Differentiating Parkinson Disease From Traumatic Encephalopathy Syndrome

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The recent publication detailing Muhammad Ali’s clinical diagnosis of levodopa-responsive young-onset Parkinson disease has provided an educational opportunity for clinicians and trainees to enhance their diagnostic acumen, especially when encountering cases with a history of head trauma. A clinical diagnosis of Parkinson disease is usually made in patients with progressive bilateral asymmetric levodopa-responsive features accompanied by motor and nonmotor symptoms with or without resting tremor, with or without a history of traumatic brain injury. By contrast, progressive cognitive impairment after multiple repetitive impacts to the head can provide insights into other diagnoses, particularly chronic traumatic encephalopathy (CTE).

To move from a diagnosis of CTE syndrome to CTE will require pathology. CTE pathology is commonly characterized by hyperphosphorylated tau (p-tau) protein, which is present as neurofibrillary tangles, astrocytic tangles, and neurites, and these tend to cluster around smaller blood vessels in the cortex and commonly are observed at sulcal depths. CTE pathology is common in the frontal and medial temporal lobes, as well as the thalamus and brainstem. TDP-43 pathology also commonly occurs in most cases with CTE. CTE = chronic traumatic encephalopathy; TDP-43 = TAR DNA-binding protein–43.
the head, with or without neurobehavioral dysregulation, with or without a postural action tremor (which may be transitory), more commonly leads to a diagnosis of traumatic encephalopathy syndrome. This teaching aid can be used to highlight these important features and to illustrate that it is possible for the syndromes to co-occur inclusive of their respective pathologies. Finally, all clinicians should appreciate that head trauma is a risk factor for the later occurrence of Parkinson disease (Figure).

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Reference