Filling in the missing puzzle piece between cardiac MIBG scintigraphy findings and Parkinson’s disease pathology

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123I-meta-iodobenzylguanidine (MIBG) cardiac scintigraphy can be used to assess the pathophysiology of postganglionic presynaptic cardiac sympathetic nerve endings and is the most frequently used imaging agent to assess cardiac sympathetic innervation. In 1994, Hakusui et al were the first to report reduced cardiac MIBG and preserved thallium accumulation in Parkinson’s disease (PD). Subsequently, many studies confirmed the usefulness of 123I-MIBG cardiac scintigraphy for investigating early sympathetic involvement in PD and for differentiating PD from neurodegenerative parkinsonism including multiple system atrophy (MSA), progressive supranuclear palsy, and corticobasal degeneration together with vascular parkinsonism and essential tremor.

With respect to the pathophysiology involved in reduced MIBG uptake in PD, reduced MIBG uptake may be associated with the presence of denervation supersensitivity within the heart, resulting in hyperdynamic cardiac contractility in response to a β1-stress condition.2 Patients with reduced MIBG uptake showed reduced cardiac contractility during exercise, suggesting that this response represents impaired exercise capacity due to cardiac sympathetic denervation in patients with PD.3 Pathological studies demonstrated that cardiac sympathetic nerve degeneration occurred in PD and in cases of incidental Lewy body disease (LBD).4 However, no postmortem studies have directly investigated the relationship between antemortem 123I-MIBG scintigraphy results and cardiac sympathetic denervation findings in cases of LBD.

In their JNPN paper, Takahashi et al5 clearly showed a tight quantitative correlation between cardiac 123I-MIBG uptake and corresponding loss of sympathetic axon loss in the cardiac tissue samples of 23 patients with autopsy-confirmed LBD who underwent 123I-MIBG cardiac scintigraphy in life. Although Orimo’s group provided important evidence for 123I-MIBG cardiac scintigraphy in PD,4 this result further provides a scientific basis to confirm the reliability of MIBG cardiac scintigraphy as a powerful clinical tool to detect loss of these axons as a biomarker for the presence of LBD.

Since this was a multisite retrospective study of MIBG cardiac scintigraphy findings and autopsy samples of patients with LBD, differences in heart-to-mediastinum (H/M) ratios among facilities must be addressed. Recently, the cross-calibration phantom method made it possible to convert institutional H/M ratios to standard H/M ratios comparable to the most common medium energy collimator in Japan.6 The authors standardised the H/M ratios for each subject using the established calculated conversion coefficient. Such methodological progress contributed to the analysis of multicentre results in this study.

As the authors stated, there are several limitations to this study. First, the intervals from 123I-MIBG cardiac scintigraphy to death were relatively variable among patients. Second, information regarding concomitant diseases, which may influence the H/M ratios, is insufficient. Third, the control subjects only included one MSA and one patient with Alzheimer’s disease. Further prospective studies will be helpful in addressing these issues.

Overall, the study by Takahashi et al is an exciting article that confirmed the validity of 123I-MIBG cardiac scintigraphy as an excellent in vivo marker of degeneration of the cardiac sympathetic nerve in patients with LBD. Although careful clinical evaluation is still necessary, low cardiac MIBG uptake can certainly be a red flag for the presence of Lewy bodies in parkinsonian and demented patients.

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