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ORIGINAL ARTICLE

Neurological Evaluation of Children Followed Up For Speech Retardation

Konuşma Geriliği Sebebi ile Takip Edilen Çocukların Nörolojik Açıdan Değerlendirilmesi

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ABSTRACT

Aims: The aim of this study was to assess the neurological aspects of patients admitted to the pediatric neurology outpatient clinic with the complaint of speech retardation.
Methods: A retrospective analysis was performed on the data of the patients underwent treatment for speech retardation between February-October 2022 at the pediatric neurology outpatient clinic of Dr. Ali Kemal Belviranlı Obstetrics and Pediatrics Hospital and Konya City Hospital.
Results: The study included 149 patients with a mean age of 41.1+16.2 months, 74.5% of the patients were male. It was revealed that 8.7% of the children had a history of prematurity, 16.1% had a family member with speech retardation. In electroencephalography, epileptic disorders were found in 2.6% of the patients, psychiatric disorders (8.7% autism) in 16.7%, hearing loss in 0.7%, developmental retardation in the isolated language area in 75.8%, and global development retardation in 24.2%. Brain magnetic resonance imaging showed an abnormality of 3.3%. Furthermore, 52.3% of our patients were deficient in stimuli, while 59.7% used intensive media.
Conclusion: In terms of early diagnosis and treatment of speech retardation and accompanying pathologies, the diagnostic approach in children brought in due to speech retardation is critical.

Keywords: Speech retardation, developmental retardation in language, language impairment, child

ÖZ

Amaç: Bu çalışmanın amacı çocuk nöroloji polikliniğine konuşma geriliği şikayeti ile başvuran hastaların nörolojik yönden değerlendirilmesidir.
Metot: Dr. Ali Kemal Belviranlı Kadın Doğum ve Çocuk Hastanesi ile Konya Şehir Hastanesi çocuk nöroloji polikliniğinde Şubat-Ekim 2022 tarihleri arasında konuşma geriliği tedavisi gören hastaların verileri retrospektif olarak incelendi.
Bulgular: Çalışmaya yaş ortalaması 41.1+16.2 ay olan 149 hasta alındı, hastaların %74.5'i erkekti. Çocukların %8,7'sinde prematüre öyküsü olduğu, %16,1'inin ailesinde konuşma geriliği olduğu saptandı. Elektroensefalografide hastaların %2,6'sında epileptik bozukluklar, %16,7'sinde psikiyatrik bozukluklar (%8,7 otizm), %0,7'sinde işitme kaybı, %75,8'inde izole dil alanında gelişme geriliği ve %24,2'sinde global gelişme geriliği saptandı. Beyin manyetik rezonans görüntüleme %3,3'lük bir anormallik gösterdi. Ayrıca hastalarımızın %52,3'ünün uyaran eksikliği olduğu, %59,7'sinin yoğun medya kullandığı saptandı.
Sonuç: Konuşma geriliği ve eşlik eden patolojilerin erken tanı ve tedavisi açısından konuşma geriliği nedeniyle getirilen çocuklarda tanısal yaklaşım önemlidir.

Anahtar Sözcükler: Konuşma geriliği, dilde gelişme geriliği, dil bozukluğu, çocuk

Introduction

Speech retardation is defined as a situation in which a child's speech is less fluent than expected for his/her age or contains more speech and voice errors that are incompatible with his/her age (1-3). Individual differences in the acquisition of basic criteria related to language development can be seen, but the order of development is universal. Language development characteristics include producing first words around the first year of life, having a vocabulary of 50 words at 18 months of age, having binary word combinations (such as mom come) at two years of age, longer expressions between 2-3 years of age, development of adjectives, actions, and independent morphemes in receptive and expressive language, and the use of longer and complex sentences at 4-5 years of age (4). While healthy brain regions are required for the development of human language, healthy cognitive processes should be guided (memory, processing speed, attention) (5).

It is critical to diagnose speech retardation as soon as possible to treat any underlying causes, especially reversible ones like epilepsy and oropharyngeal deformities. Psychosocial problems associated with children who did not receive early intervention have been shown to persist into adulthood. Children with speech delays in their early years are more likely to struggle academically (6). As a result of the screening test results, which are known in the literature as the 'wait-and-see approach,' the approach of not intervening has been abandoned today, with the assumption that children with typical development in the recipient language between 18-24 months but with a limited vocabulary retardation in the expressive language will catch up with their peers in the future, such as 4-5 years of age(7-9) .A close follow-up is essential when a speech issue is suspected; 2/3 of children with speech delays before the age of 3.5 require treatment after one year (10). According to current research, children with

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speech retardation should be directed for detailed language and speech evaluations as soon as possible (11).

Aside from medical risk factors and causes such as hearing loss and epilepsy, familial and environmental factors have also been associated to speech disorders (12). The lack of stimulus can be defined as the inadequate presentation of environmental factors suitable for the age and developmental level of the child (13-15). Although etiological research is important, the literature on the subject is limited (12-16). The goal of this study was to evaluate the children who were monitored for speech retardation from a neurological standpoint.

Material and Methods

All patients aged 1-9 years at the time of application and were brought to the pediatric neurology outpatient clinics of Dr Ali Kemal Belviranlı Obstetrics and Pediatrics Hospital and Konya City Hospital between February and October 2022 were included in our study, which was conducted using an observational, retrospective, and cross-sectional design. The records of these patients who were followed up in the pediatric neurology outpatient clinic were examined and recorded in the patient follow-up forms retrospectively. Patients' history, background, and family history as well as diseases of the mother during pregnancy, perinatal trauma, infections, asphyxia at birth, gestational week at birth, birth weight, child's medical history, ototoxic drug use history, and psychosocial history were all reviewed. In addition, lack of stimulus, a family history of significant disease, and a family history of speech retardation were all recorded on the patient follow-up forms. The lack of stimulus was defined as the inadequate presentation of environmental factors suitable for the age and developmental level of the child (13-15). During the physical examination, information such as the child's head circumference, dysmorphic features, abnormal findings, visual and hearing evaluations, and data on department of otorhinolaryngology consultations requested in terms of hearing and oropharyngeal pathologies were transferred from patient files to patient follow-up forms. According to accompanying psychiatric additional abnormalities (such as autism spectrum disorder, attention deficit and hyperactivity disorder, cognitive retardation, dyslexia), the patients in our study were referred to pediatric mental health and diseases, and the discovered psychiatric additional abnormalities were recorded in the patient follow-up form. Isolated language developmental delay was defined as patients without additional psychiatric abnormalities whose development in the developmental domains other than language was assessed in accordance with their age during their neurological examination (4, 17). Global developmental delay is defined as patients who have developmental delays in at least two of the developmental domains (17). The patient follow-up form from the patient files includes the findings of the neuroimaging procedures (brain magnetic resonance imaging-MRI, and electroencephalography-EEG), performed to rule out intracranial pathologies of the patients. Additionally, the patient follow-up form contained a review of the biochemical and laboratory tests performed on the patients to determine the etiology. The main factors influencing developmental language disorder are defined in the literature as a family history of language disorder or dyslexia, male gender, low socioeconomic level, low parental education level, presence of neglect or abuse, preterm birth, and low birth weight (18, 19). First, we performed a logistic regression analysis to examine the effect of "Effect of Psychiatric Additional Abnormality and Speech Retardation" and "Effect of Isolated Language Developmental Retardation" as defined in the literature.

Statistical analysis

Data were analyzed with IBM SPSS V23. For quantitative data, the results were presented as mean±standard deviation, and median (minimum - maximum), and for categorical data, as frequency and percentage. The risk factors affecting the development of accompanying psychiatric abnormalities and isolated language development retardation were investigated using binary logistic regression analysis. The significance level was accepted as $p<0.050$.

Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The ethics committee approval (date: 04.11.2022, meeting number: 163, decision number: 2022/4027: (11679) was obtained for the study. We collected no data that could be used to identify patients. As this was a non-interventional retrospective study, informed consent forms were not considered necessary.

Results

Our study included 149 patients, 111 (74.5%) of whom were male, with a mean age of 41.1+16.2 months (median: 36.9 months, min-max:17.2-91 months), and admitted to the pediatric neurology outpatient clinic with the complaint of speech retardation. Premature birth history was observed in 13 (8.7%) of the patients, and intrauterine growth restriction (IUGR) was noted in seven (4.7%). It was found that 16.1% (n=24) of the patients had a family member with speech retardation. According to the findings, 2.6% (n=4) of the patients had epileptic disorder in electroencephalography (two focal, two generalized), and 3.3% (n=5) had abnormalities in brain magnetic resonance imaging (MRI). Brain MRI revealed periventricular nodular heterotopia and mega cisterna magna in one patient, arachnoid cyst in one patient, periventricular gliosis in two patients, and polymicrogyria and periventricular leukomalacia in one patient. In the psychiatric evaluation, it was detected that 8.7% (n =13) of the patients had autistic spectrum disorder, 4.7% (n =7) had attention deficit and hyperactivity

Table 1. Demographic and clinical information of the patients

	Frequency (n)	Percent (%)
Gender (male)	111	74.5
Prematurity	13	8.7
Type of Birth (caesarean section-C/S)	70	47
Consanguinity	24	16.1
History of Speech Delay in Relatives	24	16.1
History of Neonatal Intensive Care Unit Hospitalization	14	9.4
Presence of prenatal problems	9	6
Intrauterine Growth Restriction (IUGR)	7	4.7
Ototoxic Drug Use (Gentamicin)	1	0.7
Seizure History	5	3.4
Hearing Test Abnormality (sensorineural)	1	0.7
Neurological Examination Abnormality	47	31.5
Autistic Spectrum Disorder (ASD)	13	8.7
Attention Deficit Hyperactivity Disorder (ADHD)	7	4.7
Cognitive Retardation	4	2.7
Dyslexia	1	0.7
Associated Psychiatric Abnormalities (ASD, ADHD, Cognitive Retardation, Dyslexia)	25	16.7
Accompanying Motor Developmental Retardation	12	8.1
Oropharyngeal Pathology (All Ankyloglossia)	2	1.3
Metabolic Disease (All Partial Biotinidase Deficiency)	4	2.7
Lack of Stimulus	78	52.3
Media Exposure (>1 hour/day)	89	59.7
Developmental Retardation in Isolated Language	113	75.8
Global Developmental Retardation	36	24.2
Epileptic Abnormality in EEG	4	2.6
Brain MRI Abnormality	5	3.3

Table 2A. Investigation of Risk Factors Affecting Speech Retardation with Psychiatric Additional Abnormality

	Univariate		Multivariate	
	OR (% 95 CI)	p	OR (% 95 CI)	P
Gender (reference: girl)	2.884 (0.811 – 10.249)	0.102	6.045 (1.135 – 32.201)	**0.035**
Speech retardation in relatives	0.669 (0.183 – 2.44)	0.542	2.201 (0.371 – 13.046)	0.385
Media exposure (Reference: No)	1.92 (0.748 – 4.927)	0.175	105.073 (10.484 – 1053.06)	**<0.001**

Table 2B. Examination of risk factors affecting isolated language developmental delay

	Univariate		Multivariate	
	OR (% 95 CI)	**p**	**OR (% 95 CI)**	**P**
Gender (reference: girl)	0.506 (0.192 – 1.332)	0.168	0.389 (0.115 – 1.319)	0.130
Prematurity	4.158 (0.522 – 33.15)	0.178	4.665 (0.388 – 56.034)	0.225
Speech retardation in relatives	1.72 (0.547 – 5.413)	0.354	1.241 (0.268 – 5.746)	0.782
IUGR history	0.787 (0.146 – 4.242)	0.781	0.854 (0.1 – 7.309)	0.885
Lack of stimulus (Reference: No)	2.396 (1.103 – 5.204)	**0.027**	30.354 (4.536 – 203.111)	**<0.001**

disorder, 2.7% (n=4) had cognitive retardation, and 0.7% (n=1) had dyslexia. Sensorineural hearing loss (SNHL) was observed in 0.7% (n=1) of the patients, and ankyloglossia in 1.3% (n=2). Demographic and clinical information of the patients are summarized in Table-1.

A binary logistic regression analysis was performed to examine the risk factors for speech retardation with additional psychiatric abnormality as univariate and multivariate models. While gender had no statistically significant effect in the univariate model (p=0.102), in the multivariate model, males had a 6.045 times higher risk of speech retardation with additional psychiatric abnormalities than females (p=0.035). While there was no effect of media exposure in the univariate model (p=0.175), it was 105.073 times higher in those with media exposure than in those without mental health abnormalities and speech retardation in the multivariate model (p<0.001). Other risk factors were not statistically significant (p>0.050).

Binary logistic regression analysis was completed to examine risk factors affecting isolated language developmental retardation in univariate and multivariate models. The risk of isolated language developmental delay was 2.396 times higher in those with stimulus deficiency in the univariate model (p=0.027), but it was 30.354 in the multivariate model (p<0.001).

Discussion

Language and speech development is a complex process shaped by the interaction of genetic, environmental, and cultural factors and is accepted as the basic element of cognitive and social development in humans (20). If a child does not know the words 'mother' and 'father' by the 12th month, cannot combine two words by the age of two, cannot form a three-word sentence by the age of three, or speaks incomprehensibly, it is recommended that they be evaluated for speech retardation (21). The mean age of the patients in our study was 41.1+16.2 months (median: 36.9 months, min-max:17.2-91 months), which was slightly lower than the literature's average of 41.3-65.9 months, but it was found that speech delay was detected late in the majority of patients. (1, 13). This situation led us to believe that the families and physicians who had previously evaluated the patient were primarily concerned with the patients' physical development and that the language development steps had not been adequately evaluated.

It has been reported that 8% of preschool children in the United States have an inherited effect on speech delay (22). This rate has been reported to be in the range of 2-8 percent in similar publications from developed countries (23). Another study revealed that only 2.5% of preschool children aged two to five had a delay in speech acquisition (13). Although there is no consensus on the etiology of speech problems, some risk factors have been noted. Gender is known as a risk factor (11, 24). It is known that the risk is three times higher in men. According to studies, this is due to boys

having a later maturation of the central nervous system than girls. In addition, the presence of individuals who have experienced speech delay in the family increases this risk twice (25). Low birth weight and prematurity were also identified as risk factors. Children born at less than 85% of their ideal birth weight or prematurely are twice as likely to have delayed speech (22). Although there was an 8.7% history of premature birth and a 4.7% history of intrauterine growth restriction (IUGR) in our study, neither was associated with an increased risk of speech retardation in the logistic regression analysis. Furthermore, many factors such as the child's intelligence capacity, social environment, family socioeconomic status, number of siblings, child personality, caregiver attitude, and educational status may influence speech and language development (13). In our study, 74.5% of the patients were male, and boys were more likely than girls to have speech retardation. When the risk factors affecting speech retardation with psychiatric abnormality were examined in our study using logistic regression analysis, the risk of speech retardation with psychiatric abnormality was 6.1 times higher in males than females in the multivariate model (p=0.035). Furthermore, in our study, 16.1% (n= 24) of the patients had another person in their family with speech retardation, and a lower rate of family history was reported in around 42% compared to the reported literature (1). Birth asphyxia, epileptic disorder, and oropharyngeal deformities are all risk factors for speech retardation, according to research (13, 26). Epileptic discharges in the speech center may also prevent speech, a hypoxic central nervous system may have a negative effect during an epileptic attack, and aphasia in Landau-Klefner syndrome (LKS), which is acquired epileptic aphasia, may develop in this manner. Neuron functions are known to be disrupted during epileptic discharge. It has been reported that epileptic discharges in the dominant hemisphere impair verbal tasks, whereas discharges in the non-dominant hemisphere impair perception and application of nonverbal tasks (27). According to one study, the rate of epileptic disorder in dysphasic children with speech retardation is 8%, which is significantly higher than the rate in the general population. In the same study, this rate was 36% in patients with regression and 58% in LKS (28). Epileptic disorder was observed in 6.7% of children with speech retardation in one study, with an estimated relative risk of 5.28 (29). In our study, epileptic disorder was detected at a rate of 2.6% on EEG, which, while lower than the literature, was higher than the reported prevalence of epilepsy in the general pediatric population in Türkiye, which was 0.8% (30).

Oropharyngeal deformities were found at a low rate of 1.3% in our study (ankyloglossia in two patients). Several studies in the literature have reported a strong cause-effect relationship between hearing loss and speech retardation (31). However, there are also studies with findings to the contrary (32). It is a well-known fact that having good hearing in the first years of life is critical for the development of speaking ability (21). In our study, we determined that 0.7% (n=1) of the patients had hearing loss (sensorineural). We believe that due to the importance of early treatment in terms of plasticity, both hearing loss (such as cochlear implants) and oropharyngeal pathologies (surgical treatment), such as ankyloglossia, should be evaluated by an otorhinolaryngology specialist in the early period, along with a pediatric neurologist. Furthermore, auditory neuropathy can be seen alone or as part of other diseases such as Charcot-Marie-Tooth (CMT) disease and Friedreich's Ataxia (33, 34). Although clinical findings consistent with a genetic neuropathy such as CMT or Friedreich's Ataxia were not found in our one patient with sensorineural hearing loss, partial biotinidase deficiency and hypothyroidism were discovered during the examinations. Hearing loss has been linked to biotinidase deficiency and hypothyroidism, according to the literature (35, 36). While no other hypothyroid patients were found in our study, three patients had partial biotinidase deficiency without hearing loss, and partial biotinidase deficiency was found in our population at a rate of 2.7%. Patients with profound biotinidase deficiency have a mean serum enzyme activity of less than 10%, while those with 10-30% of mean normal serum activity have partial deficiency (37). According to newborn screening data, the incidence of profound biotinidase deficiency was 1/80000 in 2006, with partial biotinidase deficiency reported between 1/31000-40000 (38). In a study conducted in Türkiye, the incidences of profound and partial biotinidase deficiency were reported as 1/14866 and 1/53093, respectively, with 93 cases of profound and 26 cases of partial biotinidase deficiency expected in the country each year (39). Due to the high number (n=4) and rate (2.7%) of partial biotinidase deficiency in our study, we believe that biotinidase deficiency should be re-evaluated in children with speech retardation. Furthermore, since 25% sensorineural hearing loss was detected in these cases, we believe that more detailed and long-term hearing loss follow-up of patients with biotinidase deficiency is required.

A study conducted in Türkiye between 2011 and 2014 found that 52.2% of children with speech retardation had global developmental delay and 21% had a high risk of autism (1). In our study, we observed global developmental retardation in 24.2% of the patients we evaluated for speech retardation, autism spectrum disorder in 8.7%, attention deficit and hyperactivity disorder in 4.7%, cognitive retardation in 2%, dyslexia in 0.7%, and developmental delay in isolated language area in 75.8%. Global developmental delay and autism were at a lower rate in our study, despite being at a higher rate in the literature, and developmental retardation was found higher in the isolated language area. Again, in our study, 50-60% of the patients had a lack of stimuli or had been exposed to media for more than an hour. This difference was thought to be related to the fact that our study was conducted following the covid-19 pandemic, which limited social communication by causing lockdowns and increased

media exposure. As a matter of fact, the fact that only one of our patients had a history of covid-19 suggests that our patients' families generally adhered to the Covid-19 isolation recommendations. In our study, the risk of speech retardation with additional psychiatric abnormality was 105.1 times higher in those exposed to media ($p<0.001$).

The majority of the patients (52.3 %) in our study had stimulus deficiency, and in the logistic regression analysis, it was 2.4 times higher ($p=0.0027$) in those with stimulus deficiency than in those without isolated language developmental retardation in the univariate model, and 30.4 in the multivariate model ($p<0.001$).

According to the findings, 3.3% (n=5) of the patients had abnormalities in brain magnetic resonance imaging (MRI), and one patient had periventricular nodular heterotopia and mega cisterna magna, one patient had an arachnoid cyst, two patients had periventricular gliosis, and one patient had polymicrogyria and periventricular leukomalacia. The presence of polymicrogyria in the patient's bilateral frontoparietal localization suggested that the motor area of speech was affected (40-42). Periventricular nodular heterotopia, a developmental malformation, has also been linked to speech retardation, particularly when combined with polymicrogyria (42). Periventricular gliosis in two patients and periventricular leukomalacia in one patient suggested a previous hypoxic and/or ischemic process, though asphyxia was not present in these patients (43). In one of our patients with isolated language developmental delay, a four cm diameter arachnoid cyst was discovered in the right parasagittal area of the posterior fossa. Despite the fact that the association of speech disorder with arachnoid cysts with frontal and sylvian fissure localization has been reported in the literature, we were unable to find a case of arachnoid cyst with speech retardation in this location (44, 45). Again, we found no evidence in the literature linking mega cisterna magna in this anatomical location to speech retardation. This suggested that arachnoid cyst and mega cisterna magna were detected incidentally.

The limitations of our study were that it was a retrospective study, that no control group was used, that the number of patients was small, and that the follow-up period was short since it was a cross-sectional study.

However, in evaluating the research findings, we aimed to determine the clinical causes of speech retardation that are preventable, reversible, and treatable, and to draw attention to the significance of early intervention. As a result, despite its limitations, our study concludes that speech retardation in children should be prioritized by patients, families, educators, and clinicians, and early intervention for the etiology is required.

As a result, in our study, particularly in children with speech retardation, environmental abnormalities such as lack of stimulus, intense media exposure, metabolic abnormalities such as biotinidase deficiency, epilepsy, hearing loss, and ankyloglossia emerged as preventable and treatable causes, and it should be considered in children brought in due to speech retardation, and early treatment should be planned for these children.

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