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ORIGINAL COMMUNICATION

The neuropsychological correlates of pathological lying: evidence from behavioral variant frontotemporal dementia

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Abstract To assess the neuropsychological bases of deception in a case of pathological lying. Pathological lying describes a clinical picture in which an individual repeatedly and apparently compulsively tells false stories. Developmental studies and neuroimaging studies suggested that executive functions and Theory of Mind are necessary for deception and that a dysfunctional prefrontal cortex may be involved in pathological lying. A patient presenting a pattern of behavioral alterations, including pathological lying, underwent a neurological, neuroradiological, neuropsychiatric, and neuropsychological examination. Psychopathological symptoms and cognitive deficits (executive functions and Theory of Mind) were suggestive of a behavioral variant frontotemporal dementia (bvFTD), while the lack of prefrontal hypometabolism was suggestive of a bvFTD phenocopy syndrome. This first observation of pathological lying as a symptom of bvFTD contributes to characterize its spectrum of psychopathological features. Moreover, this clinical case contributed to describe the possible neurocognitive deficits involved in the development of pathological lying. Further studies are needed to investigate how a prefrontal impairment affecting executive functions and Theory of Mind may cause a susceptibility to pathological lying.

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Introduction

Pathological lying (PL) describes a clinical picture in which an individual repeatedly and apparently compulsively tells false stories, and has been reported in psychiatric disorders as malingering, confabulation, factitious disorder, and personality disorders [1]. Empirical evidence on lying and deception, coming from different research perspectives, suggested that a dysfunction of the prefrontal cortex (PFC) may be involved in PL: children lie to conceal their transgressions and their ability to maintain these lies increases with age, in a manner strictly related to the development of Theory of Mind (ToM: the ability to understand and predict other people's behavior by attributing independent mental states to them) [2] and executive functions (EF) [3], both related to the PFC [4, 5]; moreover, pathological liars showed structural PFC alterations [6] and in healthy subjects deception recruits a subset of the PFC involved in ToM [7, 8]. PL is probably due to a PFC dysfunction but, however, its neurocognitive bases are unknown; we present the clinical case of a pathological liar that may provide preliminary cues about the neurocognitive bases of pathological lying.

Case report

A 57-year-old married and college-educated man underwent our attention in April 2010 because of a worsening pattern of altered behaviors. His relatives (wife and

daughter) reported that almost 3 years before he began to manifest personality changes that had progressively worsened over the past years. He actually presented a behavioral pattern characterized by apathy, verbal aggressiveness, impulsivity, occasional compulsive shopping, frequent lies, and lack of insight. Two years before, he was fired from his job as a truck driver because he was unable to manage and accomplish his assignments; since he was fired he attempted to do other jobs but he was always fired each time after brief periods because of similar difficulties to accomplish his assignments. His relatives reported that these altered behaviors were uncharacteristic of the patient personality at baseline; there was no history of head trauma, loss of consciousness, seizures, or previous contacts with mental health providers. He reported that he was involved in several activities that his relatives subsequently described as false: i.e., he reported to be an expert of software programming, an instructor of climbing, a radio-amateur, and to have played on a famous soccer team. His relatives reported that he usually lied to everybody (relatives, close friends, and strangers), or describing himself as involved in false activities or with false abilities or, for example, inviting other persons to false appointments (i.e., parties and dinners). They reported that when he was faced with the fact that his stories were totally false, he did not give any explanation and pretended nothing happened.

Neuroimaging detected a mild dilatation of the lateral ventriculi and of the periencephalic liquoral spaces (magnetic resonance imaging: MRI) and a normal brain glucose metabolism (fluorodeoxyglucose positron emission tomography: FDG-PET). Blood chemistry tests (blood cell count, liver and kidney function, electrolytes, thyroid hormones, B12 vitamin, folate, and VDRL) were carried to exclude other organic causes of this clinical picture. Neurological examination resulted negative. The neuropsychiatric assessment reported higher scores at the neuropsychiatric inventory [9] (30/144) and at the frontal behavioral inventory [10] (31/72). We adopted a previously reported methodology [11] to evaluate the presence of anosognosia in the patient: we used the frontal behavioral inventory to compare patient and caregiver concepts of symptom presence and severity, as previously reported [11]. The lower score of the frontal behavioral inventory administered to the patient (10/72) compared to the score obtained interviewing caregivers (31/72) suggested the presence of anosognosia. In the clinical interview, this decreased awareness also involved the behavior of telling lies, but in relation to this clinical symptom he showed especially an anosodiaphoria (i.e., lack of concern) [12].

At the neuropsychological examination, the patient appeared temporally and spatially oriented (Mini Mental State Examination 26/30) [13], with a fluent speech. He showed mild difficulties of episodic memory, while working memory, verbal fluency, planning, decision-making (Iowa Gambling Task) [14] and affective ToM (Reading the Mind in the Eyes) test [15] was markedly impaired. Neuropsychological performances of the patient are reported in Table 1.

Discussion

The pattern of behavioral alteration, as rated by higher scores in the neuropsychiatric inventory and in the frontal behavioral inventory test, was suggestive of a probable behavioral variant frontotemporal dementia (bvFTD), whose diagnostic clinical criteria [16] include an insidious onset and gradual progression, an early decline in social interpersonal conduct, an early impairment of regulation of personal conduct, an early emotional blunting, and an early loss of insight.

Also, the cognitive profile, characterized by a marked impairment of EF and affective ToM, was suggestive of bvFTD [17]. A hypometabolism of the PFC, especially in its orbital portions, is one of the earliest signs detected by neuroimaging in bvFTD patients [18]. Being that the PFC is involved in EF and ToM [4, 5], early deficits of executive functions [19] and of ToM [20–24] have been reported in patients with bvFTD.

Instead, the absence of cortical atrophy (MRI) and brain hypometabolism (PET) was suggestive of a non-neurodegenerative phenocopy of the bvFTD [25]. This clinical entity was suggested by a recent study [26] that, combining MRI and FDG-PET findings in a sample of clinically diagnosed bvFTD patients, found that the absence of brain atrophy in these patients was predictive of normal metabolism in frontotemporal regions, irrespective of disease duration. The lack of neurodegeneration in these patients strengthened the case for the existence of a non-neurodegenerative phenocopy of bvFTD [25, 26]. Interestingly, our patient, whose behavioral alterations were compatible with a diagnosis of bvFTD, presented neuropsychological deficits suggestive of a progressive neurodegenerative bvFTD but neuroimaging findings suggestive of a non-neurodegenerative bvFTD phenocopy. Considering the heterogeneous cognitive performance and that formal thought disorder, depressed mood, and mania were not evident at the formal psychiatric interview with the SCID-II [27], we excluded the presence of malingering or late-onset psychosis [28]: our clinical impression was that the diagnosis that best fit to the clinical picture of the patient was that of bvFTD. Resuming, this case of bvFTD, with some clinical features (neuropsychological deficits) suggestive of the progressive neurodegenerative subtype and other clinical features (neuroimaging) suggestive of the non-progressive non-neurodegenerative phenocopy syndrome, raises important questions about the

Table 1 Neuropsychological features of the patient

Cognitive function	Task	Score	Comment
Estimated premorbid IQ	Brief Intelligence Test [34]	98	Normal
Global cognitive status	Mini Mental State Examination [13]	26/30	Preserved
Episodic verbal memory	RAVLT immediate recall [35]	20.3	*Impaired*
Episodic verbal memory	RAVLT delayed recall [35]	3	*Impaired*
Naming	Boston Naming Test [36]	30/30	Preserved
Verbal fluency	Phonemic verbal fluency [37]	5.8	*Impaired*
Verbal fluency	Semantic verbal fluency [37]	4	*Impaired*
Visuospatial functions	Benton's Judgement of Line Orientation [38]	27/30	Preserved
Constructional praxis	Copy of the Rey Figure [39]	36/36	Preserved
Selective spatial attention	Barrage [39]	39/60	Preserved
Processing speed	Trail Making Test Part A [40]	35	Preserved
Set-shifting	Trail Making Test Part B [40]	160	Preserved
Abstract reasoning	Raven Colored Progressive Matrices [41]	26.6/36	Preserved
Inhibition	Stroop Interference Test [42]	21 s. 0 error	Preserved
Executive functioning	Frontal Assessment Battery [43]	12.5/18	*Impaired*
Planning	Tower of London [44]	9/36	*Impaired*
Cognitive estimation	Cognitive Estimation Test [45]	11	Preserved
Verbal working memory	Backward digit span [46]	2	*Impaired*
Decision-making	Iowa Gambling Task [14]	–6	*Impaired*
Affective Theory of Mind	Reading the Mind in the Eyes [15]	12/36	*Impaired*

Impaired performances are in italics

IQ intelligent quotient; *RAVLT* Rey auditory verbal learning task

sensitivity and the accuracy of current consensus criteria1 for bvFTD and advocates a role for neuroimaging in clinical diagnosis [16]; the addiction of neuroimaging criteria (i.e., structural and functional frontotemporal abnormalities) may improve the specificity of the diagnosis, that is still mainly based on the behavioral alterations.

The persistent phenomenon of telling lies, observed during the visit and reported by his relatives, appeared as continuous in the life of the patient independently by the context (familiar and unfamiliar); our clinical impression was that it appeared as a compulsive behavior, compatible with a diagnosis of PL [1]. In our patient this behavior appeared characterized as a compulsion to tell lies, maybe linked to an increased impulsivity, and not necessarily finalized to manipulate and deceive other subjects. For example, a computer program crashed during the neuropsychological assessment and the computer needed to be restarted: observing these "troubles" the patient offered his help, "on the basis of his long experience in the field of computers" (expertise that his relatives described as totally false): this episode was suggestive of how a banal event could trigger the possibility to tell a lie, which was impossible to inhibit.

Other characteristics of lying of the patient fit with our diagnosis of PL [1]: it is questionable whether pathological lying is always a conscious act and whether liars always have control over their lies; an external reason for lying (such as financial gain) often appears absent and the internal or psychological purpose for lying is often unclear; the lies are often unplanned and rather impulsive; the liar may become a prisoner of his lies; the liar may acknowledge, at least in part, the falseness of the tales when energetically challenged, and telling lies may often be seen to be an end in itself. In the clinical interview, the patient showed a decreased awareness for his behavioral alterations, as usually reported in bvFTD patients [29, 30]. As reported before, in regards to his behavior of telling lies, the patient showed a decreased awareness (i.e., anosognosia) and a lack of concern (anosodiaphoria), which probably contributed to the persistence of this clinical phenomenon.

To our knowledge, any study investigated the neurocognitive functioning of pathological liars; the neuropsychological assessment of our patient may help to preliminary shed light on the neurocognitive bases of PL. Neuropsychological deficits of the patient (affective ToM and executive functions) were suggestive of a dysfunction involving both the dorsolateral and the orbital/ventromedial portions of the PFC. The dorsolateral PFC provides a cognitive and controlled elaboration of information [4] and is involved in the cognitive component of ToM (inference on other people's beliefs) [31]; the orbital/ventromedial PFC provides an

emotional and automatic processing of information [5] and is involved in the affective component of ToM (inference on other people's feelings) [31]. Neuropsychological deficits were coherent with studies showing that in healthy children and healthy adults, ToM and EF are necessary for lying and deception [3, 7, 8]. In healthy subjects, ToM is involved in the generation or detection of lies [7], while EF are involved in the inhibition of true responses [8]; on the basis of these neuroimaging findings, it could be hypothesized that in pathological liars, an impaired ToM is involved in the abnormal generation of lies while impaired EF are involved in their poor inhibition.

Conclusions

In conclusion, we felt this case to be twofold interesting. First, to our knowledge, this is the first observation of pathological lying as a symptom of a neurodegenerative disorder affecting the PFC, as bvFTD; this observation contributes to characterize the spectrum of psychopathological features in bvFTD patients [32], confirming their unique predisposition to break social rules, up to criminal violations [33]. Second, this case preliminary suggests that an impairment of EF and of ToM may result in PL, describing its possible neurocognitive bases. Further studies with samples of pathological liars are needed to confirm that pathological lying is associated with a prefrontal impairment affecting EF and ToM and how a contemporary dysfunction of these mental processes may cause a susceptibility to PL.

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Conflict of interest None.

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