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Diagnostic Yield of EEG and Cranial MRI in Isolated Speech Delay and Longitudinal Outcomes: A Single-center Retrospective Cohort

İzole Konuşma Gecikmesinde EEG ve Kraniyal MRI'nın Tanısal Katkısı ve Uzun Dönem Sonuçları: Tek Merkezli Retrospektif Kohort

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Abstract

Objective: To evaluate the diagnostic yield of electroencephalography (EEG) and cranial magnetic resonance imaging (MRI) in children with isolated speech delay (ISD) and to identify clinical factors predicting long-term outcomes.

Methods: Children aged 0-9 years presenting with ISD between January 2020 and January 2025 were retrospectively reviewed. Patients with neurological, psychiatric, or hearing disorders were excluded. EEG and MRI findings were categorized as normal or abnormal. Groups were compared across three age strata (0-2, 2-4, and ≥4 years). Statistical analyses included chi-squared tests, trend tests, and multivariable logistic regression.

Results: Among 245 children (72.2% male), EEG background slowing was detected in 6.25%, 4.82%, and 1.52% of 0-2, 2-4, and ≥4-year groups, respectively (p=0.354; trend p=0.161). Interictal epileptiform discharges increased with age (7.29%, 13.25%, and 18.18%; p=0.109; trend p=0.035). MRI abnormalities were found in 8.33%, 6.02%, and 7.58% (p=0.837). Consanguinity was observed in 13.9% of all cases. Intellectual disability developed in 0%, 2.41%, and 16.67% of the age groups, respectively (p<0.001). Logistic regression confirmed an age-related effect (odds ratio=9.75; 95% confidence interval, 2.40-39.59; p=0.001).

Conclusion: In ISD with otherwise normal development, MRI has low diagnostic yield, while EEG abnormalities-though more frequent with age-are not independent predictors of adverse outcomes. Persistent ISD at ≥4 years significantly increases the risk of later intellectual disability, underscoring the need for longitudinal monitoring and targeted evaluations.

Keywords: Isolated speech delay, diagnostic yield, neurodevelopmental outcomes, longitudinal follow-up

Öz

Amaç: İzole konuşma gecikmesi (İKG) olan çocuklarda elektroensefalografi (EEG) ve kraniyal manyetik rezonans görüntülemenin (MRI) tanısal değerini değerlendirmek ve uzun dönem sonuçları öngören klinik faktörleri belirlemek.

Yöntem: Ocak 2020-Ocak 2025 arasında İKG ile başvuran 0-9 yaş arası çocuklar retrospektif olarak incelendi. Nörolojik, psikiyatrik veya işitsel bozukluğu olan hastalar dışlandı. EEG ve MRI bulguları normal veya anormal olarak sınıflandırıldı. Gruplar üç yaş kategorisinde (0-2, 2-4 ve ≥4 yaş) karşılaştırıldı. İstatistiksel analizlerde ki-kare testi, trend testleri ve çok değişkenli lojistik regresyon kullanıldı.

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Öz

Bulgular: Toplam 245 çocuğun (%72,2 erkek) EEG'de arka plan yavaşlaması; 0-2, 2-4 ve ≥4 yaş gruplarında sırasıyla %6,25, %4,82 ve %1,52 oranlarında tespit edildi (p=0,354; trend p=0,161). Yaşla birlikte artan interiktal epileptiform deşarjlar sırasıyla %7,29, %13,25 ve %18,18 oranlarında izlendi (p=0,109; trend p=0,035). MRI anormallikleri ise sırasıyla %8,33, %6,02 ve %7,58 oranlarında saptandı (p=0,837). Tüm olguların %13,9'unda akraba evliliği mevcuttu. Zihinsel yetiyitimi yaş gruplarında sırasıyla %0, %2,41 ve %16,67 olarak bulundu (p<0,001). Lojistik regresyon, yaşa bağlı etkinin anlamlı olduğunu doğruladı (olasılık oranı=9,75; %95 güven aralığı, 2,40-39,59; p=0,001).

Sonuç: Normal gelişim gösteren İKG olgularında MRI'nın tanısal katkısı düşüktür. EEG anormallikleri yaşla artsa da olumsuz sonuçların bağımsız bir göstergesi değildir. Dört yaş ve sonrasında devam eden konuşma gecikmesi, ileride zihinsel yetiyitimi gelişme riskini anlamlı şekilde artırmaktadır. Bu durum, bu çocuklarda uzun dönemli izlem ve hedefe yönelik değerlendirmelerin önemini vurgulamaktadır.

Anahtar Kelimeler: İzole konuşma gecikmesi, tanısal değer, nörogelişimsel sonuçlar, uzun dönem izlem

Introduction

Speech and language delay is a common reason for referral to pediatric services and has heterogeneous etiologies. "Speech" refers to the motor act of sound production, whereas "language" denotes the symbolic system for communication. Clinically, the two often co-occur. Population prevalence spans a wide range (≈1%-12.6%), rising to 3%-20% in school-age children[1,2]. Although some "late talkers" catch up, persistence beyond 4 years is more often associated with lasting impairment or an underlying organic/neurologic condition[3]. Isolated speech delay (ISD) refers to a delay in expressive speech/language development in a child who otherwise demonstrates age-appropriate global development and normal neurological examination, with no evidence of hearing impairment or neuropsychiatric comorbidity such as autism spectrum disorder, and without language regression or clinically suggestive seizure history[1]. In children presenting with ISD in the absence of red flags, most recommendations discourage routine electroencephalography (EEG) and neuroimaging because of their low diagnostic yield and limited impact on management. Conversely, guideline-based evaluations emphasize targeted investigations when specific clinical features are present, including regression, abnormal neurological examination findings, focal deficits, abnormal head growth (microcephaly/macrocephaly), or suspected global developmental delay or intellectual disability (ID). In such contexts, EEG (preferably including sleep) may be indicated when seizures or epileptic encephalopathy are suspected, whereas cranial magnetic resonance imaging (MRI) is recommended primarily when neurological signs or broader developmental concerns exist[6]. In ISD without regression, seizure history, or abnormal neurological examination, the clinical value of routine EEG has been considered limited[4].

Conversely, when global developmental delay/ID is suspected or when focal neurological signs, regression, or abnormal head growth are present, neuroimaging and further work-up are recommended[5]. In healthy children, the prevalence of incidental interictal epileptiform discharges (IEDs) is about 2%-6.5%[6-8], and EEG background slowing is often non-specific[8]. Incidental cranial MRI findings in children can reach 16%-20% in large series, with a smaller subset requiring clinical action[9,10]. We aimed to compare the yield of EEG and MRI across age strata in individuals with ISD who had otherwise normal baseline evaluations, and to report the frequency of ID during longitudinal follow-up.

Materials and Methods

Participants

In this retrospective cohort study, children aged 0-9 years who presented to a tertiary pediatric neurology clinic between January 2020 and January 2025 with ISD were included. Age at presentation defined three strata: 0-2, 2-4, and ≥4 years. Inclusion required a normal neurological examination, a normal audiology, a child psychiatry review confirming the absence of comorbid diagnoses such as autism spectrum disorder or selective mutism. Exclusion criteria included neurological conditions expected to cause global developmental delay/ID, overt structural or organic etiologies, and abnormal neurological examinations. Clinically overt delays (e.g., only single words at 18 months; ≤3 words at 2 years) and borderline cases were corroborated with Denver II. Standardized data abstraction captured demographics, EEG (background slowing, IED presence), cranial MRI (normal vs. abnormal), consanguinity, family history of late talking, and follow-up outcomes. The primary longitudinal outcome was ID; according to the clinical protocol, microcephaly, autism, and global developmental delay were also screened during follow-up. The study was

approved by the İzmir Bakırçay University Non-Interventional Clinical Research Ethics Committee (approval no: 344/324, date: 06.10.2021).

Statistical Analysis

Categorical variables were compared with Pearson's chi-squared test or Fisher's exact test when expected counts were small. Given the ordered age strata, linear trend was evaluated using the Cochran-Armitage test. Continuous variables were summarized as mean ± standard deviation or median (min-max), as appropriate. The primary outcome was ID during follow-up. Multivariable binary logistic regression was used to assess independent predictors, with covariates coded as follows: age group (ordinal: 0-1-2), EEG-IED (0/1), consanguinity (0/1), and cranial MRI abnormality (0/1). Two-sided p<0.05 was considered statistically significant.

Results

Demographics

The cohort comprised 245 children; 72.2% were male (n=177). Age strata were: 0-2 y (n=96; 39.2%), 2-4 y (n=83; 33.9%), and ≥4 y (n=66; 26.9%). The mean follow-up duration was 3.2±1.7 year. The sex distribution did not differ by age group [Pearson $\chi^2(2)=0.385$, p=0.825; linear-by-linear $\chi^2=0.014$, p=0.905] (Table 1).

EEG/MRI Findings and Family Variables

Background slowing was observed in 6.25%, 4.82%, and 1.52% for 0-2, 2-4, and ≥4 years, respectively (overall p=0.354; trend p=0.161). IED frequencies were 7.29%, 13.25%, and 18.18% across age strata (overall p=0.109; trend p=0.035). MRI abnormalities occurred in 8.33%, 6.02%, and 7.58% of participants (p=0.837; trend p=0.808). Overall, Consanguinity was present in 13.9%, with no significant difference between age groups (p=0.130; trend p=0.100). The prevalence of family history of late talking was 7.3%, 6.0%, and 12.1% (p=0.371). Details appear in Table 2.

Table 1. Demographics and sex distribution by age group

Age group	n	Male, n (%)	Female, n (%)
0-2 years	96	70 (72.9)	26 (27.1)
2-4 years	83	58 (69.9)	25 (30.1)
≥4 years	66	49 (74.2)	17 (25.8)
Total	245	177 (72.2)	68 (27.8)

Statistics: Pearson $\chi^2(2)=0.385$, p=0.825; linear-by-linear $\chi^2=0.014$, p=0.905 for sex×age group (3×2)

Follow-up Outcome (ID) and Multivariable Analysis

ID during follow-up occurred in 0% (0-2 y), 2.41% (2-4 y), and 16.67% (≥4 y) (overall $p=7.1\times10^{-6}$; trend $p=8.0\times10^{-6}$). In multivariable logistic regression, the age-step effect was significant for ID [odds ratio (OR)=9.75; 95% confidence interval (CI), 2.40-39.59; p=0.001], whereas IEDs, consanguinity, and MRI abnormalities were not independent predictors. Model details are shown in Table 3.

Discussion

This study systematically evaluated EEG and MRI findings and age-stratified risk of subsequent ID in children with ISD who had a normal baseline clinical assessment. Our results align with the heterogeneous spectrum described for ISD (1-4), and the most prominent signal was an age-step effect: each step from 0-2 to 2-4 to ≥4 years markedly increased the adjusted odds of later ID (OR≈10). Clinically, persistent delay at ≥4 years constitutes an independent warning sign for lasting language-cognitive difficulties and warrants closer surveillance.

Our EEG results merit two complementary interpretations. First, given that incidental IEDs in healthy children occur in approximately 2%-6.5%[6-8], the significant linear increase in IED frequency with age (trend p=0.035) may partly reflect rising pre-test probability among older, persistently delayed referrals. Second, IEDs did not retain independent prognostic value in multivariable models, indicating that isolated IEDs are insufficient as a standalone predictor of adverse outcome; thus, EEG is better employed selectively, guided by clinical context[4,5]. When language regression, nocturnal events, or suspicion of seizures are present, sleep EEG may be informative; otherwise, routine EEG is unlikely to alter management. Background slowing was rare and largely non-specific, consistent with foundational EEG sources[8].

On neuroimaging, the diagnostic yield was low, and many findings were incidental or benign. Large pediatric series report incidental MRI findings in 16%-20% of children, with a smaller actionable subset[9,10]. Our rates were below that spectrum, supporting a symptom-driven, risk-targeted imaging strategy in ISD when the clinical examination is otherwise normal. Red flags such as abnormal head growth, focal neurological signs, regression, or epilepsy suspicion should prompt neuroimaging[5]; otherwise, routine MRI is unlikely to add value and may expose children to cost and potential sedation.

Consanguinity was present in 13.9% of the cohort and showed no independent association with ID in adjusted

Table 2. Clinical and paraclinical findings by age group

Variable	0-2 y (n=96)	2-4 y (n=83)	≥4 y (n=66)	p-value	Trend p-value
EEG-background slowing, n (%)	6 (6.25)	4 (4.82)	1 (1.52)	0.354	0.161
EEG-IED (any), n (%)	7 (7.29)	11 (13.25)	12 (18.18)	0.109	0.035
EEG-IED (frontal proportion), %	42.9	54.5	50.0	0.890	-
Cranial MRI-abnormal, n (%)	8 (8.33)	5 (6.02)	5 (7.58)	0.837	0.808
Consanguinity, n (%)	11 (11.5)	9 (10.8)	14 (21.2)	0.130	0.100
Family history of late talking, n (%)	7 (7.3)	5 (6.0)	8 (12.1)	0.371	-
Follow-up-intellectual disability, n (%)	0 (0.0)	2 (2.41)	11 (16.67)	$7.1x10^{-6}$	$8.0x10^{-6}$

EEG: Electroencephalography, MRI: Magnetic resonance imaging, IED: Interictal epileptiform discharge, p-value: Pearson's chi-squared or Fisher's exact, as appropriate, trend p-value: Cochran-Armitage test for linear trend across ordered age groups (0-2<2-4<≥4)

Table 3. Multivariable logistic regression (primary outcome: intellectual disability)

Model	Dependent variable	Covariates	OR	95% CI	p-value	n/events
A	Intellectual disability (follow-up)	Age group (0-2=0, 2-4=1, ≥4=2); EEG-IED (0/1); consanguinity (0/1); cranial MRI abnormal (0/1)	9.75	2.40-39.59	0.001	241/12

Logistic regression using binary coding of covariates (present=1/absent=0). ORs are adjusted for the listed covariates; for age group, the OR denotes a one-step increase

EEG: Electroencephalography, MRI: Magnetic resonance imaging, IED: Interictal epileptiform discharge, OR: Odds ratio, CI: Confidence interval

analyses. Considering that consanguinity prevalence in Türkiye ranges between 18% and 24%[11,12], family structure alone should not drive testing decisions; rather, phenotypic features and developmental trajectory should guide evaluation. Power limitations (modest event counts) may, however, have reduced the sensitivity for detecting small effects, underscoring the need for larger studies.

Practically, our findings support an age- and context-based algorithm: (i) 0-2 years-if no regression, seizure concern, or examination red flags are present, emphasize close follow-up and language-rich environmental input; (ii) 2-4 years-if risk signs are absent, continue surveillance and speech-language therapy; if present, consider targeted EEG (preferably sleep) and imaging; (iii) ≥4 years-persistent delay warrants broader differential diagnosis and prioritization of sleep EEG and phenotype-driven genetic evaluation. This approach may reduce unnecessary testing and focus resources on higher-risk subgroups[3-5,13].

Overall, our findings are consistent with existing recommendations that routine EEG and cranial MRI are unlikely to provide clinically meaningful diagnostic information in children with ISD who have a normal neurological examination and no regression or suspicion of seizures. In our cohort, MRI abnormalities were infrequent and largely incidental, which supports a symptom-driven imaging strategy rather than routine use. Although IEDs increased with age, EEG abnormalities did not independently predict subsequent ID, reinforcing the view that EEG should be reserved for cases with clinical suspicion of epilepsy, nocturnal events, or regression, with EEG ideally incorporating sleep recording. Importantly, our longitudinal data extend the guideline framework by highlighting a clear age-related risk signal: persistent ISD at ≥4 years was strongly associated with subsequent ID, suggesting that this subgroup may benefit from closer monitoring and broader targeted evaluation (e.g., developmental, genetic, and neuropsychological assessment) even when initial neurological examination is normal. Thus, our study supports current selective-testing recommendations while providing age-based risk stratification that may help refine follow-up intensity and evaluation priorities in persistent ISD.

Study Limitations

Limitations include a single-center, retrospective design and limited ID events, resulting in wide CIs. A minimum follow-up of one year may miss late-emerging outcomes. EEG recordings did not uniformly include sleep, and MRI-phenotype correlations were not available for all participants. Despite these constraints, the robustness and magnitude of the age-step association support its clinical relevance. Prospective, multicenter studies with standardized sleep EEG and comprehensive developmental/genetic assessments are needed to refine risk stratification in persistent ISD.

Conclusion

In children with isolated speech delay and otherwise normal baseline clinical findings, routine cranial MRI appears to have limited diagnostic value, and EEG abnormalities, although more frequent in older children, do not independently predict adverse neurodevelopmental outcomes. The most clinically relevant finding of this study is the strong age-related risk signal, indicating that speech delay persisting at or beyond 4 years of age is associated with an increased likelihood of subsequent intellectual disability. Therefore, while routine EEG and MRI should not be broadly recommended in uncomplicated ISD, children with persistent delay, especially after 4 years of age, require closer longitudinal follow-up and targeted developmental, neuropsychological, genetic, and electrophysiological evaluation when clinically indicated.

Ethics

Ethics Committee Approval: The study was approved by the İzmir Bakırçay University Non-Interventional Clinical Research Ethics Committee (approval no: 344/324, date: 06.10.2021).

Informed Consent: Retrospective cohort study.

Footnotes

Authorship Contributions

Surgical and Medical Practises: H.G.T., Concept: H.G.T., M.B.Ö., Design: H.G.T., M.B.Ö., Data Collection or Processing: H.G.T., Analysis or Interpretation: H.G.T., M.B.Ö., Literature Search: H.G.T., M.B.Ö., Writing: H.G.T.

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