Future Target of Treatment for Social Impairment of Autism Spectrum Disorders

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Introduction

Impairment in social and communication function is the core symptoms of autism spectrum disorder (ASD). ASD is a common neurodevelopmental disorder affecting approximately 1% of children. The ability to understand emotion in others is essential for the health development of human. Abnormalities of emotion perception may contribute to psychiatric disorders associated with social problems in ASD. There are no known efficacious treatments for the core social impairment although effects such as risperidone and aripiprazole, and selective serotonin reuptake inhibitors (SSRI) have reported their efficacy in the treatment of disruptive repetitive behaviors [1]. Number of antipsychotic drugs such as risperidone and aripiprazole, and selective serotonin reuptake inhibitors (SSRI) have reported their efficacy in the treatment of disruptive repetitive behaviors [1], however, some adverse effects, particularly weight gain, fatigue, agitation, irritability [1] and tardive dyskinesia (Research Units on Pediatric Psychopharmacology Autism Network 2002) are also associated with these drugs. Amitriptyline has been found instrumental for treatment-resistant youth with ASD accompanied by behavioral symptoms (Bhatti et al. 2012). The use of neuropeptide oxytocin has marked its affectivity in reducing the severity of repetitive behaviors; however, this affectivity was found to last only for a short period of 240 minutes after its infusion (Hollander et al. 2003). There still lies a dearth of any such drugs that can commonly used be used for the treatment of the core social and communication impairment that are the hallmark of ASD.

Why there are Less Medical Drugs for Social Impairment in ASD

However, social impairment of ASD is not less complicated and multi-factorial condition compared to that of schizophrenia. There are several reasons for limited efficacy of medical treatment. First, Hans Asperger who had the first described “Asperger disorders” had recommended educational and behavioral training [2,3]. After that, early implementation of educational interventions is the usual form of treatment. Second, ASD is a heterogeneous and multi-factorial condition, and identifying subgroups of individuals will be necessary to gain traction into its pathophysiology and novel opportunities for treatment. Third, major pilot clinical studies have reported that the sample size is generally small, so the resultant conclusions are found to be tentative [4]. Lastly, there are less clinical reports which tried to confirm the efficacy of medical drugs, other various medical compound or polysaturated fatty acids proposed by the pilot studies.

Useful Pharmacological Drugs

Accumulating evidence indicate that the gross abnormalities in these neurotransmitter systems such as serotonin and dopamine systems may underpin the neurophysiologic mechanism of ASD. Particularly, risperidone solution, a novel antipsychotic which combined dopaminergic and serotonergic action, has shown to be effective in impaired social interaction [5, 6]. Oxytocin may mediate the benefits of positive social interaction and emotions [7].

Newer lines of research, including signal transduction and antioxidants, will be of considerable interest in the future. Abnormal functional connectivity, which affects the delivery of afferent signals, may be involved in the pathophysiology of autism spectrum disorders (ASD) [8]. Alternatively, individuals with ASD may be more vulnerable to oxidative stress due to deficit antioxidant defense mechanisms. Polysaturated fatty acids, arachidonic acid (ARA) and docosahexaenoic acid (DHA) play key roles in brain network maturature. The impaired social interactions observed in ASD may be related to altered signal transduction based on alterations in brain architecture or synaptic response. It is well documented that docosahexaenoic acid (DHA) and arachidonic acid (ARA) are both important in brain development, and that ARA plays a key role in signaling related to neuronal development [9]. An alternative explanation may be based on plasma antioxidant activity in response to our supplementation regimen. Oxidative damage has a pathophysiological role [10], and deficient antioxidant defense mechanism system against reactive oxygen species have been proposed in pathophysiology of ASD [11].

These consideration taken together suggest that supplementation with large doses of ARA added to DHA may improve impairment of social interaction of ASD [12,13].

Conclusion

It is important to note that risperidone solution, intranasal oxytocin, and ARA added to DHA (ARA/DHA ratio of 1/1) have been reported to improve impaired social interaction of ASD. Although early implementation of educational interventions are a common component of treatment. Psychopharmacological treatments coupled with educational interventions may induce more excellent impairment of impaired social interaction of ASD. Alternative therapies frequently utilized, of which the clinician needs to be aware. I will encourage researchers to submit their papers on alternative therapy for social impairment of ASD.

References

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