Neuroanatomical correlates of trait Anhedonia in patients with Parkinson's disease

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1.0 Introduction

Anhedonia is the inability to experience pleasure from normally pleasant stimuli, commonly assessed within the context of psychiatric and neurological disorders [1]. The aim of this study is to investigate the association between trait Anhedonia and brain anatomy in Parkinson's disease (PD) patients, using Voxel-Based Morphometry (VBM). Indeed, we are interested at evaluating whether Anhedonia is associated with a specific neuroanatomical correlate, which might be help clinicians to better recognize the presence of this psychiatric symptom.

2.0 Materials & Methods

Patients meeting UK Brain Bank criteria for the diagnosis of idiopathic PD were recruited from the Neurology Unit of the University "Magna Graecia" of Catanzaro. Inclusion criteria were: (1) no evidence of clinical symptoms affecting brain anatomy, such as hallucinations, pathological gambling, levodopa-induced dyskinesias and dementia; (2) negative MRI exams and (3) a minimum 24-month duration of levodopa treatment. 25 PD patients were included in this study. **Anhedonia was evaluated using Snaith-Hamilton Pleasure Scale (SHAPS)** [2]. All patients underwent complete neuropsychological assessment focused on frontal functions.

3D T1-weighted morphological images were acquired using 3T scanner. VBM was performed to assess differences in gray matter volume between patients with or without Anhedonia. Significance threshold was set at FEW < 0.05 within specific ROIs strongly involved in either depression or in Anhedonia (Motor Cortex, Midbrain, Cingulate Cortex, Orbitofrontal Cortex and Amygdala).

3.0 Results

3.1 Clinical Findings

Table 1 showed demographical, clinical and neuropsychological comparisons between 11 PD patients with and 13 PD without Anhedonia. As previously demonstrated, our PD patients with Anhedonia were obviously characterized by higher level of depression and anxiety with respect to other PD patients. Despite similar demographical and clinical conditions, anhedonic patients showed also lower performance in verbal fluency and working memory.

The higher the SHAPS scores were found, the higher was the presence of depression. Otherwise, SHAPS scores did not correlated with clinical factors (i.e. UPDRS) or Neuropsychological performance.

Table 1. Clinical and Neuropsychological data

Clinical Data			
Variables	**PD with Anhedonia**	**PD without Anhedonia**	***p* values**
N°	11	14	
Gender (f/m)	5/6	6/8	0.68
Educational Level (y)	8 (1-13)	8 (4-13)	0.49[§]
Age (years)	60.81 ± 7.85	62.30 ± 7.77	0.63
Disease duration (years)	3 (1-7)	5.50 (1-13)	0.10
Hoehn-Yahr Stage	2 (1– 3)	2 (1 –3)	0.15
UPDRS-ME Off	43.90± 16.11	33.61± 14.12	0.10
UPDRS-ME	27.00 ± 10.22	22.61± 9.18	0.28
Psychological Data			
SHAPS	5.36 ± 2.94	0.69 ± 0.75	0.00
BDI	28.00 ± 15.63	10.38 ± 5.72	0.001
STAI-X1	57.45 ± 7.77	42.61 ± 9.65	0.000
STAI-X2	59.00 ± 10.75	39.61 ± 6.92	0.000
NeuroPsychological Data			
MMSE	25.73 ± 2.91	26.21 ± 2.99	0.69
Token Test	29.97 ± 2.27	30.38 ± 2.03	0.64
RAVLT Immediate recall	31.66 ± 8.14	38.76 ± 7.18	0.03
RAVLT Delayed recall	6.50 ± 1.77	7.30 ± 2.27	0.3
Verbal Fluency	21.67 ± 5.03	29.08± 7.66	0.01
Digit Span Forward	5.07 ± 0.71	5.21 ± 0.66	0.63
JLO	19.26 ± 3.45	20.03 ± 4.13	0.63
WEIGL	7.24 ± 2.33	8.89 ± 3.00	0.15
FAB	12.58 ± 3.14	14.56 ± 2.53	0.10

PD: Parkinson's disease; UPDRS-ME: Unified Parkinson's Disease Rating Scale-Motor Examination; SHAPS: Snaith-Hamilton Pleasure Scale; BDI: Beck Depression Inventory; STAI (X1-X2): State-Trait Anxiety Disorder; MMSE: Mini-Mental State Examination; RAVLT IR and RAVLT DR: Rey Auditory-Verbal Learning Test Immediate and Delayed Recall; JLO: Judgment of Lines Orientation; FAB: Frontal Assessment Battery.

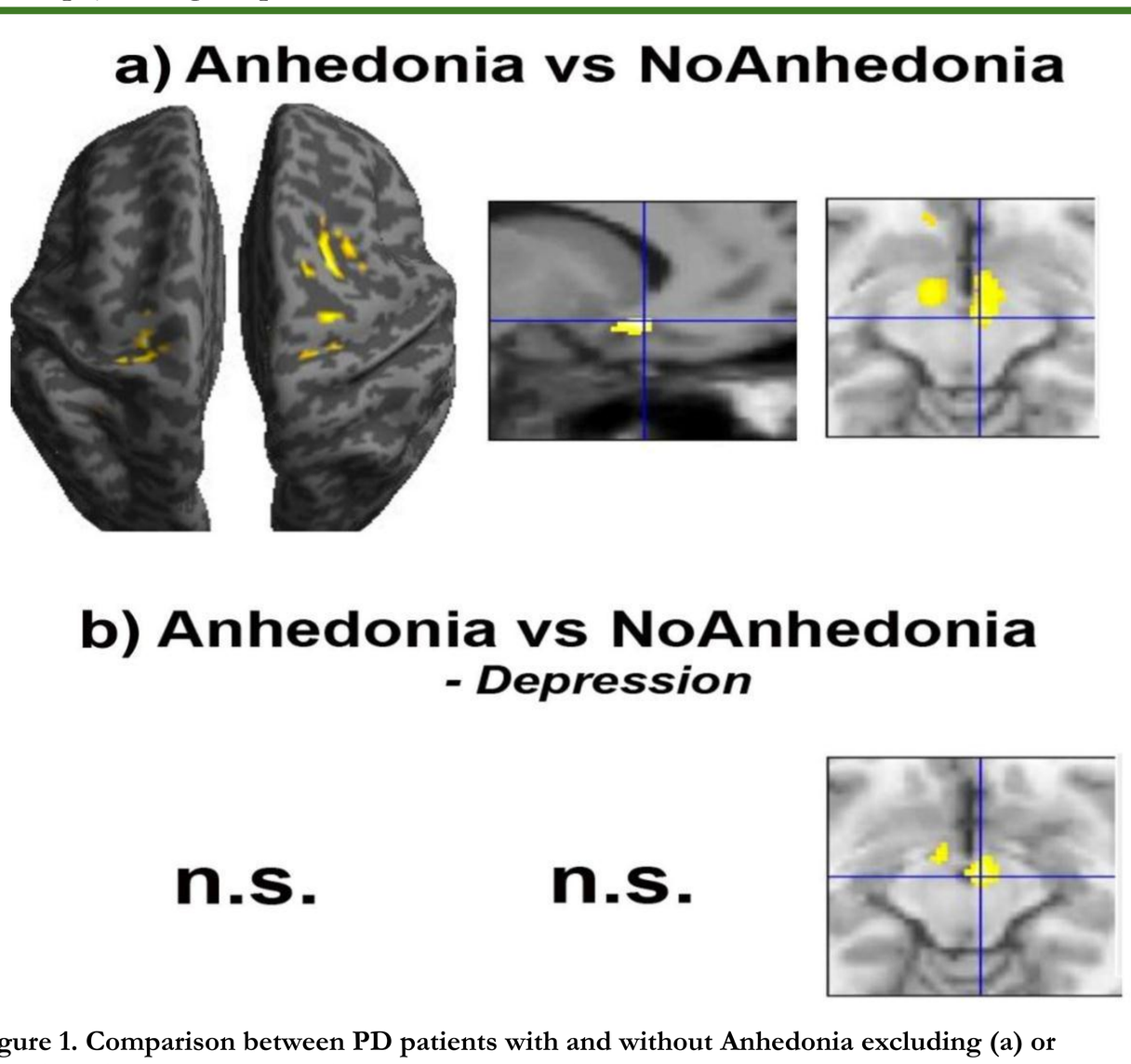

Figure 1. Comparison between PD patients with and without Anhedonia excluding (a) or including depression scores (b) as nuisance variable.

3.2 *Neuroimaging Findings*

PD patients with Anhedonia were characterized by significant gray matter losses in the motor cortex, as well as, in the subgenual cingulate cortex and in the ventral tegmental area (VTA) (Fig.1.a).

All these regions have been demonstrated to be strongly associated to depression status either in psychiatric or neurological realms. For this reason, to disentangle the impact of the Anhedonia with respect depression, we repeated t-test analysis using BDI scores as nuisance variables. No significant differences were detected, except for the gray matter volume loss in the VTA (Fig.1.b).

4.0 Conclusions

Anhedonia trait is strictly related to depression either in PD or in other psychiatric disorders. In fact, Anhedonic depressed PD patients were characterized by neural abnormalities in depression-related neural networks. However, when we tried to remove the impact of depression scores (using a statistical strategy) from VBM maps in order to isolate the role of Anhedonia, only neuroanatomical abnormality within the VTA persisted. VTA is one of the dopamine neuron cell bodies that constitutes the mesolimbic reward system, and dopamine release by the VTA is known to be crucial for reward processing. The detected neural pattern might be considered as a reliable biomarker of Anhedonia in PD patients.

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