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Transcranial sonography: a useful tool in patients affected by parkinsonism with normal dopaminergic functional imaging

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Introduction: Scans without evidence of Dopaminergic Deficit (SWEDD) represent 10% of patients affected by parkinsonism resembling Parkinson's Disease (PD) [1]. According to new PD diagnostic criteria normal imaging result is defined as absolute exclusion criterium but the significance of DAT still remain unclear [2]. Recent findings argue that striatal dopamine transporter imaging may reflect dopaminergic activity rather than number of surviving neurons or their striatal projection axons [3]. Hyperechogenicity of Substantia Nigra (SN), detected by Transcranial sonography (TCS) could represent a useful tool to identify these patients [4].

Objective: To evaluate the TCS in the patients affected by SWEDD-parkinsonism.

Methods: 3 patients with a clinical diagnosis of tremor-dominant parkinsonism and normal [123] IFP-CIT SPECT scans were recruited. TCS was performed with a 2.5 MHz transducer using a transtemporal window. Hyperechogenicity of SN was defined as an echogenic area above of 0.20 cm2.

Results: Patient 1: female; 55 years old; age at motor symptoms onset: 47 years; disease duration at SPECT: 8 years; disease duration at TCS: 8 years; SN hyperechogenicity (right: 0,41 cm2, left 0,38 cm2);
Patient 2: female; 76 years old; age at motor symptoms: 74 years; disease duration at SPECT: 1 years; disease duration at TCS: 2 years; SN hyperechogenicity (right:0,46 cm2, left:0,33 cm2);
Patient 3: male; 70 years old; age at motor symptoms: 53 years; disease duration at SPECT: 14 years; disease duration at TCS: 17 years; SN hyperechogenicity (right: 0,28 cm2, left 0,22 cm2);
All patient respond to dopaminergic therapy.

Conclusions: Clinical diagnosis of SWEDD-parkinsonism with normal dopaminergic functional imaging is challenging and still debated, and maybe alternative diagnosis could be considered. TCS, which detects SN hyperechogenicity found in up to 90% of patients with PD, could be a useful tool in identify these patients, when presynaptic dopaminergic nerve terminals are still preserved.

References:

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