



Published in final edited form as:

Afr J Health Sci. 2020 ; 33(2): 12–21.

Childhood autism spectrum disorder: insights from a tertiary hospital cohort in Kenya

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Abstract

Introduction: Autism spectrum disorder (ASD) is characterized by impairments in behavior, social communication, and interaction. There is little data on ASD from sub-Saharan Africa (SSA) describing clinical characteristics in large cohorts of patients. Preliminary studies report a high male sex ratio, excess of nonverbal cases, possible infectious etiologies, and comorbidities e.g. epilepsy.

Objective: To describe the clinical characteristics of children diagnosed with ASD in an African context.

Methods: A retrospective medical chart review identified 116 children diagnosed with ASD according to DSM-5 criteria at a pediatric neurology clinic in Nairobi, Kenya.

Results: The male to female ratio was 4.3:1. The median age at presentation was 3 years with speech delay as the most common reason for presentation. Expressive language delay was observed in 90% of the population. Sixty percent who obtained imaging had normal MRI brain findings. Only 44% and 34% of children had access to speech therapy and occupational therapy respectively. Epilepsy and ADHD were the most prevalent comorbidities.

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Competing interests

The authors declare that they have no competing interests.

Conclusion: An early median age at presentation and preponderance of male gender is observed. Access to speech therapy and other interventions was low. A prospective study would help determine outcomes for similar children following appropriate interventions.

Keywords

Autism; sub-Saharan Africa; comorbidities; language delay; intellectual disability; birth history

Introduction

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by impairments in social communication and interaction, and restricted or repetitive patterns of behavior, interests, and activities. ^[1] The burden of ASD in sub-Saharan Africa (SSA) remains unclear owing to the lack of large scale population studies in this region. A high prevalence has been observed in males ^[2], with intellectual disability and epilepsy reported as the most common co-morbidities ^[3–5], a high proportion of nonverbal cases of ASD ^[3], and possible infectious disease etiology ^[6] have been described in SSA. Underdiagnosis and late presentation are common, where a median age of 8 years at presentation has been reported in some areas. ^[7–10] Franz et al. ^[3] indicated excesses of non-verbal cases of ASD (71%), compared to 25% in developed countries. Studies in Tanzania and Kenya describe a close temporal relationship between severe malaria and the diagnosis of ASD. ^[6, 11]

Magnetic resonance imaging (MRI) abnormalities have been documented in children with ASD in high resource countries, but there is no data from SSA. White matter hyperintensities (WMH) in regions of brains of children with ASD are reported ^[12], associated with cognitive and functional impairments which could be of particular relevance to behavioral impairment observed in ASD. ^[12–14] Furthermore, previous research has shown an increase of cerebral hyperintensities among patients diagnosed with neuropsychiatric disorders such as attention deficit hyperactivity disorder (ADHD), a common comorbidity among ASD patients. ^[15]

Material and methods

The aim of this study was to describe specific characteristics of children diagnosed with ASD based on data from Aga Khan University Hospital in Nairobi, Kenya. This hospital is an urban, private not for profit institution, which provides emergency and follow-up services for the resident population and also functions as a referral hospital within the Eastern African region.

Study design

This was a retrospective cross-sectional study of patient medical records that identified children with a diagnosis of ASD between 2011 and 2016. Patient medical records consisted of physical files and electronic databases.

Participants

Children with concerns for behavioral and neurodevelopment disorders who were either self-referred, or referred by their pediatrician for care at a tertiary care clinic were included in this study.

Procedure

Two experienced pediatric neurologists evaluated all patients and utilized the same criteria to make the diagnosis of ASD for each child following comprehensive interviews with parents and the child, as well as clinical observation. Children who met the criteria outlined in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition ^[1] were diagnosed to have ASD. Both neurologists also assessed children for comorbidities such as Attention Deficit Hyperactivity Disorder (ADHD) and epilepsy using standard screening tests and clinical evaluation. A diagnosis of ADHD was made in patients who fulfilled the DSM 5 criteria. Criteria established by the International League against Epilepsy (ILAE) ^[16] were used to diagnose epilepsy.

A research assistant extracted data from the relevant medical charts according to pre-determined criteria. Each medical record was allocated a unique study identification number at the time of data entry to maintain patient confidentiality. Ethical Approval was sought and obtained from the Human Ethics Research Committee at Aga Khan University Hospital prior to the start of the study. Variables of interest were obtained, coded and entered into a Microsoft Excel spreadsheet. These variables included presenting complaints, anthropometric measures, birth, developmental and family history, co-morbidities, features of learning disability (if present), and referral status.

Measures

Anthropometric data were documented using growth charts developed by the Centers for Disease Control (CDC). ^[17]

The Modified Checklist for Autism in Toddlers, Revised (M-CHAT-R) version ^[18] was administered to parents of children between 16 and 30 months of age. The M-CHAT-R has been validated in international settings and demonstrated adequate psychometric properties to effectively assess risk for developing ASD among children 16 – 30 months of age. ^[18, 19] The M-CHAT-R consists of 20 questions coded with pass or fail criteria according to the following scoring algorithm. Those who fail 3 – 7 items are classified as having a medium risk of an eventual diagnosis of ASD while those who fail more than 7 items are classified as high risk for ASD. ^[18, 19].

In the context of the high burden of CNS infections and other environmental causes of neurological problems and developmental disabilities in SSA, MRI brain scans were done to establish neuroimaging evidence for such factors being contributory in individual cases. Radiology reports from these MRI brain scans, as available in the clinical records, were reviewed. All MRI reports were anonymized prior to the collection and analysis of subject data. Clinical findings recorded in reports were summarized and classified as: normal

findings, white matter abnormalities, gray matter abnormalities, congenital malformations, focal infarcts, and heterogeneous lesions.

Data analysis—Statistical analysis was conducted using IBM statistics SPSS version 23 software. Data were summarized using descriptive statistics, including frequencies, proportions, means, standard deviations, and median as appropriate. For variables with missing data, the analysis was confined to available data.

Results

Demographics

A total of 1,711 medical records were identified from children who attended the neurology clinic during the study period. Of those, 116 (7%) were records of children with a diagnosis of ASD. The baseline characteristics of the study population are presented in Table 1. Among those with ASD, the median age at presentation was 3 years with the age range being 1 to 23 years. There were more males (94, 81.1%) than females (22, 18.9%) at a ratio of 4.3:1. Twenty-seven (23%) children in this cohort were self-referred while 89 (77%) were referred by other medical practitioners.

Presenting Complaints

Delayed language development was the most common reason for presentation to the pediatric neurology clinic and was reported in 102 (87.9%) children, as they had not spoken their first word by 16 months. Only n = 21 (18.1%) could follow simple instructions by 9 months of age (see Figure 1). Auditory assessments were conducted for 43 (37.1%) children of whom 5 (4.3%) were reported to have impaired hearing. The uptake of speech therapy and occupational therapy was documented for 51 (43.9%) and 40 (34.4%) children respectively following evaluation. The median age at walking however was 12 months, (range 8–36 months) Hyperactivity as presenting complaint was reported as a significant concern in 59 (50.9%) children while a short attention span was documented for n=25 (21.6%) children (See Table 1).

Birth circumstances

Pre-term births occurred in 10 (8.6%) children while vaginal delivery was reported for 51 (44%). Caesarian section delivery was recorded for n=45 (38.8%) children. Neonatal sepsis and meningitis were reported for 10 (8.6%) and 5 (4.3%) patients respectively. More details regarding the cohort birth details are provided in Table 2.

Physical features

Eleven patients (17.5%) had a head circumference above the 97th percentile and only 1 (0.9%) patient had a head circumference below the 3rd percentile^[17] at presentation. Seven children (6.2%) had documented facial dysmorphism. Neurocutaneous markers of hypo-pigmented lesions were documented in 4 children (3.5%), and 13 children (11.5%) had hyper-pigmented lesions but none were thought to represent a syndromic or neurocutaneous disorder.

Family history

A total of 62 children (53%) in the ASD cohort were the first-born children in the family. Two (3%) patients had an immediate family member and 12 (10%) had a second or third-degree relative known to have ASD. Epilepsy was reported in 2 (1.7%) family members, while 62 (53.4%) patients reported good health in the family.

Comorbidities

Attention deficit hyperactivity disorder was the most common psychiatric comorbidity in this cohort (n = 24, 21%), with epilepsy in 23 (20%) children. Sleep disorders were reported in 14 (12.1%) children while elimination disorders (encopresis or enuresis) were reported for 12 (10.3%) children. Data on anxiety and intellectual disability was not available.

Medication use

A total of 46 (39.7%) children had medications prescribed as part of their management. These included sodium valproate for 19 (16%), clonazepam for 9 (8%), and clobazam for 4 (3%) for children with epilepsy. Methylphenidate was prescribed for 6 (5%) children with ADHD, while melatonin was available for 5 (4%) and risperidone for (3%) children who predominantly presented with difficulty in initiating sleep and aggressive behavior respectively.

Educational status

Because most (95, 81.9%) of children in this cohort were 6 years or younger at presentation, it is difficult to describe formal educational status as many were too young to have begun primary education. However, among those who had attained school-going age (6 years and above) at presentation, 17 out of 29 (59%) children did not attend school due to lack of appropriate placement required to meet the child's needs. A total of 50 (43%) children in this cohort were eventually enrolled in school. Among these, parents of 18 (36%) reported that the child had difficulties understanding what was taught in school. Half, 9 (18%) were enrolled in a mainstream school and the other half 9 (18%) attended schools that provided learning support.

M-CHAT-R

A total of 31 (26.7%) children aged 16 to 30 months completed the M-CHAT-R. Details of this evaluation are provided in Table 3. All patients who completed the M-CHAT-R also met DSM-5 criteria for ASD.

Neuroimaging

A total of 32 patients (27.5%) had MRI of the brain, of whom 12 (38.7%) had abnormal findings. All had varying degrees of white matter hyperintensities (WMH), though none of these were associated with restricted diffusion (see Table 4). Six (18.8%) of these subjects had unilateral WMH in the frontal lobe, while three had (9.4%) bilateral frontal WMH.

Four of the patients with WMH had speech delay. All six patients with unilateral WMH in the frontal lobe presented with comorbidities including developmental delay and seizures.

One patient had atrophy of corpus callosum, another had hemimegalencephaly. Atrophy of the frontal lobe and perisylvian operculum region was reported in one child while another had craniofacial disproportion and microcephaly. The remaining $n = 20$ (62.5%) patients who had an MRI did not have remarkable findings.

Discussion

This study of a hospital-based cohort of young children diagnosed with ASD identified clinical similarities and some differences in comparison with other cohorts, both from sub-Saharan Africa and high-income countries (HIC).

The median age at presentation to this neurology clinic was three years, which is considerably younger than other African cohorts, in which children had a diagnosis of ASD at a median age of eight years. [10, 20–24] This median age in the present Kenyan cohort is similar to the median age at diagnosis for children in the USA. [25] The difference at age of presentation in this cohort and that from other SSA countries may be related to the socio-economic and educational status of the families that sought services at this institution. Whereas various factors including lack of awareness and stigma [23] have been identified as contributing factors to late diagnosis of ASD in SSA, these issues may have had a smaller impact on this study cohort. The availability of a diagnostic service at this hospital may also have facilitated an earlier diagnosis.

There were more male than female patients at a ratio of 4.3:1 in this cohort. This is consistent with data [4, 20] from other settings such as Nigeria and Denmark. Similar to other studies from SSA, expressive language delay was present in a majority of the patients in this Kenyan cohort and was more common than receptive language delay. [9, 24] Whereas this cohort can be assumed to have access to more resources given their higher socioeconomic status, only half of these children had attended a speech therapy service. There is an extremely small number of practicing speech therapists in Kenya due to lack of local training, which may explain the low proportion receiving speech therapy.

Although prenatal, perinatal, and neonatal complications, as well as pre-term and post term births, have been shown to occur more frequently in populations with ASD in comparison to populations without ASD [16, 26–32], the majority of the children in this cohort reported uncomplicated, vaginal births at term. This observation would need to be explored further in larger population studies or cohorts from SSA.

The occurrence of WMH, especially in the developing brains of children, has been significantly related to cognitive and functional impairment. [13–15] The presence of WMH in this cohort may be related to potential academic challenges as indicated by difficulty coping with school. While only one subject in this cohort demonstrated an abnormal corpus callosum, this has previously been found as a common abnormality associated with ASD. Atrophy of the corpus callosum has been associated with delayed processing speeds as well as deficits in executive function. [13, 14] Overall MRI brain imaging in this cohort did not contribute to the determination of etiology or association with the level of severity of ASD and would probably not be an essential investigation in a large scale study, even in this

context with its higher prevalence of infectious diseases and other possible environmental contributions to ASD etiology and phenotype.

The primary limitation of a cross-sectional, chart review study is that generalizability to a larger population is limited. Therefore, we are not able to determine how well the characteristics of this hospital-based cohort represent the overall population in Kenya or SSA. Kenya is characterized by rich cultural and linguistic diversity, and future studies are indicated to evaluate characteristics of representative cohorts. This sample is potentially reflective of a population of higher socioeconomic status, as the sample was drawn from a cohort seeking services at a private hospital, though this was not measured directly. Further, we did not have direct measures of intellectual functioning, which has important diagnostic and treatment planning implications. This should be an included focus of future studies. Finally, a diagnosis was determined based on clinical interviews alone. Although the diagnosis was made by highly trained and experienced physicians, additional approaches using standardized instruments would have increased the validity of diagnostic determination and allowed for better comparison with prior research.

Conclusion

This study described the clinical features of a cohort of children with a diagnosis of ASD in SSA which are similar to those in HICs. Expressive language delay was observed in the majority of this cohort, with less than half reporting access to necessary interventions to improve outcomes. Extending clinical investigations to representative samples, as well as including formal assessment of intellectual functioning is an important area for future studies on children with ASD in the African context.

Acknowledgments

We wish to acknowledge the support provided by James Kaburia, the medical records department and Peter Gatiti, librarian at Aga Khan University, Nairobi, as well as funding generously provided by the Stanley Center for Psychiatric Research, Broad Institute of MIT and Harvard.

Funding:

This study was funded by a grant provided by the Stanley Centre for Psychiatric Research, Broad Institute of MIT and Harvard University.

List of Abbreviations

ADHD	Attention Deficit Hyperactivity Disorder
ASD	Autism Spectrum Disorder
DSM 5	Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition
ICD 10	International Classification of Disease, version 10
Kgs	Kilograms
M-CHAT	Modified Checklist for Autism in Toddlers

MRI	Magnetic resonance Imaging
NDD	Neurodevelopmental Disorder
NHIF	National hospital insurance fund
SSA	Sub-Saharan Africa
WMH	White matter hyperintensities

References

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 5th ed (DSM-5) Arlington, VA: American Psychiatric Publishing; 2013.
2. Bakare MO, Munir KM. Autism Spectrum Disorders in Africa. *Afr J Psychiatry*. 2011;14(3):208–210. doi: 10.4314/ajpsy.v14i3.3.
3. Franz L, Chambers N, von Isenburg M, de Vries PJ. Autism spectrum disorder in sub-saharan africa: A comprehensive scoping review. *Autism Res*. 2017;10(5):723–749. doi: 10.1002/aur.1766. [PubMed: 28266791]
4. Bakare MO, Ebigbo PO, Ubochi VN. Prevalence of Autism Spectrum Disorder among Nigerian Children with Intellectual Disability: A Stopgap Assessment. *J Heal Care Poor Underserved*. 2012;23(2):513–518. doi: 10.1353/hpu.2012.0056
5. Bakare MO, Kerim MM. Autism Spectrum Disorders in Africa, A Comprehensive Book on Autism Spectrum Disorders. Mohammad-Reza Mohammadi (Ed). 2011 DOI: 10.5772/17469 Available from: <https://www.intechopen.com/books/a-comprehensive-book-on-autism-spectrum-disorders/autism-spectrum-disorders-in-africa>
6. Mankoski RE, Collins M, Ndosi NK, Mgalla EH, Sarwatt FS. Etiologies of autism in a case-series from Tanzania. *J Autism Dev Disord*. 2006;36(8):1039–1051. doi: 10.1007/s10803-006-0143-9 [PubMed: 16897390]
7. Newton CR, Chungani DC, The continuing role of ICNA in Africa: how to tackle autism? *Developmental Medicine and Child Neurology*. 2013;55(6):488–489. doi: 10.1111/dmcn.12150. [PubMed: 23614818]
8. Lotter V Cross Cultural Perspectives on Childhood Autism. *J Trop Pediatr*. 1980; 26(4):131–133. doi: 10.1093/tropej/26.4.131-a [PubMed: 7463549]
9. Zeglam AM, Maouna A. Is there a need for a focused health care service for children with autistic spectrum disorders? A keyhole look at this problem in Tripoli, Libya. *Autism* 2012;16(4):337–339.
10. Bakare MO, Kerim MM. Excess of non-verbal cases of autism spectrum disorders presenting to orthodox clinical practice in Africa – a trend possibly resulting from late diagnosis and intervention. *S Afr J Psychiatr*. 2011;17(4):118–120. [PubMed: 22229024]
11. Carter JA, Lees JA, Gona JK, et al. Severe falciparum malaria and acquired childhood language disorder. *Dev Med Child Neurol*. 2006; 48(1):51–57. [PubMed: 16359594]
12. Boddaert N, Zilbovicius M, Philipe A, et al. MRI findings in 77 children with non-syndromic autistic disorder. *PLOS one*. 2009; 4(2):e4415. doi: 10.1371/journal.pone.0004415. [PubMed: 19204795]
13. Chen R, Jiao Y, Herskovits EH. Structural MRI in autism spectrum disorder. *Pediatr Res*. 2011; 69(5 Pt 2):63R–8R. doi: 10.1203/PDR.0b013e318212c2b3.
14. Jokinen H, Ryberg C, Kalska H, et al. Corpus callosum atrophy is associated with mental slowing and executive deficits in subjects with age-related white matter hyperintensities: the LADIS Study. *J Neurol Neurosurg Psychiatry*. 2007;78(5):491–496. [PubMed: 17028118]
15. Amat JA, Bronen RA, Saluja S, et al. Increased number of subcortical hyperintensities on MRI in children and adolescents with Tourette's syndrome, obsessive-compulsive disorder, and attention deficit hyperactivity disorder. *Am J Psychiatry* 2006;(6)163:1106–1108. [PubMed: 16741215]
16. Berg AT, Berkovic SF, Brodie MJ, et al. Revised terminology and concepts for organization of seizures and epilepsies: Report of the ILAE Commission on Classification and Terminology,

- 2005–2009. *Epilepsia*. 2010; 51(4):676–685 doi: 10.1111/j.1528-1167.2010.02522. [PubMed: 20196795]
17. Centers for Disease Control and Prevention, National Center for Health Statistics. CDC growth charts: United States. <http://www.cdc.gov/growthcharts/>. 5 30, 2000.
 18. Robins DL, Fein D, Barton ML, Green JA. The Modified Checklist for Autism in Toddlers: An Initial Study Investigating the Early Detection of Autism and Pervasive Developmental Disorders. *J Autism Dev Disord*. 2001;31(2):131–144. [PubMed: 11450812]
 19. Seif E, Habib D, Noufal A, et al. 2008. Use of M-CHAT for a multinational screening of young children with autism in the Arab countries, *International Review of Psychiatry* 2008;20(3):281–289. doi:10.1080/09540260801990324 [PubMed: 18569180]
 20. Jensen CM, Steinhausen HC, Lauritsen MB. Time trends over 16 years in incidence-rates of autism spectrum disorders across the lifespan based on nationwide Danish register data. *J Autism Dev Disord*. 2014;44(8):1808–1818. [PubMed: 24554161]
 21. Bello-Mojee MA, Omigbodun OO, Bakare MO, Adewuya AO. Pattern of impairments and late diagnosis of autism spectrum disorder among a sub-Saharan African clinical population of children in Nigeria. *Glob Ment Heal*. 2017;4:e5. doi: 10.1017/gmh.2016.30
 22. Harrison AJ, Zimak EH, Sheinkopf SJ, Manji KP, Morrow EM. Observation-centered approach to ASD assessment in Tanzania. *Intellect Dev Disabil*. 2014;52(5):330–347. doi: 10.1352/1934-9556-52.5.330 [PubMed: 25247726]
 23. Ruparelia K, Abubakar A, Badoe E, et al. Autism Spectrum Disorders in Africa: Current Challenges in Identification, Assessment, and Treatment. *J Child Neurol*. 2016;31(8):1018–1026. doi/10.1177/0883073816635748 [PubMed: 26979098]
 24. Dhadphale M, Lukwago MG, Gajjar M. Infantile autism in Kenya. *Indian J Pediatr*. 1982;49(396):145–148. doi: 10.1007/BF02914974 [PubMed: 7106921]
 25. Mandell DS, Novak M, Zubritsky C. Factors associated with age of diagnosis among children with Autism Spectrum Disorders. *Pediatrics*. 2005;116(6):1480–1486. [PubMed: 16322174]
 26. Bilder D, Pinborough-Zimmerman J, Miller J, McMahon W. Prenatal, Perinatal, and Neonatal Factors Associated With Autism Spectrum Disorders. *Pediatrics*. 2009;123(5):1293–1300. doi: 10.1542/peds.2008-0927 [PubMed: 19403494]
 27. Guinchat V, Thorsen P, Laurent C, Cans C, Bodeau N, Cohen D. Pre-, peri- and neonatal risk factors for autism. *Acta Obstet Gynecol Scand*. 2012;91(3):287–300. doi: 10.1111/j.1600-0412.2011.01325.x. [PubMed: 22085436]
 28. Zhang X, Lv CC, Tian J, et al. Prenatal and perinatal risk factors for autism in China. *J Autism Dev Disord*. 2010;40(11):1311–1321. [PubMed: 20358271]
 29. Curran EA, O'Neill SM, Cryan JF, et al. Research Review: Birth by caesarean section and development of autism spectrum disorder and attention-deficit/hyperactivity disorder: A systematic review and meta-analysis. *J Child Psychol Psychiatry Allied Discip*. 2015;56(5):500–508. doi: 10.1111/jcpp.12351.
 30. Curran EA, Dalman C, Kearney PM, et al. Association Between Obstetric Mode of Delivery and Autism Spectrum Disorder. *JAMA Psychiatry*. 2015;72(9):935–942. Doi: 10.1001/jamapsychiatry.2015.0846 [PubMed: 26107922]
 31. Wang C, Geng H, Liu W, Zhang G. Prenatal, perinatal, and postnatal factors associated with autism. *Medicine (Baltimore)*. 2017;96(18):e6696. doi: 10.1097/MD.0000000000006696. [PubMed: 28471964]
 32. Walker C, Krakowiak P, Baker A, Hansen R, Ozonoff S, Picciotto I. Preeclampsia, Placental Insufficiency and Autism Spectrum Disorder or Developmental Delay. *J Pediatr*. 2015;169(2):154–162. doi: 10.1001/jamapediatrics.2014.2645. [PubMed: 26585995]

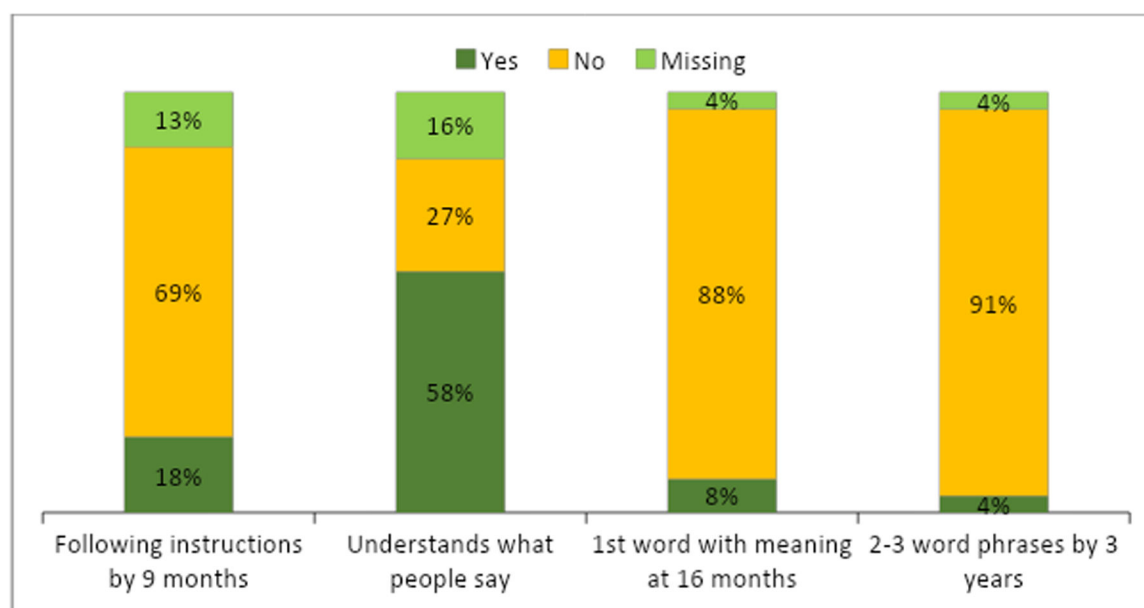


Figure 1.
Bar graph illustrating language development in children with autism

Table 1

Demographic characteristics and summary of presenting complaints.

Age (years)	n = 116	%
1–3	62	53.4
4–6	33	28.5
7–9	11	9.5
10–12	7	6.0
13–23	3	2.6
Sex		
Male	94	81
Female	22	19
Residence		
Urban	110	94.8
Rural	6	5.2
Chief complaint	N	%
Speech delay	110	94.8 (90.8% –98.8%)
Hyperactivity	59	50.9 (41.8%–60%)
Short attention span	25	21.6 (14.1%–29.1%)
Aggressive behaviour	19	16.4 (9.7%–23.1%)
Poor motor function	10	8.6 (3.5% – 13.7%)
Learning difficulties	9	7.8 (2.9%–12.7%)
Jerky movements	7	6 (1.7%– 10.3%)
Staring episodes	6	5.2 (1.2% –9.2%)

Note: Some patients had more than one complaint recorded.

Table 2**Maternal factors and birth history**

Variable (n=116)	N	%
Birth history		
Term births	84	72.4
Preterm births	10	8.6
Missing data	22	19
Mode of delivery		
Vaginal delivery	51	43.9
CS due to slow progression of labor	15	12.9
CS due to fetal distress	9	7.8
CS due to previous scar	9	7.8
CS due to other medical conditions	5	4.3
CS due to placenta praevia	4	3.4
CS due to breech presentation	3	2.6
Missing data	20	17.2
Birth complications		
Uncomplicated	72	62.1
Birth asphyxia	21	18.1
Meconium staining	2	1.7
Complicated delivery-other	5	4.3

Table 3:

Screening of patients (n=31) below 36 months of age using M-CHAT-R screening tool

Level of risk	N	%
Low risk (0–2 risk responses)	0	0
Medium risk (3–7 risk responses)	15	48.4
High risk (8–20 risk responses)	16	53.3

Table 4

Patient characteristics and findings on MRI Brain

Age (Months)	Sex	WMH Region	Chief Complaint	Psychiatric Comorbidities	Epilepsy
71	M	Bilateral Frontal	Speech Delay, Hyperactivity, Learning Difficulty	ADHD	Y
52	M	Right Centrum Semi Ovale	Speech Delay, Concentration Difficulty, Aggression	ADHD	N
84	M	Bilateral frontal	Speech Delay	Elimination Disorder	N
30	F	Right Frontal	Speech Delay, Hyperactivity	ADHD	N
115	F	Right Lentiform Nucleus	Speech Delay, Hyperactivity, Aggression	ADHD, Sleep Disorder	N
69	M	Subcortical	Speech Delay	None	N
52	M	Bilateral Frontal	Speech Delay, Hyperactivity, Motor Deficit, Concentration Difficulty	None	N
48	M	Bilateral Peritrigonal	Speech Delay, Hyperactivity	Other NDD	N
72	M	Left Frontal	Myoclonic Jerks	ADHD	Y
48	M	Left Frontal Subcortical, Bilateral Centrum Semiovale	Speech Delay, Hyperactivity	ADHD	N
32	M	Left Frontal Subcortical	Speech Delay	Sleep Disorder	Y
16	M	Corpus Callosum	Speech Delay, Jerky movements	Elimination Disorder	Y

* WMH = White Matter Hyperintensities, NDD = Neurodevelopmental Disorder, ADHD = Attention Deficit/Hyperactivity Disorder