Rethinking neurobehavioral comorbidity in Panayiotopoulos syndrome

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doi: 10.1111/dmcn.14460

This commentary is on the original article by Wald et al. on pages 985–992 of this issue.

In 2016, *Epilepsy & Behavior* released a Special Issue exploring new ways of thinking about cognition and behaviour,[1] following the revised approach to classifying the epilepsies published by the International League Against Epilepsy (ILAE) Commission on Classification and Terminology (2005–2009).[2,3] At its heart, this new approach uses a descriptive or phenomenological taxonomy to classify epilepsy and its syndromes, allowing all of the features of a syndrome to be carefully characterized. The well-crafted paper by Wald et al. describing the neurocognitive and behavioural profile of Panayiotopoulos syndrome provides an expert illustration of this new approach.[4] In their study, they precisely profile a range of impairments in cognition and behaviour in a syndrome that was previously considered neurocognitively and behaviourally 'benign'. In so doing, the researchers link the cognitive and behavioural features of the syndrome to the seizure semiology and its proposed underlying brain network dysfunction, allowing the essential neurobehavioral comorbidity of this syndrome to be captured.

This brings to light a striking observation, namely the extent to which the subjective psychological profile of attentional and thinking problems in Panayiotopoulos syndrome maps on to the objectively measured deficits, particularly in psychomotor speed, sustained attention, and visuospatial functions. Importantly, this moves us from thinking about cognition and behaviour in terms of the secondary effects of seizures to what may be the more fundamental manifestations of Panayiotopoulos syndrome itself. At the same time Wald et al. measure and capture potential secondary effects by carefully documenting the academic underachievement of their patients relative to seizure and drug treatment variables.

In the supplementary material, Wald et al. provide an excellent level of individual case detail. This clearly captures the spectrum of impairment in Panayiotopoulos syndrome, showing that individuals with more severe neurological features and neurobehavioral comorbidity tend to have more significant academic difficulties. This provides highly practical information for the treating clinician, who must provide prognostic counselling to patients and their families about the educational and psychosocial impact of the syndrome, including consideration of clinical, behavioural, and educational interventions that might minimize poor outcomes.

Going forward, this type of deeper phenotyping is vital for all epilepsy syndromes if we are to link cognitive and behavioural phenotypes with their corresponding neurological phenotypes in this new approach to classification. This, in turn, will assist the identification of causes, genetic or otherwise, allowing us to better understand and treat the cognitive, behavioural, and seizure expression of the underlying network dysfunction. And by leading to a more exact account of the neurocognitive and behavioural features of Panayiotopoulos syndrome, Wald et al. encourage others to profile their own patients more carefully, ultimately improving differential diagnosis and treatment for all patients.

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