


Word-Finding Difficulties as a Prominent Early Finding in a Later Diagnosis of Attention Deficit Hyperactivity Disorder

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Abstract

Objective Attention deficit hyperactivity disorder (ADHD) is a common neuropsychological disorder primarily diagnosed in childhood. Early intervention was found to significantly improve developmental outcomes, implicating on the role of early identification of ADHD markers. In the current study, we explored the developmental history of children referred to neurological assessment to identify early ADHD predictors.

Methods A total of 92 children and adolescents (41 females) recruited at a pediatric neurology clinic, with suspected ADHD ($n = 39$) or other neurological difficulties ($n = 53$) such as headaches, seizures, tic disorders, orthostatic hypotension, postischemic stroke, intermittent pain, and vasovagal syncope. Developmental history information was obtained from caregivers, and evaluation for possible ADHD was performed. Developmental details were compared between children with and without current ADHD diagnosis.

Results Word-finding difficulties (WFDs) in preschool age was reported in 30.4% of the sample. Among children diagnosed with ADHD, 43% had WFDs history, compared with only 5% in children without ADHD. Among children with WFDs history, 93% were later diagnosed with ADHD compared with 42% in children without WFDs history. The relationship between WFDs and ADHD was significant (chi-square test [$1, N = 92$] = 20.478, $p < 0.0001$), and a logistic regression model demonstrated that besides from a family history of ADHD, the strongest predictor for ADHD in school age children was a history of WFDs.

Conclusion Preliminary evidence supports a predictive link between preschool WFDs and later ADHD diagnosis, highlighting the importance of early WFDs clinical attention.

Keywords

- ▶ attention-deficit hyperactivity disorder
- ▶ word-finding difficulty
- ▶ language
- ▶ disability
- ▶ child development

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Introduction

Attention deficit hyperactivity disorder (ADHD) is a common neuropsychological disorder with frequent comorbidities, such as conduct disorder, depression, anxiety, learning disabilities, and speech problems.¹ According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR)² diagnostic criteria, presenting signs of ADHD before the age of 12 years is mandatory. ADHD is usually diagnosed in school-age children, despite symptoms appearing earlier in many cases.³ Based on a recent systematic review, the mean age of diagnosis in European countries was 6.2 to 18.1 years, albeit the age of onset ranged between 2.25 and 7.5 years. Thus, diagnosis of ADHD is often delayed.⁴ Since one of the critical functions of primary care is to recognize the symptoms of an illness at an early stage, and since early ADHD diagnosis enables effective treatment strategies implementation, identification of early prognostic markers might be crucial.⁴

It has been suggested that ability to prospectively predict the onset and persistence/remission of ADHD might facilitate a more personalized approach to intervention.⁵ Thus, in the current study, we aimed to focus on clinical setting, in an attempt to identify ADHD predictors in clinically referred children, by exploring their developmental history. Selecting a clinical comparison group of children referred for neurological assessment is pivotal to replicate clinical observations, especially in cases where differential diagnosis could be ambiguous.

The delay between symptom onset and diagnosis might reflect a difficulty in diagnosing ADHD at younger ages. Recently, it has been suggested that ADHD can be diagnosed reliably at preschool age, where hyperactivity and impulsivity are the most prominent symptoms, and some neurocognitive deficits associated with the disorder are already detectable.⁶ However, ADHD diagnostic criteria do not address preschoolers separately and have been suggested to be inaccurate when diagnosing this population.⁷ From a developmental perspective, it is often hard to determine whether a young child's inattentive, hyperactive, and/or impulsive behavior reflects variability in typical development or is in fact a clinical symptom of ADHD.⁷

Albeit the difficulty in establishing diagnosis in preschoolers, early diagnosis of ADHD is essential for earlier intervention, which can be crucial for a better prognosis.^{8,9} Indeed, a recent meta-analysis addressing ADHD revealed significant postintervention reductions in ADHD symptoms with early diagnosis.⁵ Early diagnosis is crucial as early treatment may modify neuronal connections and improve symptoms.¹⁰ From the neurodevelopmental perspective, the rationale proposing a better prognosis following early intervention is clear: early childhood seems to be a period of greater neuroplasticity, thus allowing appropriate reception of intervention.¹¹ Earlier interventions may rewire connections in the developing brain leading to more fruitful, long-lasting effects.⁸ Early interventions have a well-founded rationale regarding potential confounders' prevention⁹ such as comorbid disorders, low self-esteem, and challenging relationships with family members. Thus, regarding the

benefits of early intervention, identifying "at-risk" children for ADHD might impact the child's emotional, academic, and social life by moderating the severity of their ADHD.⁸

ADHD has a strong hereditary component, denoting an increased risk in families of individuals with ADHD. In a meta-analysis aggregating data from 102 studies, the estimated heritability of ADHD in children younger than 12 years reached 75%.¹² A few more early markers have already been identified, including poor neurocognitive and executive functions,¹³ delays or deficits in qualitative features of motor development (fine and gross),^{14,15} temperamental activity, and vocabulary delay.¹⁶

Findings from a recent meta-analysis indicate that multiple neurocognitive and behavioral alterations are involved in the early development of ADHD, with the most significant effect sizes found for sensory processing, activity level, and aspects of executive function (inhibition, flexibility, planning/organization, intraindividual variability, impulsivity, and global executive function).⁶ However, it may be suggested that these areas reflect (or are similar to) ADHD symptoms, and a broader perspective, including focus on earlier developmental symptoms, is still needed. Indeed, in a meta-analysis, ADHD was significantly associated with poorer general cognitive, language, and motor abilities; social and emotional difficulties; early regulatory and sleep problems; and sensory processing difficulties in the first 5 years of life. Further findings regarding narrative synthesis indicating early alterations in brain structure and resting-state neurophysiological activity call for attention to earlier stages of language development.¹⁷

A domain that is of special interest when seeking for early markers for ADHD is language proficiency. Language is assumed to play a substantial role in the development of regulatory skills, providing psychological tools needed to master behavior and cognition.¹⁸ It has been suggested that early self-regulation skills play a particularly important role for vocabulary development in preschool.¹⁹ Children with lower language abilities as toddlers (6–24 months) exhibited impaired executive and regulative skills at kindergarten age (4–5 years).²⁰ Since ADHD is often referred to as a regulatory problem,²¹ it might be suggested that impaired language abilities are a significant predictor for ADHD. Indeed, speech and language development delay at 9 to 18 months were found among others to be a predictor of ADHD at preschool.¹⁴ Furthermore, poor language skills (i.e., phonology, syntax, lexicon, and conceptual knowledge) at the age of 3 years predicted inattention/hyperactivity symptoms at the beginning of primary school.²² More recently, in data from 9,021 children, early markers of later ADHD diagnosis included fine motor delay at 18 months, high temperament at 24 months, and difficulties in various aspects of language development such as speech delay at 24 months and grammar difficulties in early school years. Notably, a high polygenic risk increased the impact of these markers, in addition to being an independent early marker for ADHD.¹⁷ Children with ADHD were found to have higher rates of pragmatic language difficulties, with specific difficulties with inappropriate initiation, presupposition, social discourse, and narrative coherence.²³

Thus, it has been suggested that detection of language delay in early communication warrants follow-up of the child's development of self-regulation,²⁰ and might be of significant interest when seeking predictors for ADHD.

A specific aspect related to both language ability and executive functions is word-finding difficulties (WFDs).²⁴ WFDs occur when a child is unable to produce words despite having an understanding of their meaning.²⁴ In learning a language, the child must be able to focus on relevant linguistic information selectively and naturally ignore irrelevant information. In ADHD, the deficits in executive attention and working memory might end up negatively influencing speech and language development in the early years of life, which are skills that depend on phonological awareness and therefore affect the cognitive processes of language.²⁵ Among preschoolers receiving speech therapy, about a quarter, are reported to have WFDs,²⁶ although verbal fluency was not found to predict ADHD symptoms.²² Thus, WFDs represent a specific measure that warrants further research.

As noted in the literature,²⁷ the common association of ADHD primarily with hyperactivity has resulted in underdiagnosis within certain populations. The increased likelihood of occurrence in specific demographic groups underscores the importance of practitioner awareness. The presence of overlapping symptoms can complicate precise diagnosis and treatment, underscoring the significance of identifying and addressing concurrent conditions. The ability to identify high-risk groups empowers practitioners to maintain heightened vigilance concerning potential ADHD cases.

Typically, when seeking to identify early signs of ADHD, the conventional approach involves comparing individuals with ADHD to the general population. However, to find signs that are specific to ADHD and not just caused by different neurological issues, it is important to compare the development of children with ADHD with those who have other neurological problems. This helps us identify traits that are unique to ADHD and not just part of the broader range of neurological differences. By focusing on these specific traits, we can get better at recognizing and diagnosing ADHD more precisely. This targeted approach aligns with the notion of recognizing high-risk groups, enabling practitioners to be more effective in identifying potential cases of ADHD within those populations.

In this focused study, we addressed the utilization of WFDs in ADHD prediction. The novelty in our work lies in the easy-to-detect predictor and the comparison with a clinical sample of population with other neurological conditions. If confirmed, our hypothesis implies that inquiring about WFDs in a basic parent-report screening questionnaire might contribute to the identification of children at risk of developing ADHD, thus allowing for early treatment at a better overall prognosis.

Methods

Procedure

The present study was performed at the Child Neurology Unit at a tertiary center that provides services to a large

population of Jewish and Arab citizens, the majority being middle-class families. Ethics approval was obtained from the hospital's review board. As part of the screening process of patients referred to the clinic, parents were invited to participate in the current study. Parents were asked to fill a developmental history questionnaire. A neurological assessment was administered by a trained and experienced pediatric neurologist, independent of the reported questionnaire.

Participants

A total of 92 children and adolescents (41 females, 51 males; aged 6–18 years, mean age 11.51 years, standard deviation = 3.59) were participants. Sixteen children were Muslim Arabs. All patients were recruited at the Child Neurology Unit, of whom 39 had suspected ADHD and 53 suffered from other neurological conditions (27 with headaches, 18 with verified/suspected seizures, 4 with tic disorders, 1 with orthostatic hypotension, 1 postischemic stroke, 1 with intermittent pain, and 1 with vasovagal syncope). All children referred to the clinic were included in the study, excluding children with intellectual disability or autism spectrum disorder.

Measures

Developmental History

A detailed screening parental report questionnaire regarding developmental history, similar to a developmental and behavioral development intake, was developed for use in the current study. Parents were asked to report in a binary fashion if their child had experienced difficulties in the areas of perinatal history, neurodevelopmental history, motor development (fine and gross), language development (e.g., speech delay, WFDs), learning difficulties (LDs) (in reading, writing, or arithmetic), history of ADHD in a first-degree relative, hypersensitivity to touch or sound, breathing difficulty during sleep, and difficulties with social interactions. Language development difficulties were verified using patients' medical records of WFD diagnosis obtained from initial speech therapist evaluations at the child development center in our medical center. WFDs were not specifically sought but were rather part of the speech therapist's findings. Nevertheless, the children included in the study had all depicted WFD as the major finding at preschool-age evaluation.

ADHD screening was based both on the parental report regarding inattention and hyperactivity/impulsivity symptoms describing on the DSM-5 criteria ADHD,²⁸ as well as a clinical ADHD evaluation by a trained neurologist, based on the DSM-5 criteria for ADHD,²⁸ and the short Conner's ADHD rating scale.²⁹

Statistical Methods

Analyses were conducted using IBM SPSS Statistics 25 program. First, we explored the proportions of early WFDs and ADHD diagnosis. Next, a chi-square test of independence was performed ($\alpha=0.05$), to explore the association between WFDs and ADHD. Then, a logistical regression model was employed between WFDs and ADHD, with ADHD as the

dependent variable. Additional dependent variables were added to the model as confounders including speech delay, family history of ADHD, learning disabilities, social difficulties, hypersensitivity, and motor difficulties (fine and gross). The association of each dependent variable and ADHD was examined using a chi-square test. Significantly associated variables were included in the logistic regression model.

Results

The study cohort's characteristics among participants with and without ADHD are presented in ► **Table 1**.

Of the 92 participants, 30.4% ($n=28$) of the sample were reported to have had a history of WFDs. Among participants with a history of WFDs, 93% were diagnosed with ADHD ($n=26$). Among patients without a reported history of WFDs ($n=64$), only 42.2% received an ADHD diagnosis ($n=27$). Correlations of participant's characteristic and developmental history with ADHD diagnosis are presented in ► **Table 2**.

About 57.6% of the participants ($n=53$) were diagnosed with ADHD. Of these cases, 43% were found to have a history of WFDs, whereas among individuals without ADHD ($n=39$), a history of WFDs was found only in 5%.

Chi-square test of independence examining the association between WFDs and ADHD was found to be significant (chi-square test [$1, N=92$] = 20.478, $p < 0.0001$), indicating that children with WFDs in preschool age were more likely to receive diagnosis of ADHD.

Next, the association between WFDs and ADHD was explored using a logistic regression model. Speech delay, family history of ADHD, learning disabilities, social impairments, hypersensitivity, and motor difficulties (fine and gross) were explored as confounders. A significant association was found between ADHD and a first-degree relative diagnosed with ADHD, gross and fine motor difficulties, and hypersensitivity to touch and learning disabilities. Other confounders including general speech delay, breathing difficulty during sleep, and social impairments were not significantly associated with ADHD. Chi-square values and Fisher's

exact test (p -values) for each analysis are presented in ► **Table 3**.

The logistic regression model was significant (chi-square test = 58.792, $p < 0.001$), and explained 56.6% (average of Nagelkerke R-square and Cox and Snell R-square) of the variance in ADHD diagnosis. The model correctly classified 79.8% of cases. The model indicated family history of ADHD as the strongest predictor, as it seems to increase the odds by a measure of 8.6 for an ADHD diagnosis (odds ratio [OR] = 8.612, $p < 0.05$). The second strongest predictor according to the model was WFDs in preschool age, as children reported to have WFDs in preschool age were 8.33 times more likely to have a later ADHD diagnosis (OR = 8.33, $p < 0.05$). Other confounders identified as significant predictors of ADHD were learning disabilities and fine motor difficulties. Gross motor difficulties and hypersensitivity to touch or sound were not found to significantly predict an ADHD diagnosis. Odds ratios and p -values of the logistic regression model are presented in ► **Table 4**.

Discussion

In the present study, we examined the association between developmental history in preschool age and later ADHD diagnosis. Specifically designed for the study, a simple parental report questionnaire was used, similar to a common developmental and behavioral development screening intake. ADHD diagnosis was verified independently, by a trained pediatric neurologist. WFDs in preschool children later diagnosed with ADHD were found to be second to a family history of ADHD as the strongest predictor of a later ADHD diagnosis.

It is important to note that both examined groups in our study consisted of children referred to our outpatient clinic. The ADHD group comprised otherwise healthy children suspected of having ADHD. To ensure a relevant comparison, we juxtaposed them with children referred for noncognitive-affecting neurological problems such as headaches, tics, and essential tremors. This control group matched the ADHD

Table 1 The cohort's characteristics

	ADHD		No ADHD	
	N	%	N	%
Speech delay	12	22.6	3	7.7
Family history of ADHD	45	84.9	18	46.2
Learning disabilities	34	64.2	5	12.8
Social difficulties	16	30.2	6	15.4
Hypersensitivity	31	58.5	10	25.6
Gross motor difficulties	11	20.8	1	2.6
Fine motor difficulties	23	43.4	3	7.7
Hearing impairment	4	7.5	1	2.6
Sleep disorders	16	30.2	9	23.1

Abbreviation: ADHD, attention deficit hyperactivity disorder.

Note: $N=92$ ($n=53$ for ADHD condition and $n=39$ for no ADHD condition).

Table 2 Correlations of participant’s characteristic and developmental history with ADHD diagnosis

Variable	1	2	3	4	5	6	7	8	9	10	11
Demographic variables											
Gender	1	0.249 ^a	0.16	0.165	0.277 ^b	0.098	0.038	0.005	0.56	0.152	0.223 ^a
Age	0.249 ^a	1	-0.219 ^a	-0.22 ^a	-0.159	-0.124	-0.181	-0.065	-0.252 ^a	-0.121	-0.204
Study variables											
ADHD diagnosis	0.16	-0.219 ^a	1	0.472 ^b	0.2	0.455 ^b	0.508 ^b	0.178	0.327 ^a	0.267 [*]	0.392 ^b
Word-finding difficulties	0.165	-0.22 ^a	0.472 ^b	1	0.411 ^b	0.27 ^a	0.337 ^b	0.139	0.215 ^a	0.095	0.162
Speech delay	0.277 ^b	-0.159	0.2	0.411 ^b	1	0.08	0.214 ^a	0.164	0.137	0.179	0.18
Family history of ADHD	0.098	-0.124	0.455 ^b	0.27 ^a	0.08	1	0.305 ^b	0.079	0.146	0.029	0.135
Learning disabilities	0.038	-0.181	0.508 ^b	1	0.214 ^a	0.305 ^b	1	0.154	0.242 ^a	0.253 ^a	0.19
Social difficulties	0.005	-0.065	0.178	0.139	0.164	0.079	0.154	1	0.069	0.027	0.17
Hypersensitivity	0.56	-0.252 ^a	0.327 ^a	0.215 ^a	0.137	0.146	0.242 ^a	0.069	1	0.107	0.311 ^b
Gross motor difficulties	0.152	-0.121	0.267 ^a	0.095	0.179	0.029	0.253 ^a	0.027	0.107	1	0.33 ^b
Fine motor difficulties	0.223 ^a	-0.204	0.392 ^b	0.162	0.18	0.135	0.19	0.17	0.311 ^b	0.33 ^b	1

Abbreviation: ADHD, attention deficit hyperactivity disorder.

Note: To calculate correlations between categorical variables, Cramer’s V measure was used. To calculate correlations between continuous and categorical variables, a point-biserial correlation was conducted.

^ap < 0.05.

^bp < 0.01.

Table 3 Chi-square values and *p*-value (Fisher's) for the variables as ADHD predictors

Variable	Chi-square test	Significance (Fisher's exact test)
WFDs	20.478	0.000 ^a
ADHD in family	18.636	0.000 ^a
Learning disabilities	23.499	0.000 ^a
Fine motor difficulties	14.127	0.000 ^a
Hypersensitivity	9.814	0.003 ^b
Gross motor difficulties	6.555	0.012 ^b
Social difficulties	2.877	0.137
Speech delay	3.679	0.85

Abbreviations: ADHD, attention deficit hyperactivity disorder; WFDs, word-finding difficulties.

^a*p* < 0.001.

^b*p* < 0.05.

Table 4 Logistic regression, odds ratio, and *p*-values of ADHD predictors

Variable	Odds ratio	Significance
ADHD in the family	8.612	0.013 ^a
Word-finding difficulties	8.33	0.014 ^a
Fine motor difficulties	7.253	0.042 ^a
Learning disabilities	4.395	0.041 ^a
Gross motor difficulties	6.153	0.210
Hypersensitivity	2.537	0.164

Abbreviation: ADHD, attention deficit hyperactivity disorder.

^a*p* < 0.05.

group in terms of demographics, ages, and sociocultural backgrounds. Given the study's emphasis on the early childhood history of WFD, unaffected by subsequent neurological conditions, this selection of a comparison group was considered suitable for maintaining study integrity.

The identification of ADHD in immediate family members as a significant predictor of ADHD is not surprising in view of the well-established notion of ADHD as an inherited, gene-based condition.^{12,30} Nonetheless, current findings are crucial in specifically suggesting WFDs in preschool age as a significant predictor of an ADHD diagnosis in school-age children. Current findings are also important in comparing children with ADHD with a group of children with other neurological difficulties, enabling us to explore specific characteristics of ADHD that are not expression of a general neurological diversity.

Interestingly, reports of a general speech delay, referral to speech therapy, or communication difficulties were found to be relevant to WFDs rather than a later diagnosis of ADHD. These results are relevant in implying the need to address a history of WFDs in the routine screening for developmental history in cases suspected of ADHD. This finding is probably neither a result of dysfluency nor associated with fluency speed, since we focused on WFDs in everyday life rather than word retrieval speed on a standard test. However, it may be possible that WFDs is influenced by difficulties in executive functions needed in the application of strategy to find words or

in inhibition of thoughts, such as in the cases of inattention or when losing train of thoughts. Further research is still needed to explore this suggestion, using specific measures to address various executive functions in young children as well.

Regarding hypersensitivity, a confounder found significantly related to ADHD based on a chi-square test did not prove to be a predictor of ADHD. Albeit the inconsistent association, it may be suggested that this relationship may be understood using Dunn's model of sensory processing.³¹ Based on this model, young children who are hypersensitive to stimuli due to low thresholds and who act in response to those thresholds tend to be hyperactive or distractible.

Concerning the motor domain, only fine motor difficulties were found to predict ADHD. This difference is consistent with previous findings suggesting that different behavioral processes are involved in fine and gross motor performance, based on the finding that attention and impulse control predicted both fine and gross motor skills in children with ADHD, whereas activity level predicted gross (but not fine) motor.³²

On a similar note, we found LDs to significantly predict ADHD. This relation is in line with the known high comorbidity between these conditions, remarkably reaching 45.1%.³³ However, such evidence of co-occurrence might not reflect a predictive association, and it may be that ADHD and LDs are two distinct conditions that do not result from one another.³⁴ Some similarities between ADHD and LD symptoms exist, culminating in a child receiving both diagnoses.³⁵

The present findings emphasize the importance of closely monitoring young children with WFDs. It may be suggested that clinical attention early through development might contribute to early identification and possibly intervention for children with ADHD. Clinical attention to a symptom such as WFD is applicable even in regular medical/pediatric visits or during developmental screening at the family health center. Hence, early identification plays a crucial role in guiding professionals to direct their clinical attention toward these symptoms. Notably, research indicates that interventions such as phonology and semantics training yield improvements in diverse educational outcomes.³⁶

Early identification holds significant value, not just from these findings but also in how psychologists can apply them effectively. Furthermore, comprehending the WFD-ADHD link aids psychologists in addressing language issues and potential ADHD risks. Collaborating well with parents and educators facilitates insight-sharing and supportive environments. By addressing both the language-related challenges and the underlying ADHD characteristics, a more comprehensive and nuanced approach to intervention and support can be achieved, ultimately enhancing the developmental trajectory of these children.

The main limitation of our study is the use of a parent-report questionnaire which may be subject to potential report biases. Furthermore, a major drawback is the long retrospective report, asking parents to report their child's history several years later in his/her development. Albeit inquiry into developmental history is a common clinical practice more than several years after the fact, the accuracy of report might be low and is subjected to recall bias. To preserve similarity to common clinical practice, in the current study, we collected information while asking to report on very prominent behavioral/developmental characteristics, such as specific symptoms (e.g., WFDs) or referral to therapy. In view of these limitations, a prospective longitudinal follow-up along with direct and standardized assessment of children with WFDs is needed to support our findings. Furthermore, as commonly seen in the clinical setting, many children in the current sample had comorbid complaints. These comorbidities could potentially play a role in the predictive relationship found between WFDs and a later ADHD diagnosis. Our small sample limited our ability to explore these questions. Future studies might contribute in our exploration of the association between WFDs and ADHD common comorbidities, such as specific learning disabilities with reading, writing, or arithmetic. In addition, future direction might include more heterogeneous and specific comparison groups, to enable not only to compare and identify characteristics that are associated with ADHD compared with general neurological complaints and diversity but also to differ among specific conditions such as ADHD versus brain injury, postconcussion, headaches, or stroke.

Conclusion

Our study aimed to explore predictors for ADHD based on screenings within the clinical setting. Albeit suggesting only preliminary evidence for the predictive relationship between early WFDs and later diagnosis of ADHD, our observations

suggest that early clinical markers of ADHD might be possible using simple and inexpensive means. Detecting such markers might help identify young children at risk for a later diagnosis of ADHD and thus, allowing for earlier intervention. Although WFDs were retrospectively reported by parents, we found that they constituted a stronger predictor for ADHD than other factors examined. Further research is needed to confirm these findings and support the value of using WFDs as an early marker of ADHD.

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Conflict of Interest

None declared.

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