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Emotions & Social Cognition

"Need to know" and the right temporal lobe: Impaired access to semantic knowledge in acquired obsessive-compulsive disorder?

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Introduction: Idiopathic obsessive-compulsive disorder (I-OCD) has been linked to abnormalities in corticostriatal circuits. Few studies have examined if the same structures are also responsible of acquired OCD (A-OCD) or if damage to anatomically-connected brain regions (e.g., temporal lobes) are also implicated in its pathogenesis. Additionally, there are some discrete obsessive-compulsive (OC) symptoms that by virtue of their presumed low occurrence and difficultly of categorization have received less attention. Amongst these, one intriguing and potentially severe type of obsessive thinking is the so-called "need to know" (NtK), a strong drive to know and obtain given information. In some patients this specific symptom, presumably resulting from impaired access to conceptual knowledge for specific verbal information (proper names, names of places), may be the principal or major feature of OCD symptomatology. We here report the cases of two male patients who developed "NtK" as the only OC symptomatology in association with malignant neoplasms involving the right temporal lobe and connected corticostriatal circuits.

Methods: We used Tractotron and Disconnectome map softwares in order to identify the regions of white matter damage overlap across both patients and the proportion of damage (lesion load) of each tract of interest for each patient. We quantified the severity of the disconnection by measuring the proportion of each tract of interest to be affected by each patient′s lesion by using Tractotron software. Additionally, Positron Emission Tomography was used in order to study metabolic abnormalities. The tracts of interest were: the uncinate fasciculus, the anterior commissure, the anterior thalamic radiations, the inferior fronto-occipital fasciculus and the inferior longitudinal fasciculus.

Results: There was a high overlap across brain lesions in patients 1 and 2. There was also a high overlap between areas that were affected (disconnected) due to the lesion. As expected, all the a priori selected pathways in the right hemisphere were affected since they cross the anterior part of the temporal lobe. Disconnection maps and metabolic changes in our patients suggest that the expression of OC symptoms underpinned by a semantic deficit due to right temporal damage is secondary to involvement of the uncinate fasciculus linking the temporal pole with the orbitofrontal cortex.

Discussion: Data from the present study concur with previous research on A-OCD and current findings in I-OCD which suggest that the temporal lobes participate in the phenomenological expression of OCD. Also, patients with lesions in the anterior temporal lobe are prone to show a specific "Need to Know" symptoms phenomenologically similar to patients with semantic dementia in later stages. The expression of OC symptoms underpinned by a semantic deficit because of anterior right temporal lobe lesion, are due to a disconnection of the uncinate fasciculus and the orbitofrontal cortex. Further research about the neurological underpinnings of specific OCD subtypes, its evaluation and treatment, are essential.

References: Berthier ML et al. Neurology. (1996) 47: 353–61.
Huey E et al. J Neuropsych Clin Neurosci (2008). 20(4):390-408

Keywords: Emotions & Social Cognition; patients; single case study; adults; psychiatric; lesion mapping, behavioural.

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