Huntington disease

Understanding the psychiatric prodrome of Huntington disease

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Presymptomatic and early symptomatic gene carriers show greater prevalence of depression and irritability than controls. Depressive symptoms worsen with proximity to onset

Huntington disease (HD) is characterised by a triad of movement disorder, cognitive decline and various psychiatric disturbances. This third feature is especially difficult to characterise, particularly in trying to distinguish emotional symptoms which may be reactive in nature from those which are an essential neuropsychiatric aspect of the disease. Depressive symptoms have been said to occur in presymptomatic individuals as many as 20 years prior to motor onset, but high rates of psychiatric disturbance have also been observed in gene negative family members.[1] In the paper by Julien and colleagues[2] in the current issue of *J Neurol Neurosurg Psychiatry*, the authors tackle the question head on, by obtaining structured psychiatric histories from a large group of at risk individuals, prior to disclosure of their genetic status ***(see page 939)***. In doing so, they make a strong case for the idea that affective disorder arises out of structural changes in the brain in HD. There was no elevation in lifetime prevalence of depression, or any psychiatric disorder, but a significant elevation in current prevalence of depressive symptoms, increasing in severity with proximity to onset.

Clinicians who treat HD are familiar with the poignant scenario of an at risk person far from the likely age of motor onset, who has just received a diagnosis of major depression, or some other psychiatric disorder common in the general population. Over interpretation of the old literature fuelled the assumption that the individual must be gene positive, or led to an unnecessary genetic test, at a time of greatest emotional vulnerability. This study shows that, while depressive symptoms close to the time of onset are not likely to be reactive, the window of vulnerability is small. Only individuals less than a year from clinical onset showed a significantly higher prevalence of depression compared with non-carriers.

The situation with ''manic'' symptoms was very different. While the correlation did not rise above the level of a trend ($p<0.06$), there were elevated, but stable, rates of irritability up to 10 years prior to motor onset, which did not convert at any point to classical bipolar disorder. The implication is that, while the course of HD may be punctuated by episodes of syndromal psychiatric disturbances, such as major depression, there are also chronic changes, such as irritability or apathy, which must not be shoehorned into existing diagnostic categories. These symptoms may be isolated or non-specific, or they may belong to syndromes of their own, which have yet to be fully refined.[3] The various psychiatric manifestations of HD have been described qualitatively on a number of occasions.[4] Quantitative research of this type will now help the field to separate intuition from fact.

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