing children and children with DCD. According to EEG findings, it was determined that children with DCD and typically developing children showed similar performance in EEG, but those with DCD showed different cortical activation patterns (31,32). Motor memory, which is responsible for the simultaneous and temporary storage and processing of the data needed in the execution of complex cognitive tasks, forms the basis of effective problem-solving and planning skills and is supported by visual-spatial inputs (33,34). In addition, it has been determined that children with DCD also have deficits in their visual-spatial memory. Tsai et al. (35) gave a task related to visual-spatial memory to children with DCD, examining the EEG findings during this task, and found that children with DCD had longer reaction times compared with their normal peers, and that they allocated less neural resources in the task assessment and response process. Pangelinan et al. (36) also recorded EEG signals of children with DCD while they were drawing a circle with a digital pen on a computer screen, and concluded that children with DCD activated less cortical area and had lower EEG signal amplitudes than their normal peers.

Approximately 50% of children with DCD also have attention disorder (37). According to some studies, it has been stated that children with DCD have atypical cerebellum and basal ganglia activation and therefore many executive functions including attention are likely to be affected (38,39). In addition, it has been stated in the literature that attention can affect neuromuscular motor performance (40). Based on this hypothesis, Fong et al. (41) evaluated attention levels during motor tasks in children with DCD using EEG and concluded that the relationship between attention and motor performance could be better-determined thanks to EEG signals, which can be obtained instantly during simple and complex motor tasks. According to the data of this study, it was found that the motor performances and EEG signals of children with DCD were lower than their normal peers, and there was a positive relationship between motor performance and EEG (41).

It is known that children with DCD have difficulties in motor skills that require bimanual activity (42). Bimanual coordination is based on the functional bonding between the motor regions of both brain hemispheres via the corpus callosum. Therefore, bimanual coordination provides an important opportunity to investigate possible dysfunctions in brain communication (43). Blais et al. (44) examined both inter-hemisphere and intra-hemisphere brain activities of children with DCD during bimanual activities using EEG. It was found that interhemispheric connections were less in children with DCD compared with their normal peers. In this study, it was noted that the performance of repetitive motor tasks did not increase the intrahemispheric connections of children with DCD. The findings of this study provide new neuroimaging evidence that children with DCD display different motor behaviors compared with their normal peers. It is emphasized that atypical

brain development in children with DCD may result from the motor inhibition information carried between hemispheres, and more studies should be conducted to investigate the structure of the corpus callosum, which can be involved in atypical interhemispheric communication, to better understand the neural connections of DCD (44). It was stated by different authors in the literature that movement stability decreased due to involuntary movements that children with DCD showed during motor activities, and this might be caused by the lack of motor inhibition (45,46,47).

5- Near-infrared Spectroscopy/Functional Near-infrared Spectroscopy

Although NIRS was developed to measure cerebral oxygenation, it is also called fNRIS. fNIRS is a non-invasive neuroimaging technique used to measure changes in the concentration of oxyhemoglobin and deoxyhemoglobin in blood during neuronal activation by sending infrared rays in the wavelength range of 700-1300 nm. It is a suitable neuroimaging method for use in studies analyzing brain functions by experimental protocols involving motor activity in children with DCD. fNIRS shows similarities with fMRI, but it is less costly than fMRI and can also be performed with a portable device (48).

There are only two studies conducted with fNIRS on children with DCD. In the first of these studies, two groups of children with DCD and typically developing children were given the task of matching cards according to their colors, shapes, and numbers, and during the task, the dorsolateral prefrontal cortex of the children was examined using fNIRS. Dorsolateral prefrontal cortex activation was found to be higher in children with DCD compared with typically developing children. Koch et al. (49) suggested that children with DCD made more effort to minimize the margin of error behaviorally, and therefore activation in the dorsolateral prefrontal cortex increased in a compensatory manner. In the second fNIRS study, Caçola et al. (50) investigated whether there was a difference in terms of the cortical activation areas between children with DCD and typically developing children during motor tasks given to evaluate visual-motor perception and writing skills. They found that less cortical area was activated and these activation areas were mostly ipsilateral in children with DCD. The fact that children with DCD use more ipsilateral paths may be due to corpus callosum disorders seen in children with DCD (51). By using advanced neuroimaging techniques such as fNIRS, these atypical neural connections seen in DCD can be analyzed better. Therefore, more studies using advanced neuroimaging techniques in children with DCD are needed.

Discussion

According to the results obtained from this literature review, it is seen that children with DCD show different neural activation patterns compared with typically developing children during motor tasks.

Electroencephalogram

According to most EEG studies included in the review, the EEG values for cortical activation of children with DCD are lower than the typically developing children. However, in one study, the EEG activation rate of children with DCD was found to be high (52). The reason for this was claimed that the high EEG activation rate could be due to compensation for the difficulties in perceptual-motor and executive functions while performing movements (52). According to the EEG studies included in the review, although the reaction time of children with DCD to a given motor task was delayed compared with children with typical development, EEG values obtained from the frontal and central regions of the brain were also found to be lower (31,53). The reason for this is that children with DCD engage less neural resources (33,41,44).

Functional Magnetic Resonance Imaging

According to the fMRI studies included in the review, it was found that children with DCD had lower activation in the left parietal cortex, cerebellum, and basal ganglion regions compared with children with typical development, and that there were atypical connections between the sensory-motor cortex, cerebellum, and basal ganglia areas (54,55,56). The left parietal lobe is the brain region responsible for three-dimensional body perception, providing somatosensory and visual information necessary for hand-eye coordination, and perception of shape, touch, and pressure (57,58). The cerebellum and basal ganglia are responsible for motor movement planning, muscle tone control, and body balance. In functional disorders of the parietal region, although there were disturbances in body perception, touch, and pressure sensations, problems in movement planning, muscle tone, and body balance were seen in the cerebellum and basal ganglia disorders (59). In studies conducted with children with DCD, it was stated that these children had problems in body perception, hand-eye coordination, body balance, pressure, and touch senses (60). fMRI studies also support these results. In addition, it was observed that children with DCD had developmental disorders in their mirror neuron systems. This situation also explains why children with DCD have more problems with motor imagery than typically developing children (61). See Table 1 for a summary of the research findings.

Magnetic Resonance Imaging

According to the MRI studies in the review, it has been observed that the gray matter volume of the patients with DCD is lower than the typically developing children, but also the cortical thickness is lower. In addition, it is stated that this atypical brain development may be associated with motor dysfunction (14,16,18). However, some children with DCD who take part in MRI studies are children with a preterm history. It is not possible to generalize the results obtained with MRI on brain gray matter because the sample groups in the studies are heterogeneous and the number of studies is low.

Diffusion Tensor Imaging

DTI is the only neuroimaging method that can analyze the white matter of the brain. According to the DTI studies in the review, the white matter structure of children with DCD is different from that of typically developing children. The amount of axial infusion in the visual-motor pathways of the brain, parietal, and frontal cortex is lower in children with DCD compared with children with typical development (15,24). This atypical white matter structure causes negative effects on motor planning and cognition in children with DCD (25).

Table 1. Summary of study findings in the literature on neuroimaging

First author, journal, year, country	Participants (number, age range, mean age)	Inclusion and exclusion criteria	Neuroimaging technique	Neuroimaging results
Querne et al. (5), Brain Res, 2008, France	DCD: 9 Age: 8.0-12.9 (9.9±1.8) TDC: 10 Age: 8.2-11.6 (10.0±1.1)	DCD: Having a diagnosis of DCD according to DSM-IV criteria Not having neurological and psychiatric disorders (e.g. cerebral lesion, ADHD) IQ >80	fMRI	In children with DCD compared to TDC: Increased activation in the left hemisphere in the middle frontal cortex, anterior cingulate cortex, and inferior parietal cortex; decreased activation in the striatum and parietal cortex in the right hemisphere
Kashiwagi et al. (6), Neuroreport, 2009, Japan	DCD: 12 Age: 9-12 (11.4±1.6) TDC: 12 Age: 9-12 (11.2±1.3)	DCD: Having a diagnosis of DCD according to DSM-IV criteria MABC <15th percentile IQ >90	fMRI	Less activation was found in the left parietal cortex and left post-central gyrus in the group with DCD compared to the TDC group. Similar activation rates were observed in both groups in the cerebellum and basal ganglia areas
Zwicker et al. (7), Pediatrics, 2010, Canada	DCD: 7 Age: 8-12 (10.8±1.5) TDC: 7 Age: 8-12 (10.9±1.5)	DCD: MABC-2 ≤16th percentile TDC: MABC-2 >25th percentile IQ >80 Not having a diagnosis of ADHD	fMRI	Although both groups were given the same motor tasks, it was observed that different areas were activated in both groups. More cortical activation was detected in the left parietal lobe, right middle frontal gyrus, right superior temporal gyrus, right cerebellar lobe areas in DCD group compared to the TDC group, while in the TDC group, more cortical activation was detected in the left superior and inferior frontal gyrus than in the DCD group
Zwicker et al. (8), Int J Dev Neurosci, 2011, Canada	DCD: 7 Age: 8-12 (10.8±1.5) TDC: 7 Age: 8-12 (10.9±1.5)	DCD: MABC-2 ≤16th percentile TDC: MABC-2 >25th percentile IQ >80 Not having a diagnosis of ADHD	fMRI	The DCD group showed less cortical activation in the cerebellar, parietal and frontal areas than the TDC group
Debrabant et al. (9), Res Dev Disabil, 2013, Belgium	DCD: 17 Age: 7-10 (9.4±0.6) TDC: 17 Age: 7-10 (9.2±0.9)	DCD: MABC-2 ≤5th percentile TDC: MABC-2 >16th percentile IQ >85 Not having a diagnosis of ADHD	fMRI	During motor tasks, less cortical activation was found in the cerebellar areas in the DCD group compared to the TDC group
McLeod et al. (13), Neuroimage Clin, 2014, Canada	DCD: 7 Age: 8-17 (13.0±2.5) ADHD: 21 Age: 8-17 (12.5±2.9) DCD + ADHD: 18 Age: 8-17 (11.5±3.0) TDC: 23 Age: 8-17 (11.3±2.8)	DCD: MABC-2 >16th percentile, IQ >80 ADHD: Being diagnosed as having ADHD according to the DSM-V criteria	fMRI	Atypical neural connections were found in the sensory-motor cortex, cerebellum, and basal ganglion areas in the DCD group and DCD + ADHD group compared to the TDC group

Table 1. continued

First author, journal, year, country	Participants (number, age range, mean age)	Inclusion and exclusion criteria	Neuroimaging technique	Neuroimaging results
Licari et al. (10), Exp Brain Res, 2015, Australia	DCD: 13 Age: 8-10 (9.6±0.8) TDC: 13 Age: 8-10 (9.3±0.6)	DCD: MABC-2 <5th percentile, TDC: MABC-2 >15th percentile Not having a diagnosis of ADHD	fMRI	Less cortical activation was detected in the right postcentral gyrus and left superior and inferior frontal gyri, and deficits were observed in the mirror neuron system in the DCD group
Biotteau et al. (11), Eur Journal of Paediatr Neurol, 2017, France	DCD: 16 Age: 8-12 (9.6±1.7) DCD + DD: 16 Age: 8-12 (9.9±1.1) TDC: 16 Age: 8-12 (10.3±1.3)	DCD: MABC-2 ≤5th percentile DD: MABC-2 >16th percentile IQ >85 Not having a diagnosis of ADHD	fMRI	Increased activation was found in the premotor cortex in the DCD and DD groups
Mcleod et al. (55), Neuroimage Clin, 2016, Canada	DCD: 6 Age: 8-16 (13±2.8) DCD + ADHD: 14 Age: 8-16 (11.3±3.8) ADHD: 19 Age: 8-16 (12.4±3.1) TDC: 21 Age: 8-16 (11±2.8)	DCD: MABC-2 <16th percentile, ADHD: Being diagnosed as having ADHD according to the DSM-V criteria	fMRI	A stronger connection was found between the middle frontal gyrus and inferior lateral occipital cortex and the left sensory-motor cortex in the DCD + ADHD group compared to the right sensory-motor cortex. On the other hand, an equal connection was found between the middle frontal gyrus and inferior lateral occipital cortex and the right and left sensory-motor cortex in the TDC, DCD, and ADHD groups
Thornton et al. (56), Hum Mov Sci, 2018, Canada	DCD: 9 Age: 8-12 (10.55±1.67) DCD + ADHD: 18 Age: 8-12 (10.94±2.62) ADHD: 20 Age: 8-16 (13.5±2.74) TDC: 20 Age: 8-12 (10.2±2.8)	DCD: MABC-2 <16th percentile, ADHD: Being diagnosed as having ADHD according to the DSM-V criteria	fMRI	Less activation was detected in the primary sensory and motor cortex areas of the DCD, DCD + ADHD, and ADHD groups compared to the TDC group
Reynolds et al. (54), Int J Dev Neurosci, 2015, Australia	DCD: 10 Age: 8-12 (10.18±1.3) TDC: 9 Age: 8-12 (10.4±1.17)	DCD: MABC-2 <16th percentile TDC: MABC-2 >25th percentile	fMRI	It was determined that there were deficits in the mirror neuron system in the DCD group, and therefore it was suggested that children with DCD had problems in motor imaging
Lloyd et al. (16), Magn Reson Med, 2010, UK	DCD: 14 Age: 8-13 (10.7±2.7)	DCD: MABC-2 <16th percentile	MRI	A negative correlation was found between the cerebellar posterior lobe gray matter volume and the root mean square error score
Langevin et al. (18), Dev Med Child Neurol, 2015, Canada	DCD: 14 Age: 8-17 (9.9±1.7) DCD + ADHD: 10 Age: 8-17 (9.7±2.3) ADHD: 10 Age: 8-17 (9.9±1.3) TDC: 14 Age: 8-17 (11.2±3)	DCD: MABC-2 <16th percentile TDC: MABC-2 >25th percentile ADHD: Being diagnosed as having ADHD according to the DSM-V criteria	MRI	Less parietal, temporal and frontal cortical thickness was detected in the DCD + ADHD group compared to the other groups

Table 1. continued

First author, journal, year, country	Participants (number, age range, mean age)	Inclusion and exclusion criteria	Neuroimaging technique	Neuroimaging results
Caeyenberghs et al. (17), Dev Sci, 2016, Australia	DCD: 11 ASD: 15 DCD + ASD: 8 TDC: 19 Age: 8-12 years	DCD: Being diagnosed as having DCD according to the DSM-IV criteria IQ >75	MRI	There was a difference between the DCD, ASD and DCD + ASD groups and TDC in terms of the clustering coefficient of brain gray matter volume, which was an indicator of brain connections. An increase in clustering coefficient was detected in the right inferior frontal gyrus in the ASD group, in the right lateral orbitofrontal cortex in the DCD group, and in the left temporal cortex and right medial orbitofrontal cortex in the DCD + ASD group It has been found that increase in clustering coefficient indicates dysfunction in neural mechanisms and is associated with poor performance in motor tasks
Reynolds et al. (61), Int J Dev Neurosci, 2017, Australia	DCD: 22 Age: 8-12 (9.9±1.1) TDC: 22 Age: 8-12 (9.7±1.2)	DCD: MABC-2 <16th percentile TDC: MABC-2 >25th percentile Not having a diagnosis of ADHD	MRI	It was determined that the gray matter volume in the frontal cortex of children with DCD was lower than the TDC
Zwicker et al. (24), Pediatr Neurol, 2012, Canada	DCD: 7 Age: 8-12 (10.2±1.6) TDC: 9 Age: 8-12 (10.4±1.7)	DCD: MABC-2 <16th percentile Kaufmann Brief Intelligence test >80 Conner Attention-Deficit/Hyperactivity Disorder Diagnostic scale <70	DTI	The average diffusion amount in the corticospinal tract was found to be lower in children with DCD than in TDC A positive correlation was found between axial white matter diffusion and MABC-2 scores
Langevin et al. (14), J Pediatr, 2014, Canada	DCD: 9 Age: 8-12 (11.5±3.18) ADHD: 23 Age: 8-12 (12.22±2.68) DCD + ADHD: 26 Age: 8-12 (11.78±2.99)	DCD: MABC-2 <16th percentile ADHD: Conners' Parent Rating scale >95th percentile	DTI	Decreased white matter diffusion in the parietal lobe in children with DCD Decreased white matter diffusion was found in the frontal lobe in children with ADHD Decreased white matter diffusion was found in both parietal and frontal lobes in children with DCD + ADHD
Williams et al. (25), Neuroreport, 2017, Australia	DCD: 7 Age: 18-40 (24.5±7.6) Control group: 9 Age: 18-40 (26.7±5.5)	DCD: McCarron Assessment of Neuromuscular Development ≤85 Not having a diagnosis of autism, Asperger's syndrome or ADHD Control group: McCarron Assessment of Neuromuscular Development >85 Not having a diagnosis of autism, Asperger's syndrome or ADHD	DTI	A significant decrease was detected in the fractional anisotropy of white matter in the corticospinal tract and superior longitudinal fasciculus in the DCD group, and there was a significant decrease in the mean diffusion amount of white matter in the internal capsule and inferior longitudinal fasciculus in the DCD group compared to the control group

Table 1. continued

First author, journal, year, country	Participants (number, age range, mean age)	Inclusion and exclusion criteria	Neuroimaging technique	Neuroimaging results
Debrabant et al. (15), J Pediatr, 2016, Belgium	DCD: 21 Age: 8-10 years (9.2±1.1) TDC: 20 Age: 8-10 years (9.4±1.7)	DCD: MABC-2 ≤5th percentile	DTI	White matter diffusion in the visual-motor tracts was lower in the DCD group
Hyde et al. (26), Neuroimage Clin, 2018, Australia	DCD: 7 Age: 18-46 years (23.29±4.31) Control group: 20 Age: 18-46 years (26.16±4.34)	DCD: Being diagnosed as having DCD according to the DSM-V criteria	DTI	Axon diameters and numbers in the left Superior Longitudinal Fasciculus were lower than the control group, while a decrease in the volume of the right Superior Longitudinal Fasciculus was also detected in the DCD group. A positive correlation was found between this decrease and deficiencies in motor skills
Lust et al. (53), Child Care Health Dev, 2006, Holland	DCD: 10 Age: 8-12 years (10.4±1.1) TDC: 7 Age: 8-12 years (9.6±1.7) Adult group: 14 Age: 19-29 years (22.4±4.7)	DCD: MABC-2 <16th percentile	EEG	In the test estimating the right and left hand pictures rotated in 45 degrees in right and left directions; while accurate prediction, response times and EEG activations were found similar in the DCD and TDC groups, better results were obtained in the adult group and it was found that this test showed age-related changes
DeCastalneu et al. (31), Hum Mov Sci, 2008, France	DCD: 24 (3 age groups of 8-9, 10-11 and 12-13 years) TDC: 24 (3 age groups of 8-9, 10-11 and 12-13 years)	DCD: Being diagnosed as having DCD according to the DSM-V criteria MABC-2 ≤5th percentile IQ >80	EEG	According to the EEG findings, it was found that intrahemispheric connections between the frontal-central areas increased more in only the 8-9 age group with DCD compared to the other groups According to these findings, it appeared that younger children with DCD needed a high level of pre-programming to compensate for difficulties in perceptual-motor and executive functions of movement related to coordination disorders No significant difference was found between the groups according to EEG findings for interhemispheric connections
Tsai et al. (35), Dev Med Child Neurol, 2012, Taiwan	DCD: 24 Age: 8-16 years (11.4±2.1) TDC: 30 Age: 8-16 years (11.8±2.7)	DCD: MABC-2 ≤5th percentile Not being diagnosed as having autism or ADHD	EEG	It was found that children with DCD had a prolonged reaction time and EEG activation was lower than TDC, and it was observed that children with DCD made less effort in the evaluation and response process and used less neural resources to compensate for mistakes

Table 1. continued

First author, journal, year, country	Participants (number, age range, mean age)	Inclusion and exclusion criteria	Neuroimaging technique	Neuroimaging results
Pangelinan et al. (36), J Neurophysiol, 2013, France	DCD: 14 Age: 6-12 years (10.2±2.1) TDC: 20 Age: 6-12 years (9.8±2.2)	DCD: MABC-2 ≤5th percentile The patients whose dominant hand was found to be the right hand according to the Fagard and Corroyer 2003 Handedness test TDC: MABC2 ≥ 25th percentile Those who did not have developmental, neurological and learning problems and were right-handed	EEG	Younger children (6-8 years) with DCD had less cortical activation areas and lower EEG frequency amplitudes. Compensatory cortical activation areas were found to be more and EEG frequency amplitudes higher in older patients with DCD (10-12 years) It has been determined that this behavioral change is to show better motor performance
Fong et al. (41), Medicine (Baltimore), 2016, China	DCD: 86 Age: 6-10 years (7.9±1.7) TDC: 99 Age: 6-10 years (7.4±1.6)	DCD: MABC-2 ≤5th percentile Bruininks-Oseretsky Test of motor proficiency ≤42	EEG	EEG values of children with DCD were found to be lower than the TDC group
Blais et al. (44), Dev Sci, 2018, France	DCD: 10 Age: 12-16 years (13.49±1.76) TDC: 10 Age: 12-16 years (13.47±1.39)	DCD: MABC-2 ≤5th percentile Not having a diagnosis of ADHD according to the DSM-5 criteria	EEG	According to the EEG findings, the intrahemispheric connections of both groups were found to be similar, while the number of interhemispheric connections of the DCD group was found to be lower than the TDC group
Koch et al. (49), Exp Brain Res, 2018, Portugal	DCD: 10 Age: 8-12 years (9.9±1.2) TDC: 10 Age: 8-12 years (10.01±1.1)	DCD: MABC-2 ≤5th percentile TDC: MABC2 ≥25th percentile Those who did not have developmental, neurological or learning problems	NIRS	Dorsolateral prefrontal cortex activation was found to be higher in children with DCD compared to TDC
Caçola et al. (50), Int J Dev Neurosci, 2018, USA	DCD: 10 Age: 8-12 years (8.46±0.97) TDC: 10 Age: 8-12 years (8.22±0.86)	DCD: MABC-2 ≤5th percentile TDC: MABC2 ≥25th percentile Those who did not have developmental, neurological or learning problems	fNIRS	According to fNIRS findings, it was determined that there was more activation in the primary motor cortex and premotor cortex areas in the TDC group compared to the DCD group, both contralateral and ipsilatereral In addition, it was observed that the activated areas in the DCD group were mostly in the ipsilateral direction

DCD: Developmental coordination disorder, TGC: Typically developing children, ADHD: Attention-deficit/hyperactivity disorder, ASD: Autism spectrum disorder, MABC: Movement Assessment Battery for Children, DSM: Diagnostic and Statistical Manual of Mental Disorders, fMRI: Functional magnetic resonance imaging, MRI: Magnetic resonance imaging, DTI: Diffusion tensor imaging, EEG: Electroencephalogram, NIRS: Near-infrared spectroscopy fNIRS: Functional near-infrared spectroscopy, DD: Developmental dyslexia

Functional Near-infrared Spectroscopy

Although few fNIRS studies have been conducted on children with DCD, it is seen that there are differences in interhemispheric connections, and neural connections are more ipsilateral in children with DCD compared with those with typical development (49,50).

Conclusion

Advanced neuroimaging techniques provide a better understanding of the neural connections in DCD and detailed information about brain structure and function. However, due to the small sample sizes in the studies, no clear result could be reached to explain the underlying mechanism. The limitations of the review are that one study was in French and full texts of two studies were not available and thus could not be included. There is no consensus on the neuroradiology of DCD in the literature because the results of the studies discussed in the review vary. It is also necessary to conduct multi-center studies that better demonstrate the relationship between these different radiologic findings and clinical findings. With these studies, it is thought that the most effective methods for the treatment of DCD can be determined by guiding neuroimaging studies, physiotherapy/occupational therapy, and other intervention methods.

Ethics

Peer-review: Externally peer-reviewed.

Authorship Contributions

Design: C.Y., G.A., M.G.P., Data Collection or Processing: C.Y., G.A., Analysis or Interpretation: C.Y., G.A., M.G.P., E.M., R.K., C.G.Y., Literature Search: E.M., R.K., C.G.Y., Writing: C.Y., G.A., M.G.P., E.M., R.K., C.G.Y.

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