‘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