RESEARCH

65% Autistic Children Found To Have Mitochondrial Disorder

At an American Academy of Neurology meeting last Sunday it was revealed in a recent research paper, see below, that 65% of children with Austistic Spectrum Disorders assessed were found to have mitochondrial disorder (MtD) and so were always at risk of autism caused by one or more vaccines.

Oxidative Phosphorylation (OXPHOS) Defects in Children with Autistic Spectrum Disorders [IN1-1.004]

John Shoffner, Lauren C. Hyams, Genevieve N. Langley, Atlanta, GA

OBJECTIVE: To retrospectively survey patients with autistic spectrum disorders that were evaluated clinically for mitochondrial disease and to assess the clinical and laboratory features of this group of patients.

BACKGROUND: Autism is a developmental disorder characterized by disturbance in language, perception and socialization. A variety of biochemical, anatomical and neuroradiographical studies imply a disturbance of brain energy metabolism in autistic patients. Recent studies confirmed the previously reported high frequency of biochemical markers of mitochondrial dysfunction, namely hyperlactacidemia and increased lactate/pyruvate ratio, in a significant fraction of 210 autistic patients. (J Autism Dev Disord, 2006. 36:1137) Although rare, Mecp2 mutations can produce autistic features and the mouse model has significant mitochondrial defects. (Mol Cell Biol, 2006. 26: 5033) Additional genetic defects associated with mitochondrial dysfunction include inverted 15q11-13 duplication (Complex III defect) (Ann Neurol, 2003,53,801), A3243G mutation (mitochondrial transfer RNALeucine(UUR) gene, mtDNA depletion(J Pediatr, 2004,144,81), G8363A mutation (mitochondrial transfer RNALysine gene. (J Child Neurol, 2000,15,357).

DESIGN/METHODS: Retrospective analysis of 37 children with autistic spectrum disorders. Clinical, biochemical, metabolic, and genetic data is assessed.

RESULTS: Twenty four children (65%) had skeletal muscle OXPHOS defects: Complex I (16), Complex I and Complex III (5), Complex III (1), Complex I and Complex IV (2). Thirteen (35%) had normal skeletal muscle OXPHOS enzyme activities for Complexes I-IV. Clinical, metabolic, protein chemistry, and sequencing of coding regions of the mitochondrial DNA will be reported.

CONCLUSIONS/RELEVANCE: