



Autism Spectrum Disorder Causes and Treatment: A Review Research

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Abstract

Autism Spectrum Disorders (ASD) includes complex neurodevelopment disorders associated with characteristic symptoms which manifest in children at three years of age. These characteristic symptoms include impairments in social interaction and communicative skills as well as presence of restrictive, repetitive, pervasive and stereotypic behaviour. Aim of the present research was to focus on causes factors and method of treatment for autism spectrum disorder. In the present investigation related research papers and articles were studied and analyzed in the light of ASD. Many researches show that biological genetically and environmental factors are correlated with ASD. Drug therapy and psychological intervention found effective in treatment of autism disorder.

Keywords: Autism Spectrum Disorder

Background of the study:

Autism Spectrum Disorder (ASD) is characterized by repetitive and restrictive interests, problems with communication, and impaired social functioning (American Psychiatric Association, 1994). ASD affected 9 in 10,000 individuals (Fombonne, 2003). It is three times more likely to occur in males than in females (Burd, Fisher, & Kerbeshian, 1987).

The defining characteristics of autism were first described by Kanner (1943, pp. 242-246) in relation to a group of 11 institutionalized children whose behaviour he observed to be marked by “extreme autistic aloneness, an anxiously obsessive desire for the preservation of sameness, excellent rote memory, delayed echolalia, and limitations in the variety of spontaneous activity”. With specific regard to social interaction, Kanner (1949, p.416) further noted that the individuals with autism whom he had observed showed either “profound withdrawal from contact with people” or “mutism or the kind of language that does not seem intended to serve the purpose of interpersonal communication”.

In recent years, autism has been placed together with Asperger Syndrome (AS) and Pervasive Developmental Disorder- Not Otherwise specified (PDD-NOS), within the broad classification of Autistic Spectrum Disorder (DSM-IV; American Psychiatric Association, APA, 1994). Although people with autism share a range of core symptoms, the severity and the exact nature of symptoms displayed vary widely between individuals. ASD currently affects approximately one in every 68 individuals (CDC, 2014). It is the most commonly diagnosed developmental disorder.

Clinical picture of ASD

Autism is a disorder occurring in childhood that interferes with the normal course of social, communicative, and cognitive development. Autism is necessarily a psychological disorder of very early childhood because of the diagnosis of autism is ruled out if the disorder is first manifested later than the third year of life. The other serious psychological disorders of childhood, attention deficit disorder, anxiety, and depression, begin later in life, although there may be precursors earlier on. Children with autistic disorder show Triad impairment that characterize autism are-

Difficulties with social interaction: - The hallmark feature of autism is impaired social interaction. It is often said that a child with autism —is the beautiful child in a glass cell. A child with autism may appear to develop normally and then withdraw and become indifferent to social engagement. They fail to respond to their names, avoid eye contact, fails to imitate others. severe difficulty in initiating and maintaining social interactions and relationships, confusion about the

effects and consequences of many of his or her behaviours, and engagement in restrictive and repetitive behaviours and interests that may limit the child's ability to learn and to fit in with peers. From a teacher's or parent's perspective, problem behaviours include lack of compliance with or disruption of classroom routines, tantrums, destruction of property, and aggression against self or others.

They find social interactions to be unnatural and quite stressful. Rather than embracing relationships, they try to avoid them, choosing rather to take refuge and comfort in their own isolated worlds. They do not reciprocate play and they do not engage in normal play activities without prompting. They also avoid meeting other people's gaze, and tend instead to fixate their eyes away from people, on to inanimate objects or parts of objects (Kuba, 2014).

Problems with verbal and nonverbal communication: - Children with autism display serious difficulties in communication and language that appear early in life and persist over time. Children with autism are inconsistent in using early preverbal communication. They may use instrumental gestures to get someone else to do something for them immediately and they fail to use expressive gestures to convey feelings.

Lord, Pickles, DiLavore, and Shulman (1996) found that two year-old children judged very likely to have autism had mean expressive and receptive language ages of less than 9 months, in contrast to other skills falling between 16 and 21 months. Not only was their expressive skills continued to develop at a slower rate through age five compared to nonautistic children with developmental delays at similar nonverbal levels. Chan, Cheung, Leung and Cheung (2004) examined the verbal expression-comprehension abilities of 46 Chinese children with autism at age's five to six. Results showed that 63% of the children with autism demonstrated language impairment. Specifically, 42% were impaired in both verbal expression and comprehension abilities, and 21% demonstrated impaired expression skills. Some other studies mainly focused on language regression in autism. Kurita (1985) found that about 25% of children with autism are described by their parent as having some words at 12 to 18 months and then losing them.

A large-scale systematic longitudinal study of toddlers by Lord, Shulman, and DiLavore (2004) found that language regression after a pattern of normal language onset was unique to autism and not found among children with other developmental delays. Lord and her colleagues found that children who experienced loss of words also lost some social skills, supporting the findings from Goldberg and her colleagues (Goldberg et al., 2003), and that similar losses of social skills occurred in a smaller group of children with autism who had not yet used words at time of loss (Luyster et al., 2005). Charman et al. (2003) observed that in the preschool period and beyond, certain nonverbal skills, especially the frequency of initiating joint attention, and imitation, are strong predictors of language acquisition for children with autism.

Repetitive behaviors or problem in flexibility: - Another significant feature of autism is inflexibility. They are resistant to change. It is pervasive for all sense organs. So, mostly sensory dysfunction is common in them. Because of stimulus over selectivity (Lovaas et al., 1979) they cannot filter the desirable sensation and so due to habituation in sensory adaptation it is difficult for them to change.

Brain Size

Kanner's seminal paper noted that children with autism had enlarged heads. Although subsequent studies of external head circumference confirmed this observation, it did not receive much attention until the past decade when support for this finding began to accumulate via MRI, postmortem studies, and additional head circumference studies. The size increase appears to be a shifting of the entire autism brain and head size distribution rather than merely an excess of megalencephaly among a minority of cases that elevates what would otherwise be a normal autism population mean size (Volkmar et al., 2004).

According to Courchesne et al. (2003) both MRI volumetric analysis and simple measures of head circumference indicate that autism involves transient postnatal macrencephaly. Aylward et al. (2002) found that neonates later diagnosed with autism or PDD-NOS have normal head circumference; but by 2-4 years of age, 90% of these have larger-than-average brain volumes. Recent findings suggest that the brain may be enlarged by as much as 10% in volume in toddlers with autism. However, the magnitude of the effect appears to diminish with age. In

adolescence and adulthood the effect is less consistently found across studies and the size of the effect is diminished to a few percent increases at most. Several studies have shown the head is not significantly enlarged at birth leading to the proposal that there are specific events in the first months of life that are responsible for the brain enlargement.

Corpus Callosum

Although different studies have noted various segments of the corpus callosum to be abnormal, they consistently find a reduced callosal size. Indeed, the posterior corpus callosum in autism is actually smaller than normal and the degree of this callosal hypoplasia correlates with the degree of frontal hyperplasia. This compartmental specificity of white matter hyperplasia is consistent with the idea of differential effects on local and long -range connections (Baron-Cohen & Belmonte, 2005). Reductions in white matter may also be consistent with PET study that showed reduced interregional correlations in persons with autism, suggesting reduced functional integration and connectivity. One consequence of reduced inter connectivity in the autistic brain might be increased modularity and reduced integration of functions. Such a reduction in neural integration would be consistent with one influential theory that attributes autistic symptoms to a lack of ‘central coherence’ a cognitive processing style that makes integration of parts into wholes problematic (Volkmar et al., 2004).

Cerebellum

Studies showed that autistic children exhibited clinical and neurophysiologic deficits indicative of cerebellar dysfunction (cited by Ciaranello & Ciaranello, 1995). The cerebellum, in particular, is one of the most consistently abnormal structures in the autistic brain. Post mortem neuropathology studies have revealed that very consistent reductions in the number of Purkinje neurons in the cerebellum. The precise nature of these abnormalities, including a lack of gliosis, suggests a prenatal origin. Although classical neuropsychological models see the cerebellum as strictly a motor system, some evidence finds a role for it in cognition, sensation and attention especially as it participates as part of larger cerebellar-cortical systems. In autism, dysfunctions of cerebellar-cortical serotonergic pathways have been noted and fMRI evidence implicates the cerebellum in attention difficulties seen in autism (Volkmar et al., 2004).

Genetic Determinants of ASD

Ultimately the cognitive and neural abnormalities in autism spectrum conditions are likely to be due to genetic factors. The sibling recurrence risk for autism is approximately 4.5% or a tenfold increase over general population rates (Jorde et al., 1991). An epidemiological study of same sex autistic twins found that 60% of monozygotic pairs were concordant for autism versus 0% of dizygotic pairs (Baily et al., 1995). When a broader phenotype was considered, 92% of monozygotic pairs were concordant versus 10% of dizygotic pairs. The high concordance in monozygotic twins indicates a high degree of genetic influence, and the risk to a monozygotic co-twin can be estimated at more than 200 times the general population rate (Baron-Cohen & Belmonte, 2005). Although single-gene disorder and chromosomal abnormalities are likely to be responsible for only a fraction of all autism cases, the cooccurrence of autistic behaviour and certain neurogenetic disorders such as Fragile X syndrome, Tuberous sclerosis complex, Rett syndrome, Chromosome 15q duplication syndrome, and many others is more evidence for a genetic etiology (Sigman et al., 2006). Autism which results from mutation in a single gene. Under monogenic autism we explain about the copy number variations (CNVs) in the genes which are involved in synapse formation, such as genes for cell adhesion molecule, scaffolding proteins, cytoskeletal proteins, signaling pathways, ion channels and cell signaling molecules. Mutation in these genes disrupts regulatory or coding regions which contribute to the pathogenesis of ASD by affecting synapse formation, plasticity as well as transmission.

Neurochemical Effect on ASD

ASD is thought to have an associated serotonergic dysfunction, which is apparent from the work published by Schain and Freedman way back in 1961 (Schain and Freedman, 1961). They observed higher serotonin level in whole blood cells. Subsequent research on this aspect by many groups (Abramson et al. , 1989, Bijl et al. , 2015, Cook et al. , 1993, Gabriele et al. , 2014) supported this finding and suggested that the trait is familial. Moreover the study by Anderson et al. demonstrated that the observed higher blood serotonin level was present mainly in the platelets while maintaining the plasma level in the normal range (Anderson et al. , 1987). This

was replicated by several groups supporting platelet hyperserotonemia as a well-identified endophenotype for ASD (Anderson et al., 1990, Cook et al., 1988, Hranilovic et al. , 2008, Piven et al. , 1991). Various factors have been investigated as contributory causes for platelet hyperserotonemic condition, which include increased synthesis as well as decreased degradation of 5-HT and defects in the 5-HT signaling cascade including transporter and receptor functions (Hranilovic, Novak, 2008, Veenstra-VanderWeele et al. , 2012).

Specific immune dysfunction

Dysfunction The possible involvement of the system in the pathogenesis of autism is another area of intense research. Various mechanisms have been postulated, including exposure to infectious agents and / or specific immune dysfunction. Studies demonstrating increased risk of autism in certain geographical regions (Gillberg et al., 1991) or for particular seasons of implicate exposure to various infectious agents during early pre-or postnatal development. The best studied of these is the increased risk of autism with congenital rubella syndrome (Chess 1971). Others have invoked this mechanism for the putative causal role of the measles, mumps, and rubella vaccine (Wakefield et al., 1998), although this has largely been refuted by epidemiological studies (Madsen et al., 2002; Taylor et al., 1999). It has also been suggested that individuals with autism have a disordered immunity with both decreased and increased function of different segments of the immune system having been reported (Horing & Lipken, 2001). Recent evidence shows subtle signs of inflammation in the brains and cerebrospinal fluid of individuals with autism (Vargas et al., 2005). The immunological mechanism could be part of a complex cascade of events that are involved with the etiology of autism related to and modulated by genetic predisposition and / or environmental triggers during critical periods in development.

Psychological factors

Treatment

Recently, the trend has been to use more developmental approach to interventions, targeting the areas that have been shown to be at deficit in children with autism. Given that young children with autism show less social orienting and imitation, less capacity to follow the gaze and pointing gestures of others, less referencing of others in ambiguous situations and sharing of

interesting experiences, and less pretended play, interventions have been developed that focus on increasing all of these social skills and behaviours. An increasing number of interventions also attempt to foster peer interaction both in mainstreamed and special school programmes. Most of these developmental interventions substitute relationship-based approaches for the more didactic form of teaching that characterizes the behaviourally based interventions.

According to Bergman and Gerdtz (1997) behavioural interventions have the predominant treatment approach for promoting the social, adaptive, and behavioural functions of children and adults with autism. Behavioural approaches have been adopted increasingly for enhancing personal independence and responsible choice through skill development and habilitative training, increasingly repertoires of prosocial behaviour and leisure activities, and teaching methods of self-control and relaxation. In addition, behavioural interventions have been employed for reinforcing adaptive responses and suppressing maladaptive ones.

In one important study by Ozonoff and Cathcart (1998) found that students who received home-based structured teaching significantly improved on developmental and cognitive tasks in the areas of imitation, fine motor skills compared to students who did not receive this type of intervention at home. In addition, the experimental group exceeded the control group on other developmental measures by two to three times and gained 9.6 months after 4 months of treatment.

Similar benefits from applied behaviour analysis were found by Green, Brennan and Fein (2002) a toddler to be high risk for autism at the age of about 1 year. Treatment was delivered in the child's home and other settings. Intensive treatment continued for 3 years; by the 4th year, the child was spending most of her time in a regular preschool classroom. Direct observational data and results of norm-referenced tests documented large increases in language, social, cognitive, and daily living skills over the course of treatment.

Hwang and Hughes (2000) reviewed 16 empirical studies that investigated the effects of social interactive interventions designed to increase early social communicative skills of young children with autism by increasing their role as initiator of social interactions. Increases were found for social and affective behaviors, nonverbal and verbal communication, eye contact, joint

attention, and imitative play. Limited generalization or maintenance of target behaviors was reported.

Discussion

Autism is a disorder occurring in childhood that interferes with the normal course of social, communicative, and cognitive development. Autism is necessarily a psychological disorder of very early childhood because of the diagnosis of autism is ruled out if the disorder is first manifested later than the third year of life. The other serious psychological disorders of childhood, attention deficit disorder, anxiety, and depression, begin later in life, although there may be precursors earlier on. Because autism begins so early, be disentangled from the predetermined systematic disturbances.

In the present research trying to describe causes and treatment of ASD. for the present purpose some research were analyzed and summarized .many research focused on severity of ASD in all over word and some of them focused on symptoms ,characteristics and causes of Autism Spectrum Disorder (ASD). Some research clear clinical picture of autism disorder, how children with ASD face difficulties in social interaction and how lack social interaction affect on their overall development. Children with ASD face communicational issues they cannot develop their communication skill according to their age. Cheung (2004) examined the verbal expression-comprehension abilities of 46 Chinese children with autism at age's five to six. Results showed that 63% of the children with autism demonstrated language impairment. Specifically, 42% were impaired in both verbal expression and comprehension abilities, and 21% demonstrated impaired expression skills. Some other studies mainly focused on language regression in autism. Kurita (1985) found that about 25% of children with autism are described by their parent as having some words at 12 to 18 months and then losing them. Some behaviour pattern is common in children with autism disorder such as repetitive behaviour and inflexibility. They cannot filter the desirable sensation and so due to habituation in sensory adaptation it is difficult for them to change. Many study highlighted that children with ASD may have some other psychological issues such as ADHD, PTSD, Depression, Anxiety and aggression. Biological factors also studied by many researchers. Some researcher focused on brain and its part according to them brain size found abnormal in patient of autism also found that abnormality in corpus coliseum

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and cerebrum. Some neurotransmitter also responsible in autism disorder one of them is serotonin. Genetically abnormality found correlated with ASD. Some other study also focused on treatment of autism disorder in present review research only psychological intervention method was highlighted behavioural intervention method is also useful in treatment of autism disorder.

Conclusions

Autism spectrum disorder is a neurodevelopment disorder. Many research shows that biological genetically and environmental factors are correlated with ASD. Drug therapy and psychological intervention found effective in treatment of autism disorder.

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