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Poststroke delusions: What about the neuroanatomical and neurofunctional basis?

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ABSTRACT

Delusion is a belief about yourself, people, or events that has no accordance with reality. Although it is known that stroke could cause various psychiatric and psychological effects, including depression, anxiety, and aggressiveness, psychotic symptoms, especially delusions, are rather uncommon. The most investigated poststroke delusions are paranoid type, nihilistic, and Fregoli syndrome. We will describe two patients showing delusion symptoms (Cotard-like and erotomanic ones) that occurred after a stroke involving the right temporal lobe, the basal ganglia and insular region, persisting for a long period after the stroke onset. We have, therefore, supposed that the simultaneous involvement of these brain areas could be involved in the neuroanatomical basis of delusions, as also demonstrated by the neurofunctional evaluation.

KEYWORDS

Basal ganglia; Cotard syndrome; erotomanic delusions; functional magnetic imaging; insula

Introduction

Delusion is a belief about yourself, people, or events that has no accordance with reality. Patients can believe different delusions: that someone wants to hurt them (paranoid delusion), to be a famous or historic person (grandiose delusion), to have a connection with God (religious delusion), to feel dead (nihilistic delusion), to mistake relatives and strangers (Capgras and Fregoli delusion), to believe that their partners betray them (jealousy delusion), to be seduced by someone (erotomanic delusion), and/or to suffer from a mortal disease or that their body has a deformity (somatic delusion). Generally, delusions occur in psychotic diseases, including schizophrenia. Actually, some other diseases could cause them, such as dementia (Cipriani, Danti, Vedovello, Nuti, & Lucetti, 2014; Torrisi et al., 2016), traumatic brain injury (Guerreiro, Navarro, Silva, Carvalho, & Gois, 2009), and stroke; moreover, delusions may be due to drug effects. Although it is known that stroke could cause various psychiatric and psychological effects, including depression, anxiety, and aggressiveness, psychotic symptoms, especially delusions, are rather uncommon. In a large case series including 360 patients affected by stroke, Kumral and Oztürk (2004) found delusions in only 15 patients. The most investigated poststroke delusions are the paranoid (Devine et al., 2014) and nihilistic types,

(Sottile et al., 2015), as well as those that characterize the Fregoli syndrome (de Pauw, Szulecka, & Poltock, 1987).

Herein, we will describe two patients showing delusions that occurred after a stroke involving the right temporal lobe, the basal ganglia, and the insular region. The patients and 5 age-sex matched normal controls (NC) underwent high field magnetic resonance imaging (MRI) examination, to evaluate whole brain atrophy, normalized cerebral measurements, and functional MRI resting state, by using tools that are a part of the FMRIB software library (FSL - <http://www.fmrib.ox.ac.uk/fsl>).

Case-report 1

A 49-year-old female, housewife, divorced with a son, smoker and affected by hypertension and thyroid dysfunction, without a clinical history of psychiatric diseases, was admitted in a neurology ward owing to an ischemic stroke. Around 12 hours after the thrombolysis treatment, she developed a hemorrhagic stroke in the right temporal lobe spreading to insula, caudate nucleus and internal capsule, (Figure 1A). At a neurological evaluation, she presented a left hemiplegia and moderate hemianesthesia, without loss of consciousness. The day after the hemorrhagic complication, the patient showed paranoid delusions mainly related to

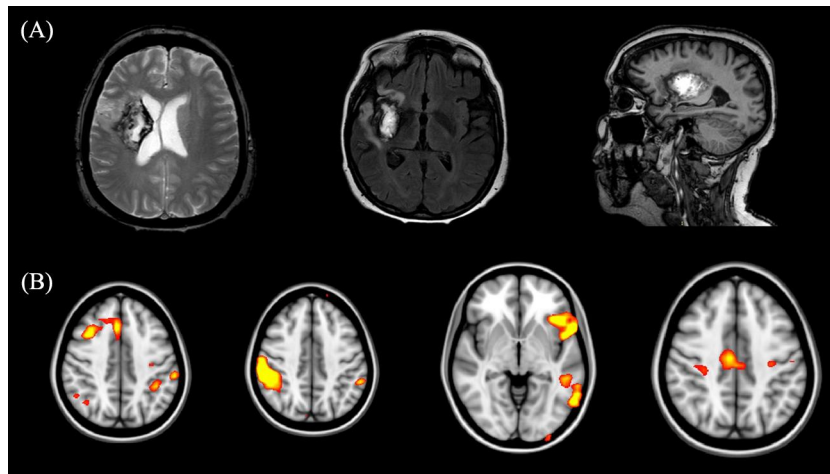


Figure 1. (A) Axial T2- weighted, Axial FLAIR and Sagittal T1-weighted, showed intraparenchymal hematoma at the level of right basal ganglia. Ischemic lesions in right cortical sub-cortical parietal lobe. (B) Functional magnetic resting state showed brain decreased activation in: Right Angular Gyrus, Right Middle Frontal Gyrus, Left Paracingulate Gyrus - Right Supramarginal Gyrus (posterior and anterior division), - Left Insular Cortex, Left Tempolar Lobe, Left middle temporal gyrus, - Precentral gyrus, postcentral gyrus, right cingulate gyrus.

the healthcare professionals, asserting that “nurses want to hurt or kill me” or “this hospital is false, it is all a sham, the patients are real but the staff is fake.” Each treatment carried out by nurses was considered an act of aggression or carelessness. Progressively, the frequency of delusions decreased. However, a nihilistic/Cotard-like delusion began to appear, referred to beliefs of death or loss of body’s parts; indeed, she often said: “I have died and reborn eight times,” “My feet were removed and replaced with others.” These delusions were associated with an intense distress. There were no hallucinations. The cognitive level, tested by verbal neuropsychological instruments because of the inability of using her hands (left hand plegia, and arterial thrombosis at the right thumb, second and third fingers) showed normal orientation, memory (Rey 15-item Memory Test Equivalent Scores = 4), attention (Digit Span Forward E.S. = 4), and language, whereas abstracted reasoning skills were abnormal (Raven’s Progressive Matrices E.S. = 0). After one month from stroke, she was administered risperidone (3 mg/die) and escitalopram (20 mg/die), with a rapid disappearance of delusions. The patient underwent MRI examination to evaluate normal brain volume - NBV (1466454 mm³) and normal cortical volume - NCV (748341 mm³). Resting state evaluation showed a decreased connectivity in the right angular gyrus, right middle frontal gyrus, left paracingulate gyrus, right supramarginal gyrus (posterior and anterior division), left insular cortex, left tempolar lobe, left middle temporal gyrus, precentral gyrus, postcentral gyrus, and right cingulate gyrus (Figure 1B).

Case report 2

A 55-year-old female, teacher, unmarried, smoker, and affected by diabetes and hypertension, without previous psychiatric symptoms, was admitted to the hospital following subarachnoid hemorrhage due to aneurysm rupture. She, therefore, underwent embolization of the left internal carotid and stent implant of the right internal carotid. Immediately after the procedure, she developed an ischemic damage to the caudate, the lenticular nuclei and right insular region (Figure 2A),

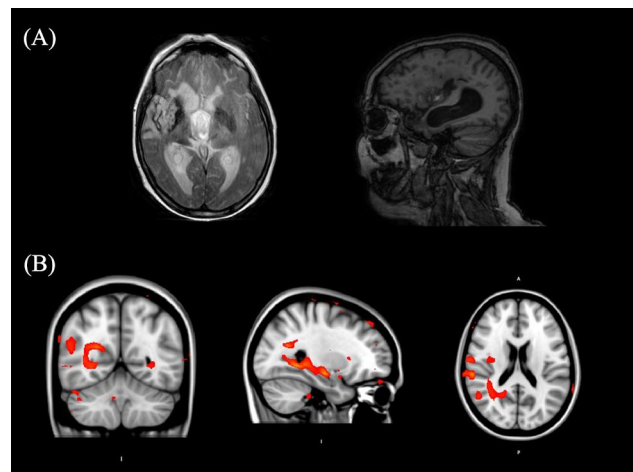


Figure 2. (A) Axial T2- weighted and Sagittal T1-weighted, showed right middle carotid artery intraparenchymal ischaemic lesion at the level of right basal ganglia. Ischemic lesions in right cortical sub-cortical fronto-temporal lobe. (B) Functional magnetic resting state showed brain decreased activation in: Supracalcarine cortex, Precuneus cortex, - Planum temporale, - Postcentral gyrus.

with psychiatric symptoms including erotomanic delusions. Specifically, the patient believed that some nurses had fallen in love with her and later had betrayed her with other patients. Moreover, her behavior presented disinhibited and histrionic behaviors. There were no hallucinations. At her neuropsychological evaluation, she presented a moderate to severe deterioration. Temporal orientation, memory, attention, and executive functions were impaired (Moca test: 20/30; Trial Making Test A equivalent score = 0, Trial Making Test B equivalent score = 0, Trial Making Test B-A equivalent score = 0, Rey 15 item Memory Test equivalent score = 1, Frontal Assessment Battery equivalent score = 0). The patient showed delusions during the whole duration of the hospitalization, but in consideration of the fact that she had no distress concerning the delusions, we decided to treat only the inappropriate behavior by administering oxcarbazepine (900 mg/die), with a complete remission of the symptomatology at three-month follow-up. The patient underwent MRI examination to evaluate NBV (1327365 mm³) and NCV (675664 mm³). Resting state evaluation showed a decreased connectivity in supracalcarine cortex, precuneus cortex, planum temporale, and postcentral gyrus.

Discussion

We have described the cases of two patients who, following a brain lesion, have developed delusions. The absence of pre-existing psychiatric symptoms, besides their onset after the event, let us argue that the cause of delusions was the cerebrovascular accident. Anyway, it is reasonable to suppose that specific personality traits had influenced delusion features, at least with regard to their contents. Both the patients were suffering from a subcortical lesion in the basal ganglia and right insular region, and these lesions have been investigated by a high field MRI, to better define localization and extent. Delusions are beliefs that remain fixed despite evidence that they are incorrect. Although the exact neural mechanism of delusional belief remains to be clarified, a predominance of right-hemisphere lesions has been reported in patients with organic delusional syndromes. Several mechanisms have been postulated to explain the development of psychotic symptoms following acquired brain lesions, including direct injury to the frontal lobe or disruption of the connections between the prefrontal cortices, associative areas, and subcortical structures causing impairment of reality monitoring functions (Gurin & Blum, 2017). Although there is no agreement on which

regions are correlated with these manifestations, as they occur after temporal, parietal (Sottile et al., 2015), frontal (Devine et al., 2014), and subcortical lesions, we supposed that the simultaneous involvement of the basal ganglia and insula could play a pivotal role in causing delusions. Several studies have suggested the role of insula in the genesis of psychiatric diseases (Crespo-Facorro et al., 2000) and in regulation of emotions (Gu, Hof, Friston, & Fan, 2013), but data on stroke as a possible etiology are scant. With this report, we have confirmed that delusions could be a consequence of stroke involving insular region, and it is likely that such symptoms may be misdiagnosed and underreported. Indeed, insula is believed to be involved in consciousness and play a role in diverse functions usually linked to emotion or the regulation of the body's homeostasis. These functions include compassion and empathy, perception, motor control, self-awareness, cognitive functioning, and interpersonal experience. In relation to these, it is involved in psychopathology and it is possible that lesions of different insular regions and/or its connections with the frontal lobe may cause delusions (Underwood, Kumari, & Peters, 2016).

Otherwise, there are some studies showing a likely influence of the lesions to the basal ganglia in the onset of psychotic symptoms, especially those involving the caudate nucleus (McMurtray, Sultzer, Monserratt, Yeo, & Mendez, 2008; Narumoto et al., 2005), corona radiata and lentiform nucleus (Abdullah, Saini, Sharip, & Rahman, 2015), and putamen (Kitabayashi et al., 2006). Interestingly, Huber, Karner, Kirchler, Lepping, and Freudenmann (2008) described cases of parasitosis delusion due to right striatal infarctions, confirming the importance of this region in inducing psychosis. The simultaneous presence of different hallucinations have also been reported, (McMurtray et al., 2014), and this could be due to a dysfunction of the connections between prefrontal and subcortical areas following a stroke.

In both our cases, the brain lesions involved the right hemisphere, and this is consistent with the majority of poststroke delusions, given that they are rarely caused by lesions of the left hemisphere (McMurtray et al., 2014). Moreover, the delusion time of manifestation in the two cases confirms the findings by other studies that delusions generally occur few days after the vascular event. A delayed onset of poststroke delusion is an uncommon event (Barboza, De Freitas, Tovar-Moll, & Fontenelle, 2013). Otherwise, although they generally last for a short period, in our case report 2 we observed the presence of delusions for a long

period, and we cannot affirm whether or not their disappearance could be attributable to the risperidone effect in case report one. However, we believe that to describe the eventual onset of poststroke delusions is of utmost importance, given that they have been associated with poorer prognosis and, as found by Almeida and Xiao (2007), and they increase death's probability after 10-years from the vascular event.

Moreover, our patients showed a decreased NCV, as compared to NC ($P < 0.1$). These results are consistent with previous voxel-based morphometry studies, which showed grey matter reduction in the medial frontal/anterior cingulate cortex and bilateral insula (Wolf et al., 2013). Finally, resting state functional MRI showed a decreased connectivity in bilateral insular regions, as reported by other studies (Vicens et al., 2016).

Although we are not completely able to exclude that the influence of insula and basal ganglia damage on the onset of delusions is a mere coincidence, the presence of the same damaged brain regions in the two cases, without a previous psychiatric symptomatology, is a good reason to hypothesize this association. The presence of the same damaged brain regions in the two cases also supports the idea that the neuroanatomical basis of delusions may encompass these complex and interconnected areas, beyond a neurobiological model of disrupted prefrontal control over somato-sensory representation.

Clinicians, including neuropsychologists, should be aware about/know the neuroanatomical basis of psychosis to timely diagnose and promptly manage stroke patient presenting with psychiatric symptoms.

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