

In Response To:

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Letters

Author Response to Letter to the Editor

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Dear Editor,

This is an author response to the letter by doctors Nicholas Dohar and Harsh V Gupta.

Despite the narrow indication for the dopamine transporter (DAT) scan to distinguish essential tremor from Parkinsonian syndromes, as approved by the Food and Drug Administration (FDA), (Available from: http://www.accessdata.fda.gov/drugsatfda_docs/nda/2011/022454sOrig1s000Lbl.pdf) DAT imaging is utilised for various other clinical symptoms in patients with parkinsonism: (1) to differentiate Parkinson’s disease (PD) from atypical parkinsonism (dementia with Lewy bodies (DLB), multiple system atrophy (MSA), corticobasal degeneration (CBD), etc.), vascular parkinsonism or psychogenic parkinsonism by revealing different patterns of decreased DAT uptake or the lack thereof,^{1,2} (2) as a marker, in PD prognostics, in that it reflects the severity of the disease,^{3,4} (3) as a predictor for levodopa-induced dyskinesias,⁵ (4) for differential diagnosis of PD from dopa-responsive dystonia^{6,7} or drug-induced parkinsonism⁸ and (5) to differentiate dystonic or psychogenic tremors from tremors due to PD.⁹ Additionally, false negativity or

positivity, along with the advent of the novel terminology, scan without evidence of dopaminergic deficit (SWEDD) syndrome, is another compelling issue. DAT scan is the only non-invasive modality developed to assess functional integrity of the dopaminergic system currently used in clinical practice, and could give valuable clinical information for selected patients. We do think that, the approved indication of the DAT scan will be broadened to meet its versatile diagnostic potential.

Our patient exhibited definite parkinsonism after increasing the dose of the typical antipsychotic medication (haloperidol); it was therefore important to ensure whether parkinsonism resulted solely from haloperidol or if there actually was subclinical parkinsonism present and haloperidol triggered its manifestation. If the latter case was true, parkinsonism had to be considered as a symptom accompanying chorea and dystonia in the natural course of the disease, broadening the diagnostic spectrum. Although decreased DAT uptake was observed in our patient, the ‘pattern’ of decreased uptake was different from classic Parkinsonian syndromes. Moreover, we already obtained the data from magnetic resonance imaging (MRI) of the brain in

advance to co-localise and compare with the DAT scan data; therefore, we were not threatened by the caveats of false-positivity.

In conclusion, we believe that in atypical, complex cases that warrant prompt diagnosis and treatment, it does not harm the patient to undergo relevant tests, if not disorienting. Our patient was in desperate need of a concrete diagnosis in order to terminate his diagnostic odyssey and the DAT scan played a crucial role in our workup.

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