‘VISUAL SNOW’ - DISTINCT FROM MIGRAINE AURA

Investigators from University of California, San Francisco; King’s College London, UK; and University of Utah, Salt Lake City, studied patients with ‘visual snow’ to characterize the phenotype and compare it to migraine aura. Of 22 patients referred with this diagnosis, 15 had additional visual symptoms, and 20 had comorbid migraine, 5 with aura. Visual symptoms included palinopsia (trailing and afterimages), entoptic phenomena (floaters, spontaneous photopsia), photophobia, and nyctalopia (impaired night vision). Duration of visual snow symptoms varied from “as long as they could remember” in 25%, to a mean age of onset of 21 +/- 9 years in the remainder. Symptoms were constant in some and progressive in others. Worsening of visual snow symptoms in 36% cases was associated with headache, migraine, migraine with aura, anxiety and depression. First degree relatives were affected in 8 patients. (Schankin CJ, Maniyar FH, Digre KB, Goadsby PJ. ‘Visual snow’ – a disorder distinct from persistent migraine aura. **Brain** 2014 May;137(Pt 5):1419-28).

COMMENTARY. ‘Visual snow’ is described as continuous tiny dots in the entire visual field similar to the noise or static of an analogue TV and lasting longer than 3 months. Frequently comorbid with migraine but considered a unique disorder distinct from migraine with aura, complicated by palinopsia, floaters, photophobia, and nyctalopia, and not explained by intake of psychotropic drugs.

AUTISM SPECTRUM DISORDER

NEOCORTICAL DISORGANIZATION AND AUTISM

Investigators from University of California, San Diego, and other centers in the US, assayed markers for neurons and glia and genes implicated in the risk of autism, in prefrontal, temporal, and occipital neocortex. Postmortem tissue samples were obtained from children with autism and unaffected children between the ages of 2 and 15 years. Prefrontal and temporal cortical tissue from 10 of 11 children with autism and from 1 of 11 unaffected children showed focal patches of abnormal laminar cytoarchitecture and cortical disorganization of neurons, but not glia. No cortical layer was spared, layers 4 and 5 being most affected. A probable dysregulation of layer formation and neuronal differentiation is proposed at prenatal developmental stages of children with autism. (Stoner R, Chow ML, Boyle MP, et al. Patches of disorganization in the neocortex of children with autism. **N Engl J Med** 2014 Mar 27;370(13):1209-19).

COMMENTARY. The authors suggest that the mechanism of this laminar disorganization might result from migration defects or de novo changes in early prenatal development. Both genetic and environmental factors contribute to autism liability. In a Swedish population study [1], the risk of autism spectrum disorder (ASD) in family members of persons with ASD was significantly higher than the risk in the general population, and the risk of ASD recurrence among family members decreased with decreasing genetic relatedness, from a 10-fold increased risk of recurrence in full siblings to a 2-fold increased risk of recurrence in cousins. Genetic factors explained half of the

liability for autism [2]. Another population-based Swedish study based only on twins estimated a heritability of 80% [3].

References.
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PARENTAL OBESITY AND AUTISM SPECTRUM DISORDER

Investigators from Oslo, Norway and other centers studied the associations among maternal prepregnancy BMI, paternal BMI, and the risk of autism spectrum disorder (ASD) in children. A study sample of 92909 children aged 4 to 13.1 (mean 7.4) years was derived from a population-based, prospective Norwegian Mother and Child Cohort Study. Among 419 children diagnosed with ASD at end of follow-up (2012), 162 were diagnosed with autistic disorder, 103 with Asperger disorder, and 154 with PDD. Maternal obesity (BMI >30) was only weakly associated with ASD risk, whereas paternal obesity was associated with an increased risk of autistic disorder (AD) and Asperger disorder. The risk of AD was 0.27% in children of obese fathers and 0.14% in children of fathers with normal weight (BMI <25). The risk of Asperger disorder was 0.38% in children (aged >7 years) of obese fathers and 0.18% in children of normal-weight fathers. The adjusted OR for AD was 1.73 and for Asperger disorder, 2.01. Parental obesity was not associated with PDD. (Suren P, Gunnes N, Roth C, et al. Parental obesity and risk of autism spectrum disorder. **Pediatrics** 2014 May 1;133(5):e1128-e1138).

COMMENTARY. Adverse effects of obesity in relation to childhood ASD concern both child and father. In a sample of 376 Oregon children with ASD, 18.1% of children met criteria for overweight and 17.0% met criteria for obesity [1].

Gluten-free casein-free ketogenic diet for autism and seizures. Pediatric neurologists at the Massachusetts General Hospital, Boston, MA, report a girl with autism and pubertal onset of seizures refractory to AEDs who benefited from treatment with a gluten-free casein-free ketogenic diet, with medium-chain triglycerides. Secondary benefits of the MCT diet included resolution of morbid obesity and improved cognition and behavior. The Childhood Autism Rating Scale score decreased from 49 (severe) to 17 (nonautistic); the child was essentially seizure free after 14 months on the diet; and a lengthy 3 Hz spike-wave EEG pattern improved, showing only occasional short discharges without clinical accompaniments [2]. Despite the limitations of a single case, this report seemed worthy of comment.

References.
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