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THE LOSS OF FAMILIARITY: A CASE STUDY OF THE COMORBIDITIES OF CAPGRAS AND FREGOLI

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ABSTRACT

This case study describes the coexistence of Capgras and Fregoli-type delusions in a 51-year-old woman. The patient reported that her children were kidnapped and replaced with doubles. She claims that several customers at her shop are her real children. Neuropsychological assessment revealed severe defects in social cognition and an increased number of perseveration responses in the self-directed signaling task. We propose that the latter finding is a measure of the familiarity phenomenon and may be associated with hypofunction in the left retrosplenial region. SPECT-CT confirmed a pattern of hypoperfusion in the retrosplenial, posterior cingulate, and prefrontal cortex.

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word; capgras syndrome; fregoli syndrome; familiarity; cognition

Introduction

Delusional misidentification syndromes (DMS) are neuropsychiatric constructs defined by irrational beliefs about the identity of people, places, objects, or events, frequently involving the delusional duplication of these beings and entities. This belief persists over time on the same stimuli (Pietrini et al., 2009). About 40% of patients with DMS have identifiable brain damage resulting from vascular lesions, neoplasm, epilepsy, major neurocognitive disorders, or autoimmune disease (Ardila, 2019; Hall et al., 2010; Lozano-Cuervo et al., 2020). Considering the etiology, these patients usually present cognitive impairment in several cognitive domains such as orientation, anterograde memory, visuospatial disturbances and executive functions (Feinberg & Roane, 2017; Roane et al., 2019).

The spectrum of delusional misidentification has been conceptualized in different forms. The best known misidentification phenomenon is Capgras syndrome, in which the patient states that his or her relatives are in fact impostors. This is not prosopagnosia, as the patient is able to recognize that the “impostor” is physically identical to the relative. This has been conceptualized as a “delusional hypoidentification” or “underpersonalized misidentification” (Feinberg & Roane, 2017). Another syndrome that has been consistently reported is Fregoli syndrome. In this case the patient states that strangers are in fact well known persons: for instance, a relative is taking the appearance of a stranger. The patient states that the stranger is physically different to the relative, but nonetheless ascribes the identity of the relative to the stranger. This has been conceptualized as a “delusional hyperidentification” or “overpersonalized misidentification” (Feinberg & Roane, 2017). In some specific, infrequent cases, a single patient may display both the Capgras and the Fregoli type delusions (Lozano-Cuervo et al., 2020; Mashayekhi & Ghayoumi, 2016; Yalin et al., 2008); or the coexistence of other

DMS, as may be the case of intermetamorphosis and the subjective-double delusion (Paillère-Martinot et al., 1994), or even Reduplicative Paramnesia, Intermetamorphosis, Reverse-Intermetamorphosis, Misidentification of Reflection and Capgras Syndrome (Arsoy et al., 2014). Out of these reported cases, two were related to structural damage in the central nervous system. Paillère-Martinot’s et al. study (1994) showed evidence of structural damage with calcifications in the left lenticular region of the brain and impairment in a visuospatial integration and non-verbal memory test, and Lozano-Cuervo (2020) reported a case of anti-NMDAR encephalitis with right frontal lobe hypermetabolism and bilateral occipital hypometabolism. For the remaining cases of coexistence of DMS, no abnormalities were reported in the neurological examination or laboratory tests, suggesting a pathophysiology related to the minor neurological abnormalities frequently categorized as part of the schizophrenia spectrum. In this study, we report the case of a 51-year-old woman with Capgras delusion for close relatives and inanimate objects, in comorbidity with Fregoli syndrome. The research reported here was conducted in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans. After informed consent, the patient was included in an institutional review board approved protocol.

Method

Case presentation

Miss G. is a 51 woman with 12 years of formal education, divorced, who is currently working as an employee at a grocery store. She has a previous diagnosis of beta-thalassemia and hemolytic anemia, as well as a splenectomy. The patient is unaware of any familiar history of neurological or psychiatric illness.

Family members were unavailable for further interviewing, and their involvement in the patient's treatment has been lacking. The Institutional clinical records show no familiar history of relevant neurological or psychiatric illness.

Her psychiatric symptoms appeared in 2005 at age 36, starting with the belief of her partner being unfaithful. She claims that he had implanted her with an electronic device, in order to listen to everything she said. Clinical records show that treatment with Mirtazapine 15 mg/d was initiated, but showed no improvement. Subsequently, in the same year she presented an episode of disorganized thinking and behavior, for which she was admitted to a hospital unit for 26 days. She was diagnosed with a depressive schizoaffective disorder and was treated with Risperidone 2 mg 6 mg/d and Mirtazapine 15 mg/d, showing partial remission. The patient attended her outpatient-care consultations regularly and an adequate adherence to treatment was logged into her medical record. At age 46, she developed a tenacious belief of misidentification and reduplication which is consistent with Capgras syndrome: the patient reported that her children had been kidnapped and that the persons living with her are in fact their doubles. She stated that the impostors are also kidnaped victims, who had their memories erased and a chip implanted, which is why she tries to get along with them. Also, she developed a persistent belief which is consistent with Fregoli syndrome: she believes that the kidnapers also implanted her with a chip, through which they can alter her perception of reality and, in turn, make her real children present themselves looking like strangers. She claims that several customers at her shop are in fact her children, who ask a series of questions disguised *with other words* as a way of testing her. Miss G must answer in a specific order to these questions to recover her family. Due to the undercover nature of the questions, she never manages to answer them correctly. Other side effects of the chip include the insertion of thoughts, such as ideas to cook certain dishes and other commands, as well as the monitoring of her activities.

Miss G has also expressed ideas which are consistent with a Capgras delusion for inanimate objects: persistently, she states that vegetables and other prepackaged food from supermarkets had been replaced by the kidnapers with products that look similar but that have been adulterated with high fat content, which is why G spends long periods of time at the store comparing products in order to identify subtle differences that allow her to differentiate the real products from the modified ones. The patient explains that the modification in the food was carried out in order to control her diet so that the device works correctly.

G also reports complex visual and auditory hallucinations, which she describes as the transmission of her real children's torture and activities, which she receives through the implanted chip. She spends a couple of hours every afternoon walking around her neighborhood trying to find her children. Despite these symptoms, the patient remains functional: taking care of herself, keeping her job, taking care of her finances, scheduling her medical appointments, and taking her medication. She maintains a cordial relationship with coworkers and boss, although she admits that her interpersonal relationships are rather limited.

Treatment with Sulpiride 200 mg 0-0-3/4 and fluoxetine 20 mg 1-0-0 was started. The results of bloodwork performed on 02/02/20 are the following: leucocytes, $11.4 \times 10^3/\text{ul}$;

neutrophils, $5.04 \times 10^3/\text{ul}$; erythrocytes, 4.69 M/ul; hemoglobin, 4.7 g/dl; glucose, 88.9 mg/dl; triglycerides, 64 mg/dl; cholesterol, 183.9 mg/dl; prolactin 167, ng/ml. Hyperprolactinemia is a common finding in patients undergoing chronic treatment with high potency first generation antipsychotics, and with some of the commonly used second generation antipsychotics (Ajmal et al., 2014). In this case, we chose to initiate a medication switch to a prolactin sparing antipsychotic (aripiprazole, 15 mg, 1/2- 0-0), which was delayed due to Miss G's economic restraints. There were no anomalies on physical and neurological examination, and neurologic signs or symptoms associated to a pituitary adenoma were absent (heteronymous hemianopsia, headache, or physical changes related to hyperprolactinemia). There were no relevant, abnormal findings in structural Magnetic Resonance Imaging. There was no significant frontal or medial temporal lobe atrophy, or global cortical atrophy. Small, punctate non-confluent hyperintense images were observed in frontal white matter (with a Fazekas 1 score). Mega cisterna magna as an anatomical variant was also observed.

Cognitive assessment

Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS) and Consensus Cognitive Battery (MCCB) were used (Central and South America version). This tool is constructed as an initiative of the National Institute of Mental Health, with the aim of homogenizing a measurement instrument that provides a valid evaluation of the relevant cognitive domains in the diagnosis of patients with psychosis in the schizophrenia spectrum. This neuropsychological battery assesses seven cognitive domains: processing speed, attention/vigilance, working memory (WM), verbal learning, visual learning, reasoning/problem solving and social cognition. It consists of 10 tests: 1) Continuous Performance Test, 2) Hopkins Verbal Learning Test, 3) Brief Visuospatial Memory Test, 4) Symbol Coding, Brief Assessment for Cognition in Schizophrenia: 5) Semantic Verbal fluency test with animals, 6) Trail Making Test: A, 7) Wechsler Memory Test, Spatial Span 8) Letter-Number Span, 9) Neuropsychological Assessment Battery: Mazes; 10) Salovey-Caruso Emotional Intelligence Test: Managing Emotions, MSCEIT (Kern et al., 2004).

The executive functions and frontal lobules battery (BANFE-2) was also used (Flores-Lázaro et al., 2014). This battery has been adapted and standardized for the Mexican population and is sensitive to school years/academic years. It is an instrument designed for the assessment of complex functions that depend on the frontal lobe of the brain. The first division evaluates processes related to orbitofrontal functions, using the following tests: Stroop Test, WCST, Mazes. The dorsolateral division consists of self-directed signaling tests, visuospatial working memory, word alphabetical order, mazes, Tower of Hanoi, consecutive addition and subtraction, and verbal fluency. Finally, the anterior prefrontal cortex is evaluated using semantic classifications, selection of sayings and metamemory (Flores et al., 2014). Other tests were used, including the face recognition test, the face retrieval test, and Rey-Osterrieth complex figure from the NEUROPSI Attention and Memory battery. This neuropsychological instrument has been adapted and

standardized for the Mexican population and is sensitive to the scholarship and age (Ostrosky-Solís et al., 1999) and Vocabulary subscale of WAIS-IV for premorbid IQ (Wechsler, 2008).

Spect-CT

A Philips Precedence system with dual-detector was used. After the IV injection of a 925MBq dose of Tc99m ECD, under baseline conditions, multi-slice Computed Tomography for anatomical location and attenuation correction was used. The study is quantified using the NEUROGAM Segami Co. method.

Results

Neurocognitive assessment

The results obtained in the MATRICS consensus battery show a slight decline in the scores in the CPT attentional test and severe alterations in the social cognition domain. Scores within average parameters are observed in processing speed, verbal, and visual working memory, learning and reasoning. The scores for this battery are shown in Table 1. The evaluation of executive functions indicates that the consultant showed longer reaction times compared to her normative group, as observed in Stroop part A and Stroop part B, as well as Proverb Test. An unusual finding was observed in the increase of perseverative responses in the Self-directed signaling test (Table 2). More interestingly, this increase in perseverative responses was only observed in Self-directed signaling test and not as a generalized response in verbal, visual or motor tasks (Table 3). No evidence of impairment in face recognition, visuospatial or visuo-constructive skill tasks was obtained (Table 3).

Table 1. Cognitive performance in MATRICS Consensus Cognitive Battery.

	T- Score	Meaningful cognitive deficits
Processing speed overall	36	Average
Symbol Coding, BACS-SC,	35	
Verbal fluency test with animals	50	
Trail Making Test: A,	33	
Attention/vigilance. overall	33	Mild impairment
Continuous Performance Test	33	
Working memory overall	53	Average
Wechsler Memory Test: Spatial Span	50	
Span Letter-Number Span	56	
Verbal learning overall	44	Average
Hopkins Verbal Learning Test	44	
Visual learning, overall	69	Average
BVMT-R Brief Visuospatial Memory Test	69	
Reasoning/problem solving overall	46	Average
Mazes, NAB	46	
Social cognition. overall	11	Severe
Emotional Intelligence Test: Managing Emotions, Salovey-Caruso, MSCEIT	11	impairment

Brief Assessment for Cognition in Schizophrenia- BACS, Neuropsychological Assessment Battery NAB, Mayer-Salovey-Caruso Emotion Intelligence Test-MSCEIT. Definition of a meaningful cognitive deficit In terms of interpretation of meaningful differences between abilities in neuropsychiatric conditions, a widely accepted rule of for a clinically meaningful difference between two ability areas is about one -half of a standard deviation. The mild impairment is below to 35 T-scores

Brain imaging

As may be seen in Figure 1, a mild to moderate hypoperfusion in the bilateral prefrontal region, and in anterior parietal and temporal regions of the brain with left dominance, was observed in the SPECT study. This pattern of hypoperfusion extends to the mesial regions of the temporal lobe, and to the posterior cingulum, with a significant deficit in the left retrosplenial cortex. Some heterogeneous areas show mild hypoperfusion in the bilateral dorsal frontal region. When compared with the net cerebral

Table 2. Executive function assessment. Scores of Executive functions and frontal lobules battery- BANFE.

	Raw scores	Scalar Score	Meaningful cognitive deficits
ORBITOMEDIAL	201	108	Average
Mazes. Crossing	0	13	Average
Card game. Percentage of risk cards	19	14	Average
Card game. Total score	41	13	Average
Stroop form "A". Stroop errors	1	11	Average
Stroop form "A". Time	139	6	Mild impairment
Stroop form "A". Hit.	83	12	Average
Stroop form "B". Stroop type errors	0	12	Average
Stroop form "B". Time (encoded).	110	6	Mild impairment
Stroop form "B". Hit.	84	11	Average
Card sorting. Maintenance errors	0	13	Average
PREFRONTAL ANTERIOR	21	112	Average
Semantic classification Number of abstract categories	5	13	Average
Proverb Test time	218	6	Mild impairment
Proverb Test correct answers	4	13	Average
Metamemory Negative errors	1	13	Average
Metamemory. Positive errors	1	11	Average
DORSOLATERAL WORKING MEMORY	104		
Self-directed signaling. Perseverance answer	10	5	Mild impairment
Self-directed signaling. Time	104	9	Average
Self-directed signaling. Hitting	16	7	Average
Consecutive subtraction "A". 40-3. Time	64	10	Average
Consecutive subtraction "A". 40-3. Matches	12	11	Average
Consecutive subtraction "B". 100-7. Time	154	10	Average
Consecutive subtraction "B". 100-7. Matches	12	11	Average
Consecutive sum. Time	64	11	Average
Consecutive sum. Matches.	20	11	Average
Alphabetical order. Essay #1	1	12	Average
Alphabetical order. Essay #2	4	8	Average
Visospatial working memory. Maximum sequence.	4	15	Average
Visospatial working memory. Perseverations	0	10	Average
Visospatial working memory. Errors	1	11	Average
DORSOLATERAL EXECUTIVE FUNCTION + WN	104	115	Average
Mazes. Planning (no exit)	2	10	Average
Mazes. Time	53	10	Average
Card sorting. Matches.	52	15	
Card sorting. Perseverations	5	12	Average
Card sorting. Time	180	15	Average
Semantic classification. Total categories	5	7	Average
Semantic classification. Total score	14	16	Average
Verbal fluency. Matches	16	11	Average
Verbal fluency. Perseverations	0	13	Average
Tower of Hanoi 3 discs. Movements	14	10	Average
Tower of Hanoi 3 discs. Time	69	11	Average
Tower of Hanoi 4 discs. Movements	16	13	Average
Tower of Hanoi 4 discs. Time	65	13	Average
Total scores BANFE	440	115	Average

perfusion pressure and taking the cerebellar perfusion pressure as reference, the regions presenting greater decrease are located in the frontal areas 9, 10, 11 and 12; parietal area 39 and paralimbic cortices, mainly 23 (posterior cingulate cortex), 29 and 30 (retrosplenial cortex). CT Fusion scan and MRI show no signs of cortical atrophy or any other structural anomalies that could explain cognitive impairment.

Clinical outcome

After two years of follow-up, Miss G maintains with intense conviction the idea of the abduction of her children and the doppelgangers living in her home. She indicates that the messages received by the legitimate children (complex auditory and visual hallucinations) have ceased. The explanation for this phenomenon is that her daughter was released but not allowed to return home, while her son was *moved to another location*.

The patient shows no subjective complaints of cognitive impairment and there are no objective signs of cognitive decline. She even maintains adequate functionality and continues to self-care, maintains her job at the supermarket and manages her finances adequately.

Table 3. Assessment of visuospatial skills and face recognition and perseverative responses.

Test	Raw scores	Scalar Score	Meaningful cognitive deficits
Face recognition	4	13	Average
Face retrieval	6	13	Average
Rey-Osterrieth copie	36	14	Average
Rey-Osterrieth retrieval	33	14	Average
Vocabulary Test	38	10	Average

Test	Perseverative responses
Motor	
Card sorting -test	5
Tower of Hanoi 3 discs. Error type I and II	0
Tower of Hanoi 4 discs. Error type I and II	0
Verbal	
Alphabetical order. Essay #1	0
Alphabetical order. Essay #2	0
Alphabetical order. Essay #3	0
Semantic classification.	0
Verbal fluency	0
Metamemory	0
Visual	
Self-directed signaling.	10
Visospatial working memory Essay #1	0
Visospatial working memory Essay #2	0
Visospatial working memory Essay #3	0
Visospatial working memory Essay #4	0

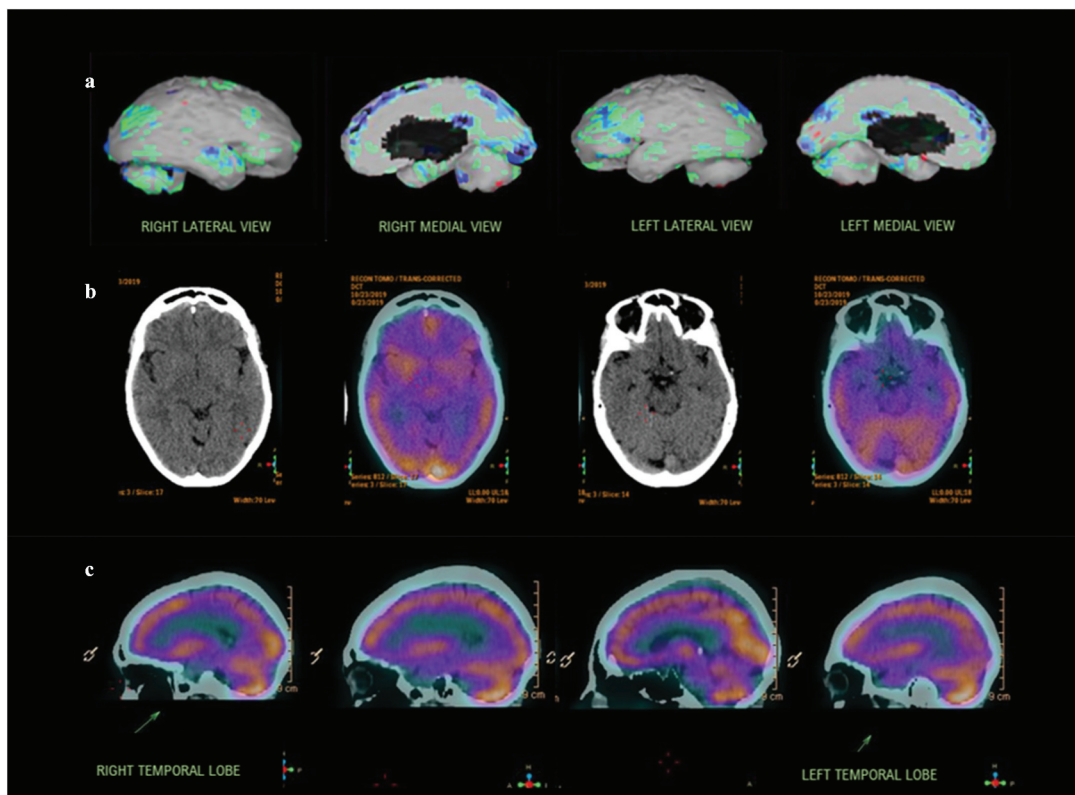


Figure 1. A) Regions with blue and green show cortical hypoperfusion (2 and 3 S.D.) in prefrontal, posterior cingulate cortex, and mesial regions; as well as left bilateral dorsolateral hyperperfusion, gray shows normal perfusion. B) A CT/SPECT fusion imaging in an axial projection showing areas of hypoperfusion in the frontal and mesial temporal regions, which are not explained by an increase in subarachnoid space or other signs of brain atrophy. C) A CT/SPECT fusion imaging in a sagittal projection showing a significant hypoperfusion in posterior cingulate cortex and retrosplenial cortex.

Discussion

The present case study describes a comorbidity of Capgras and Fregoli syndromes, which poses several questions at the psychopathologic and neurocognitive levels and which may contribute to the understanding of delusional misidentification.

It has been highlighted in previous studies that Capgras syndrome is often related to neurological conditions (Devinsky, 2009; Feinberg & Roane, 2005; Josephs, 2007; Lozano-Cuervo et al., 2020). This possibility was considered in the present case. Nevertheless, after obtaining a normal examination and a MRI without signs of neurological disease, we have no clinical evidence to diagnose a neurological disease. The possibility of a neurodegenerative disease was taken into account (Josephs, 2007). However, the patient had only mild cognitive dysfunction in the neuropsychological assessment, MRI and CT Fusion Scan showed no signs of cortical atrophy, and the psychotic syndrome started at age 36, without a significant decline of her functional ability at work at follow up. According to these facts, we considered the possibility of a primary psychotic disorder in the schizophrenia spectrum, which has been reported as the most frequent diagnosis in patients with Capgras syndrome (Bell et al., 2017). Our case is atypical from a psychopathological perspective, as it highlights the coexistence of more than one DMS in a single patient. This coexistence has been reported in previous cases, most of which have been reported in patients with primary psychiatric disorders (Arisoy et al., 2014; Mashayekhi & Ghayoumi, 2016; Yalin et al., 2008); however, patients with psychosis with a well-defined neurological basis have also been reported with a convergence of Capgras and Fregoli features (Lozano-Cuervo et al., 2020). The coexistence of both phenomena poses several questions to the current neuropsychological hypothesis of delusional misidentification syndromes, as are presented as explanations of the Capgras type delusions or the Fregoli type delusions as phenotypes with an opposite pathophysiology.

Regarding the neurocognitive perspective of the clinical problem, this case provides empirical data which may support a neuropsychological hypothesis to explain the formation of delusional misidentification. As reported by Paillère-Martinot et al. (1994), our case had discrete cognitive alterations which suggest that the misidentification and reduplication phenomena are not explained by generalized cognitive impairment.

The most influential cognitive theories regarding DMS suggest alterations in the dorsal visual area (Ellis & Young, 1990) or a disconnection between the fusiform area and the amygdala (Ramachandran, 1998). The neuropsychological assessment, as well as the structural and functional brain imaging studies obtained in the present case do not provide further support to those models. Instead, the present case poses a hypothesis related to the sense of familiarity and its relationship to cortical structures in the medial aspects of the brain hemispheres, with a particular emphasis in the posterior cingulate cortex and the retrosplenial cortex. The increased number of perseverative responses observed in the self-directed signaling task which is not present in any other motor, visual or verbal task in the G case (Table 3), offers us a clue regarding

the cognitive fault underlying misidentification, and an alternative to traditional neurocognitive testing in these cases, as follows:

The self-directed signaling task requires that the subject points to 24 figures on a sheet in an alternating manner, i.e. the subject must touch every element without pointing to the figures located immediately above, below or next to them. The task must be performed without omitting or repeating any figure; thus, the subject must develop an action strategy and keep in their WM (in a “supra-span” effect) the figures that they already pointed at, in order not to persevere (Flores et al., 2014). This task is related to the functioning of dorso-lateral prefrontal areas in their most ventral portion, which is part of the ventral-visual system for the maintenance of objects WM (Owen et al., 1996). Interestingly, Jackson and Raymond (2008) describe that the previously learned information (or familiarity) significantly increases performance in an WM task for faces, regardless of verbal memory and face analysis. The authors argue that even though familiarity and WM storage the information separately, they can share neural pathways. Taking this hypothesis into consideration, it would make sense that the damage to the sense of familiarity suggested in the DMSs may be related to lower performance in an visual WM task that was challenging enough, and that assessed the shared neural pathway, as we assume it occurs in this case.

The alterations reported in the G case, which include a possible decrease in the effect of familiarity process and the serious faults in the social cognition component reported in the MATRICS battery, could be explained by the model proposed by Darby et al. (2017), in the article entitled “Finding the imposter: brain connectivity of lesions causing delusional misidentifications”. In this paper, a pattern of functional connectivity is proposed for DMSs, including two main areas: left retrosplenial cortex, associated with the familiarity process, in which a disconnect pattern or hypofunction in this case (Figure 1) is specifically associated with the DMSs but not with other delusions; and right ventral-frontal cortex related to the violation of expectations and creation of delusions (Darby et al., 2017). This area has also been significantly associated with social cognition, emotional and decision-making processes (Hiser & Koenigs, 2018). In the present case, severe abnormalities were observed in the neuropsychological measures of social cognition, and a decreased perfusion in retrosplenial cortex was registered by means of SPECT-CT. A two-level hypothesis of delusion formation has been proposed (Davies et al., 2001), which suggests that the specific content of delusions may be related to a neuropsychological defect leading to an abnormal subjective experience. In this case, retrosplenial cortex hypoperfusion could be related to abnormalities in the sense of familiarity. A second factor is required to explain why the abnormalities in the sense of familiarity receive a delusional interpretation. In this case, this could be related to the severe defect in socio-emotional reasoning observed by means of neuropsychological assessment, and to the pattern of hypo-perfusion in retrosplenial cortex and prefrontal cortex observed with SPECT-CT.

Limitations

Beyond the intrinsic methodologic limitations of a case study, the lack of resources impeded us to perform a molecular test for C9ORF72, which has been linked to chronic psychosis in the context of slowly progressive frontotemporal dementia (Kertesz et al., 2013).

Conclusions

hypothesis related to DMS derived from studying the G case includes the cognitive evaluation of the phenomenon of familiarity, the violation of expectations, and their relationship with the disconnection patterns in the left retrosplenial cortex and right ventral-frontal cortex, respectively.

Disclosure statement

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