

Autism Spectrum Disorders and the Occurrences of Familial Thrombophilia Disorders - An Early Report ©

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Recently, growing evidence points to dysregulation of the immune system in Autism Spectrum Disorders (ASD). Various vaccines, viruses, pathogens, immune deficiencies, toxins and autoantibodies have been put forward as playing a role in the development of autism related symptoms. Additionally, a familial or genetic tendency has been observed in this condition. Further, ASD has been shown to have an association with its own unique inflammatory bowel disease, characterized by nodular hyperplasia of the submucosal lymph nodes, thickening of the basement membrane and infiltration of delta-gamma T-lymphocytes. Because thrombophilia disorders are also associated with immunological disorders, inflammatory bowel diseases, chronic fatigue/fibromyalgia, and central nervous system conditions including multiple sclerosis and cerebral palsy, it is reasonable to investigate a potential for thrombophilia in ASD. To determine this, nine families representing 10 children with ASD and 16 additional family members (15 parents and 1 sibling), selected at random from an ASD population were tested for coagulation disturbances. An Immune System Activation of Coagulation panel (ISAC) consisting of four tests including: Fibrinogen, Soluble Fibrin Monomer, Thrombin/AntiThrombin Complexes, and Platelet Activation by flow cytometry were measured on all children and available family members. In the children with ASD seven of 10 were positive for at least one marker of thrombophilia (ISAC), while 15 of 16 family members were positive for ISAC. The families were further studied by an Hereditary Thrombosis Risk Panel (HTRP) consisting of AntiThrombin Activity, Protein C level, Protein S level, APC Resistance, Factor II Activity, Lp(a) levels, PAI-1 Activity and Homocysteine levels. In the children with ASD, six of 10 had abnormal findings in the HTRP and 11 of 16 family members showed abnormal activated coagulation factors. By combining ISAC with HTRP, 10 of 10 children with ASD were at risk for thrombophilia disorders and 15 of 16 family members were as well. Eleven of the family members reported symptoms consistent with CFS/Fibromyalgia. This represents a previously unreported high prevalence of familial thrombophilia where at least one member has ASD. The data provide further evidence of immune activation in ASD, which deserves additional study to determine the potential pathophysiological role of thrombophilia in the generation of autism related symptoms. Simultaneously, family members deserve careful evaluation for their own thrombophilia-associated conditions.

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