The 'parkinsonian heart': a diagnostic and prognostic tool

África Muxí[1,2,3]

In their *JNNP* paper Tsujikawa *et al* characterise Parkinson disease (PD) at the early stages in 70 patients, according to ^{123}I-meta-iodobenzylguanidine (mIBG) myocardial scintigraphic, and report its chronological changes (mean follow-up 3 years) with its symptoms relationship.[1]

123I-mIBG is a Norepinefrine (NE) analogue that competes with NE for the same transporter mechanism of postganglionar adrenergic neurons. The uptake of mIBG into neurons is achieved mainly through the uptake-1 mechanism, a high affinity homeostatic system responsible for the reuptake of NE. Receptors for NE or mIBG are located on the synapse in sympathetic terminal nerve axons; once in the axon, the tracer is transported in the axoplasm to NE storage vesicles. Unlike NE, mIBG is not metabolised, being retained in sympathetic vesicles, allowing it to be imaged. Therefore, myocardial uptake reflects the extent of postganglionic sympathetic cardiac innervation and allows insight into pathophysiology[2] of PD.

In the nineties of the previous century, it was observed that patients with PD disclosed a significantly lower myocardial mIBG uptake than healthy controls, due to a sympathetic myocardial degeneration related to a Lewy body type degeneration of the cardiac plexus. Since then, a lot of articles have been written about the 'parkinsonian heart', and the real diagnostic and prognostic value of this molecular imaging (mIBG scintigraphy).[3]

In this context, the article has clarified two important aspects:

First, chronological change patterns of the mIBG uptake, based on progression of cardiac sympathetic denervation, are heterogeneous among patients. And secondly, mIBG scintigraphy could be an early prognostic marker in PD, because a normal or mildly low mIBG uptake at the early stages of illness was characterised by slow progression in motor dysfunctions and preserved cognitive function at follow-up.

However, the authors explain as limitations that there are still a lot of aspects to investigate, clarify or confirm. The study design was retrospective, with no pathological diagnosis confirmation; the patients' characterisations were made only according to clinical and neuroimaging data and, therefore, the study's conclusions could be biased.

An urgent need exists to develop early diagnostic biomarkers of PD to intervene at the onset of the disease[4] and to monitor the progress of therapeutic interventions. A large prospective study is required, including pathological investigation and genetic analysis, in order to clarify the mechanism of chronological change of mIBG uptake in PD. It is necessary to establish normal uptake values and to confirm the real relationship between clinical data, prognosis, the anatomical distribution of Lewy bodies and the scintigraphic evolution, along the way. Molecular Imaging is a good way to improve the understanding of fundamental molecular pathways inside organisms in a noninvasive manner, but this has to be demonstrated from universal standard-of-care images. A new field is being developed, 'Radiomics', which refers to the extraction and analysis of large amounts of advanced quantitative imaging features. In neuroimaging, we have a long way to go. Einstein said "Imagination is more important than knowledge". We could add that now, in the 21st century, molecular imaging is able to change what we could only imagine into biological knowledge.

[1]Nuclear Medicine Department, Hospital Clínic Barcelona, Barcelona, Spain; [2]Institut d'Investigacions Biomèdiques August Pí i Sunyer (IDIBAPS), Barcelona, Spain; [3]Universitat de Barcelona, Barcelona, Spain

Correspondence to Dr África Muxí, Nuclear Medicine Department, Hospital Clínic, Villarroel 170, Barcelona 08036, Spain; amuxi@clinic.ub.es

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