Autism Epidemiology, Risk Factors and Management Updates

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Abstract

Background: Autism spectrum disorder (ASD) is a group of rapidly growing disabilities characterized by impairments in social and communication and limited interests and repetitive behaviors. It is a complicated neurological disorder that is characterized by behavioral and psychological problems in children as well as cognitive impairments of social interaction, social interaction and social creativity, with a limited interest spectrum and highly stereotyped repetitive attitudes and manners. Autism is known as a devastating condition which affects both the life of a child and the family.

Aim: This review provides an overview of the prevalence, clinical presentation, diagnosis and treatment response in ASD.

Conclusion: ASD is a neurodevelopmental disorder characterized by limited social communication and restricted interests and repetitive behaviors. Research continues to reveal factors that correlate with ASD which may guide further etiologic investigation, but no final causal pathway has been elucidated. Evaluation begins with developmental screening of the general pediatric population to identify at-risk children. Earlier detection and intervention efforts are improving the long-term functioning of children with ASD. Part of the treatment program is strongly structured and based on behavior. Family counseling is typically part of the general treatment plan, involving training the parents so that they can accept the child therapies at home. Therapies in the fields of vocabulary, voice, the development of cognitive skills, physical and sensory integration should all be provided according to the particular child's needs.

Keywords: Autism; Autism Spectrum; ASD Management; Risk Factors of Autism

Introduction

Autism spectrum disorder (ASD) is a group of rapidly growing disabilities characterized by impairments in social and communication and limited interests and repetitive behaviors [1]. It is a complicated neurological disorder that is characterized by behavioral and psychological problems in children as well as cognitive impairments of social interaction, social interaction, and social creativity, with a limited interest spectrum and highly stereotyped repetitive attitudes and manners [2]. This condition was described as a devastating disease which affects both the life of a child and the family [3].

The American psychiatrist Leo Kanner used the term “early infantile autism” in 1943 to describe children lacking interest in others. An Austrian pediatrician, Hans Asperger, identified another group of children with similar symptoms, but with milder frequency and im-
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proved mental ability, separately in 1944 [4]. Autism was considered basically a type of psychosis related to childhood schizophrenia in the 1960s and 1970s and the prevalent popular theory was that it could be primarily associated with parenting styles [5]. The definitions of this condition within psychology have evolved dramatically over the last 65 years, in tandem with the advancement of the idea that mental and behavioral problems have an evolutionary etiology centered on the brain. Thus, lacking reliable biomarkers, ASD continues to be defined by symptoms alone, being evaluated by behavior observation and by asking caregivers questions [6].

Autism spectrum is estimated to have a prevalence of 1 in 68. Childhood disintegrative condition is a rare disease, with just 1.7 in 100,000 cases and this illness is estimated to have an incidence of 1 or 2 in 100,000 [7].

It is not clear what causes this condition and it is often found that children with this disorder have achieved normal developmental stages before skill regression. The age at which this disease occurs is unpredictable, although it is usually seen after reaching normal thresholds three years earlier [8]. However, before the condition is clear, certain children have still affected but these differences are not necessarily noticeable in young children. Risk factors may include prenatal attacks such as inflammation with rubella, unresolved metabolic conditions such as phenylketonuria, maternal anticonvulsants, sporadic tuberous sclerosis lesions, and postnatal diseases such as encephalitis [9].

ASD diagnosis is usually made during childhood, based on comprehensive psychiatric assessments from children's psychiatrist or psychological professionals or experts in clinical and developmental pediatrics [10]. ASD diagnosis process may require referral from a pediatrician or other primary care provider to a clinical center or care provider experienced in ASD diagnosis. Clinical examination to check for symptoms and signs of associated disorders (notably seizure disorder or epilepsy) is recommended. Although sometimes associated with single-gene conditions (notably fragile X and tuberous sclerosis) [11].

Advances in adaptive and dysfunctional behavioral psychology, neurobiology, and autism spectrum disorder pathology have resulted in increasingly new clinical management strategies. Initial approaches to treatment with ASD based on psychotherapy but it was clear over time that children with ASD were most likely to benefit with formal, special education services [12]. Many important contributions to more effective treatment included the introduction of the Support for All Handicapped Children Act, which required education as a right for special needs children; including those with and the official recognition of ASD in 1980 [13].

This review provides an overview of the prevalence, clinical presentation, diagnosis, and treatment response in ASD.

Prevalence

Increasing numbers of children diagnosed as having autistic spectrum disorders have been reported. According to reports by the CDC’s Autism and Developmental Disorder Monitoring (ADDM) network, approximately 1 in 54 children have been diagnosed with autism spectrum disorder (ASD). Studies in Asia, Europe and North America reported individuals with ASD with an estimated prevalence of 1% to 2% [14].

Risk factor

The association between parental psychiatric history and risk of child mental disorders, particularly autism, is evident given the importance and impact of family unit, parental behavior, and their communication patterns on the formation of children's personality and emotions [15]. In addition to mothers that have encountered mental disorders in their life and are known as mental illnesses, others who encounter behavioral issues such as depression, anxiety and significant stress throughout 21 - 32 weeks of gestation, a period of increased plasticity for fetal formation and growth may have irremediable consequences through epigenetic mechanisms on the expression of fetus stress response genes which are involved in neurobiology, metabolism, and physiology that can persist across the lifespan [16].

Also, metabolic syndrome, bleeding (which is associated with a significant 81% elevated risk of autism), and infection of mother during pregnancy are related to child autism [17]. Metabolic syndrome, including diabetes, hypertension, and obesity, may induce utero hypoxia that contributes to defective brain development and activation of changes in myelination, membrane adhesion, and hippocampal neuron deficiency [18].

Maternal viral infections in the first trimester of pregnancy, including rubella, measles, mumps, chicken pox, influenza, herpes, pneumonia, syphilis, varicella zoster, and cytomegalovirus and bacterial infections in the second trimester which require hospitalization, increase the risk of autism in embryo [19]. This association can be explained as an elevated maternal immune activation and thus elevated rates of inflammatory cytokines that influence the growth of the fetal brain and raise the risk of autism and other neuro-pathophysiological disorders [20].

Several researches on various types of medications have shown a significant 68% elevated risk of autism in addition to prenatal therapeutic treatment use. According on several studies, the detrimental impact of prenatal drug use is triggered by their placental crossing and disrupting fetal development [21]. Using antiepileptic medications as valproic acid leads to fetal valproate syndrome, increases oxidative stress and variable variations of gene expression, resulting in developmental delays, impaired motor skills and social interactions, and eventually postnatal growth changes. Positive connection between antidepressant medications and autism has been demonstrated in many studies [22].

Taking into account the cultural, social, educational, and psychological aspects of family life, autistic children and their families are often of poor condition. Basically, due to financial difficulties, workplace and psychological pressures these families inevitably experience toxic, inadequate sociality and unrehabilitated conditions of life [23].

Maternal and paternal age older than or equal to 34 years have been shown in several studies to be correlated with greater risk of autism in their offspring; however, in some research, the association between child autism and the age of both parents or only one parent’s age is rejected [24].

Some of the most significant risk factors are low birth weight, jaundice and postnatal infection. A birth weight neonate arising from three possible factors (genetic growth capacity, pregnancy length and fetal growth rate) below 2500 g considered poor birth weight and correlated with the double increase in autism risk [25].

**Diagnosis**

Children classified as at risk with autism spectrum should be forwarded with thorough developmental and psychiatric evaluation [26]. This evaluation may be carried out by community services, educational providers, or physicians with local development. Such assessments should be multidisciplinary, covering key symptoms, language, cognition, and behavioral, auditory, and motor skills [27].

Assessment of cognitive and linguistic functioning is also critical, as one does not expect to see these adaptive social responses and joint attention behaviors in children with a mental age that is lower than the age at which these developmental attainments appear in typical development [28]. Thus, it can be very challenging to determine whether a child with marked cognitive delays has ASD. In addition to delays or deficits in social and communicative behaviors, the presence of unusual prosody or intonation in word or nonword utterances, repetitive movements (with or without objects), and perseveration in play with objects are red flags that can aid in identifying very young children with ASD [29]. From the first years of life, children with ASD show a great deal of heterogeneity with respect to the severity, onset, course, and constellation of ASD symptoms exhibited; cognitive, linguistic, and communication impairments; social-emotional, behavioral, and regulatory problems and sensory sensitivities. There is diversity in early expression which is consistent with heterogeneity [30].
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Present guidelines from both the American Academy of Child and Adolescent Psychiatry and the American Academy of Pediatric provide routine behavioral monitoring for ASD signs in young children [31].

To meet full criteria for a DSM-IV diagnosis of AD, a child must demonstrate the following symptoms [32]:

1. Qualitative deficit in social contact as demonstrated by two of the following: failure in the use of various nonverbal activities (e.g. eye gaze, facial expression, postures of the body), inability to establish peer relationships, lack of joy sharing or lack of social or emotional reciprocity.

2. Communication impairment in at least one of the following: delay or total lack of language, impairment in the ability to initiate or sustain a conversation, stereotyped or repetitive use of language, or lack of varied spontaneous play.

3. Limited regular and stereotyped patterns of attitudes, desires, and experiences as evidenced by problems with one or more restricted patterns of interest, inflexible obedience to non-functional habits or practices, repetitive physical mannerisms, or obsession for object pieces.

Management

Despite substantial economic and social costs, there are few management options to reduce ASD-related symptoms, including both diagnostic-related symptoms and those considered to be a result of comorbid psychiatric and medical disorders known to exacerbate the severity of the symptoms. There are numerous challenges for the identification of effective treatments for ASD [34]. There are many factors that may reduce the potential effect size of an intervention like possibility that genetic, environmental, cognitive, and social heterogeneity in the ASD phenotype produce highly variable study samples, small sample sizes, the lack of significantly impaired study participants and the use of outcome measures that are not uniformly adopted or used as intended [35]. Certain interventions might be more appropriate for specific individuals at specific stages of development or based on a specific level of skill acquisition [36].

There are only two pharmaceuticals approved by the US Food and Drug Administration (FDA) for management of ASD symptoms, risperidone and aripiprazole [37]. Risperidone is an antipsychotic which was approved in 2006 for the symptomatic treatment of irritability, aggression, deliberate self-injury, and tantrums, in autistic children. This drug acts by blocking the brain’s receptors for dopamine and serotonin [38]. Risperidone is safe and effective for short-term management, with improvements observed in stereotypic behavior and hyperactivity of treated children. Side-effects associated with risperidone include weight gain due to increased appetite, drowsiness, and increased levels of the hormone prolactin, which is produced by the pituitary gland and which have a feminizing effect on females and males. Those side-effects appear to be dose-related [39].

Behavioral interventions can be done for children using an intensive delivery format, are the gold-standard management for behavioral symptoms associated with ASDs. Behavioral interventions require extensive resources to execute effectively and are expensive to implement which makes them inaccessible for many children with ASD and their families [40].

The treatment response to ASDs appears to be based primarily on validated clinical functional observations. Focusing entirely on clinical diagnosis criteria and care reaction introduces a two-dimensional phenomenological approach to ASDs [41]. To match the current cognitive and behavioral needs of the infant, interventions for young children with ASD that are intended to offer appropriate cognitive opportunities have to be individualized. The aim of discussing a variety of interventions for young children with ASD is to provide a brief explanation not meant to be comprehensive or definitive on appropriate approaches to intervention [42].

Conclusion

ASD is a neurodevelopmental disorder characterized by limited social communication and restricted interests and repetitive behaviors. Research continues to reveal factors that correlate with ASD which may guide further etiologic investigation, but no final causal pathway has been elucidated. Evaluation begins with developmental screening of the general pediatric population to identify at-risk children. Earlier detection and intervention efforts are improving the long-term functioning of children with ASD. Part of the treatment program is strongly structured and based on behavior. Family counseling is typically part of the general treatment plan, involving training the parents so that they can accept the child therapies at home. Therapies in the fields of vocabulary, voice, the development of cognitive skills, physical and sensory integration should all be provided according to the particular child’s needs.

Bibliography


