It is illegal to post this copyrighted PDF on any website. Weakness led to delayed diagnosis and unfavorable

With Psychotic Features

To the Editor: I read with interest the report by Yeo et al¹ published previously in the PCC. I have listed statements from the report with comments as follows:

- 1. "It [Huntington's disease; HD] is characterized by a triad of choreiform movements, cognitive decline, and psychiatric disturbances."
 - –Some HD patients never show choreiform movements. This was first suggested by Davenport in 1915.²
- 2. "A healthy individual without HD has around 11–35 CAG [cytosine-adenine-guanine] repeats, whereas an HD patient has 36 or more."
 - -The normal range of CAG repeats end at 26. A number of HD patients with less than 36 CAG in HTT have been reported.³ I know of 2 HD patients with 35 CAG. The probability to manifest HD increases gradually from 0 with 26 CAG to 1 with 43 CAG in the HTT gene.
- 3. "With each successive generation there is an expansion of the CAG repeat number."
 This is incorrect. Expansion of the CAG repeat usually occurs in 60% of paternal transmission.
 Contraction has been documented as well.⁴

A very common mistake in psychiatry is to not take a family history, as was the case in this patient. This critical

weakness led to delayed diagnosis and unfavorable treatment. Previously in 1972, Whittier, the physician who treated Woody Guthrie, and colleagues⁵ wrote about the difficulties faced by psychiatrists in the diagnosis of HD—nothing much has changed.

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