

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900

Open access books available

186,000

International authors and editors

200M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



What is Capgras Syndrome? Diagnosis and Treatment Approach

Aslı Enzel Koc and Cicek Hocaoglu

Abstract

Capgras delusion is a complex psychopathological phenomenon that presents in a wide range of psychiatric and neurological disorders with differing patterns dependent on the main etiology. An underlying neurological disease should be suspected where the delusion concerns a spouse or inanimate objects and is associated with visual hallucinations, while a functional disorder is suggested by multiple imposters, strangers, additional delusions, and auditory hallucinations. Misidentifications in Capgras syndrome (CS) are fixed false beliefs and, therefore, represent true delusions. Even if when patients are confronted over and over with the illogical nature of the delusion, they keep their beliefs. Surprisingly, patients may show implicit or explicit awareness of their true situation. Some research suggests that a considerable number of patients with CS have some awareness of the bizarre nature of the misidentification delusions and therefore tend not to report them, especially during initial interviews when they are less likely to be confident with the clinician. Specific questions and interventions may assist clinicians in successfully identifying patients with CS. In a series of interviews with these patients, some focus on identifying CS, rather than a single interview which is likely to increase the detection of the delusional misidentification. The clinician should always be mindful of the risk of aggression and homicide in CS.

Keywords: Capgras syndrome, misidentification syndromes, psychotic disorder

1. Introduction

In delusional misidentification syndromes (DMSs), the individual everlastingly misidentifies persons, places, objects, or events. Capgras syndrome (CS) is the most common in the umbrella term DMS [1, 2]. Perhaps the best known form of DMS is the *Capgras syndrome*, originally described by Dr. Joseph Capgras and his colleague, J. Reboul-Lachaux, in the early twentieth century [3]. They first encounter this impressive phenomenon when their patient Madame M. insisted that all her friends, family, relatives, and neighbors were being replaced or constantly misperceived as being an imposter [4]. The term *l'illusion des sosies* (the illusion of doubles) was used to describe the case of a woman who strongly believes that various “doubles” had taken the place of people she knew [3]. It is an essential feature of the Capgras syndrome, the denial of identity of known persons and the delusional belief that this person has been substituted by a double [5].

CS is characterized by the delusional denial of identity of a significant other and the belief that they have been replaced by a double. Some patients with CS may deny the identity of the actual spouse and claim that there are two spouses, the actual and the imposter [6]. Therefore there are four conditions in patient with CS: the person is recognized, and the patient affirms the resemblance of the double to the misidentified significant other; no identity is attributed to the double, who has neither name nor existence; the double is an imposter, pretending to be the original they are replacing; the original has disappeared, his/her absence remaining unquestioned [7].

2. Features of Capgras syndrome: phenomenology, diagnosis, and epidemiology

The rareness of CS, as well as its impressive clinical manifestation as a colorful syndrome, has caused most publications to present case descriptions as scientific curiosities [8, 9]. CS has also attracted the attention of novelists in fictional literature. Dostoevsky provided a dramatic description of the phenomenon in his novel, *The Possessed* [6]. Sociocultural factors essentially shape the phenomena and thus mightily influence the establishment of definitions of this disorder [10]. Therefore, it may be necessary to mention. The meaning given to the terms 'change' and 'transformation' of physical identity has been called 'incarnations' or 'possessions' of other bodies in some cultures [10]. Possessions by an evil spirit have early origins within Paganism, Wicca, Haitian voodoo, Buddhism, Hinduism, Judaism, and Christianity [11]. There is a belief in some countries that people can be possessed by Satan and made to act in strange, immoral, and antisocial ways. In the United States, among European-American Catholics, there exists a belief that demons may possess a person. Possessing demons are presumed to cause experiences of proscribed feelings, thoughts, or behaviors in the person. Occasionally, solutions involve exorcism rituals [12].

It is generally being reported as single case studies in the literature. Although an uncommon psychiatric disorder, Capgras delusion has been central to the development of theories of delusions [6]. It is not dealt with particularly in the DSM-5 and may be classified as delusional disorder, suiting either the persecutory or the unspecified type [13]. With no consensual clinical criteria for this syndrome, it is usual to refer to their original description [7]. The basic manifestation was a false belief that real and familiar persons or oneself is replaced by strange, malicious imposters [14]. In fact, CS is a 'hypoidentification' of a person closely related to the patient [6]. CS is more frequent in women than men, with a sex ratio of approximately 2:1, but this result was not found across all studies [7]. Only a few reports have described this syndrome in patients during childhood [15].

The remarkable feature of Capgras delusion is that patients are able to recognize the close relation, the related person's face, but deny his or her identity and often use subtle misperceived differences in behaviour, personality, or physical appearance to distinguish between him or her and the imagined impersonator [16, 17]. Patients with CS find ways to defend their irrational beliefs [4]. Generally, the patients support their conviction in revealing detail. This sign may be a habit or a personality trait; small misperceived differences, for instance, in physical appearance and behaviour, may vary over time [7]. And these are frequently used to distinguish the imposter from the loved one [18]. Surprisingly, patients may show implicit or explicit awareness of their true situation [6]. Some research suggests that a considerable number of patients with CS have some awareness of the bizarre nature of the misidentification delusions and therefore tend not to report them, especially during initial interviews when they are less likely to be confident with the clinician [19].

Common to all DMS is the delusional denial of identity of objects having affective significance for the patient, and it is exceptional for there to be only one imposter, but these objects are limited in number. CS may be associated with other DMSs, and these frequently evolve from one another because of this relation and similarity [7, 20].

It sometimes occurs isolated, hereby justifying its autonomy as a 'delusion' [7]. CS may be accompanied by other delusions and thus may rarely exemplify a 'monothematic' delusion [6]. Erotomaniac delusions and delusional jealousy [i.e., Othello jealousy] were identified in 9.1% and 6.4% of patients with CS, respectively [21, 22]. However, delusional misidentification syndromes uncommonly appear independent of comorbid pathology [23].

The absence of consensual clinical criteria makes the epidemiological data uncertain [7]. Thus, the prevalence of CS may be underrated. More than half of the patients of the registered cases suffered from mental disorders without any organic association, among which schizophrenia spectrum disorders were diagnosed in 6 of 10 patients with CS [21, 22]. The Capgras delusion has been reported in association with other psychiatric disorders in 60–75% of cases and in organic illnesses in 25–40% of cases [23]. The Capgras delusion has usually been recognized in the contextual relationship of psychiatric disorders and often occurs in conjunction with paranoia, derealization, and depersonalization [6]. The Capgras syndrome may represent a delusional evolution of the phenomena of depersonalization and derealization [24]. Nonspecific, derealization-depersonalization experiences are frequent, especially in psychotic disorders, and are considered a significant core symptom of CS [7]. Studies on the prevalence of this disease or comorbid disease show differences. A study has found that the prevalence of DMS in psychiatric populations was less than 1% [14]. Another study has found that its prevalence in all psychiatric inpatients is 1.3–4.1% [25]. It is around 3% for hospitalized psychotic patients [17]. In a recent prospective study of patients hospitalized for a first psychotic episode, it was found that CS was diagnosed approximately 1 in 10 of patients. The prevalence was maximal among patients presenting schizophreniform psychosis 50%, brief psychosis 34.8%, and unspecified psychosis 23.9%, and the prevalence was moderate for a major depressive episode 15%, schizophrenia 11%, or delusional disorders 11% [14]. The most common psychiatric diagnoses in CS have been paranoid schizophrenia, schizoaffective disorder, and bipolar affective disorder [23]. CS has been linked with multiple pathologies. It has been described in psychiatric as well as organic disorders. In the last few decades, reports have increasingly stressed the aetiological importance of heterogeneity of conditions that have been found in the patients with misidentification syndromes like the Capgras delusion, including cerebrovascular disease, post-traumatic encephalopathy, temporal lobe epilepsy, postencephalitic Parkinsonism, viral encephalitis, migraine, vitamin B12 deficiency, hepatic encephalopathy, chronic alcoholism, hypothyroidism, pseudohypoparathyroidism, and dementia [23]. Schizophrenia remains the most common co-occurring mental disorder associated with case reports of Capgras delusion [25, 26]. Also, family history of psychosis is reportedly present in half of CS patients [20]. Medications and drug toxicity have also been reported to cause CS [27].

3. Explanations for Capgras syndrome

Since initial reports of CS involved patients with psychiatric illness, their close relations, and how they interacted with each other, early explanations of the delusion were predominately psychodynamic interpretations. There are several psychodynamic approaches. Consequently, these explanations included suggestions

that CS might develop out of Oedipal issues in women as a defence against hostility or incestuous, guilty desires, or out of hidden homosexuality in men. Later attempts to account for CS resulted in hypotheses of anxiety-induced regression of cognitive and emotional functioning, pathological splitting of internalized object representations, insufficiently repressed conflicting or ambivalent feelings toward the implicated person, and the projection of negative emotions that come to light from these conflicting feelings [17]. In the psychodynamic theory, it is supposed that the delusion is a way in which the patient copes with the ambivalent emotions that he feels toward the close family member who is duplicated [15]. There are several explanations brought about by psychodynamic approaches of misidentification syndromes. Premorbid psychopathology, motivation, and loss of ego functions may be important in determining which vulnerable patients develop CS [6].

Capgras delusion can occur due to 'spatial disorientation, anatomic disconnection, memory and executive process impairment, and loss of ego' [4]. While psychodynamic theories consist of ambivalence theory, depersonalization theory, and regression theory, neurocognitive hypotheses focus on right hemispheric dysfunction, face-recognition processing abnormalities, and focal structural cerebral abnormalities [28]. There are two components of the visual recognition of a familiar face, one of which is responsible for conscious recognition of the face and the remembrance of associated semantic information, while the other is responsible for the limbic-mediated emotional arousal including the feeling of familiarity that accompanies the conscious recognition of a known face [9].

4. Psychodynamic proposals in Capgras delusion

Despite the sharp increase in the number of published cases accompanied by various suggestions regarding an organic etiology, to accurately explain the delusion, it is necessary to embrace the psychodynamic as well as the organic. Even if a specific neuropsychological lesion is found in the end, the psychodynamics of the individual will still be pertinent and remain substantial [29]. An association between CS and depersonalization has been thought to exist onward the time when the disorder was first described. Some authors put forward that depersonalization may be the basis of the disorder which may develop in some individuals. CS can be evaluated as a disorder of ego function which permeates the entire personality [29]. Some authors postulated that cerebral dysfunction leads to feelings of derealization and depersonalization which in turn may develop into Capgras' syndrome in the presence of paranoid ideation [29].

The psychodynamic conception of the Capgras phenomenon is basically a love-hate conflict that is resolved by reflecting ambivalent feelings onto a fictitious double [29]. On the one hand, there are a long-standing love and on the other hand a visible hatred. In those cases when it occurs, it is very substantial that before the onset of the delusion of doubles, the patient shows an increased love and sexual desire toward the object. This overreaction results from a desire for reassurance regarding the love of the object and fear of losing it simultaneously. Theories suggested that CS could arise out of an Electra complex and incest desires, Oedipal problems, and latent homosexuality. Personality disintegration coupled with an evolutionary regression to more primitive modes of cognitive and emotional functioning; division of internalized object representations; ambivalent feelings toward a familiar other that are not sufficiently suppressed; and the feelings of anxiety, guilt, and anger resulting from this struggle are reflected onto imagined imposter [20]. Instead of approving these demands, the object becomes even more repulsed and is unable to cover up these feelings that clearly aggravate the situation, and a vicious circle is established [29].

5. Face-recognition system in Capgras delusion

Usually, we do not strive for facial recognition. The ability to identify people who we met before is a headstone of our social interactions. Face recognition is a multi-stage process ending with the identification of a person. Prosopagnosia is defined as loss of familiarity to previously known faces and the inability to learn to recognize new faces. Although these patients fail to recognize faces, they are still able to show affective responses to these faces [30, 31]. Several studies have suggested that CS represents a 'mirror image' of prosopagnosia, thus suggesting different neural circuits for facial processing: a cognitive circuit (impaired in prosopagnosia) and an affective circuit (impaired in CS). In the affective circuit, the ventral route from the visual centers to the temporal lobes may be protected, also active in conscious face recognition; however, the dorsal visual track that gives the face its emotional significance is damaged. A brief disruption of the ventral visual pathway leads to prosopagnosia, whereas damage to the dorsal visual areas leads to an impaired sense of familiarity for known faces, as in CS [9, 17, 30, 32]. While the ability to identify that person is intact, patient with CS probably has a brain lesion that interferes with the patient's ability to sense a familiarity toward the significant other [15]. It has been suggested that the impairment seen in the Capgras delusion was linked to a disruption of pathways connecting face-sensitive regions to limbic cortex, which is involved in the accompanying emotional response [30]. Perhaps arising from the conflicting experience of recognizing a known face without any accompanying affective reaction, the patient can understand that the absence of this emotional arousal is to establish the belief that the person he is looking at is an imposter [9, 33]. In another connectivity study, posterior coupled with anterior right hemisphere dysfunction may have involved in the emergence of Capgras delusion [34]. Also, it has been suggested that CS results from the disconnection of the face processing regions in the inferior temporal lobe from structures in the limbic system, especially the amygdala, which is very important in assigning emotional value to familiar faces [34]. Common to the CS is a fixed false belief but infrequently transient [35]. However, anatomical disconnection models fail to efficiently consider the transient nature of the misidentification episodes [34]. Therefore, it has been suggested that CS may be associated with the 'kindling of subcortical structures'. Kindling refers to repeated subthreshold stimuli which may result in psychomotor outbursts or overt seizure activity [34]. Autonomic responses and eye movements are involved in face perception which may cause the patient believe that the person has been replaced by an imposter. Studies on patient with CS like other psychiatric disorders have shown abnormal scan paths to facial stimuli or abnormal skin conductance response (SCR) in face processing tasks [30, 33]. The absence of identity recognition, accompanied by a lack of SCR, stimulates the patient to explore unfamiliar faces, and identity recognition of familiar faces leads to a more detailed exploration in the eye region, and it results in gaze avoidance of the eye region [33]. Vision is important in accessing reserved knowledge in the etiology of CS. However, surprisingly CS has also been reported in a number of blind patients which suggests that it cannot have an exclusively visual basis [34]. Some theories assume that two deficits are necessary for delusions to occur in the case of Capgras delusion like other DMSs [32, 36]. This is also called 'two-hit' process [20]. The first one, the brain's ability to attach emotional emphasis, may be the lack of autonomic arousal which leads to the abductive inference that the person is an imposter [30]. The other deficit is an impaired ability to reassess beliefs [the global consistency-checking mechanism] which prevents the rejection of the bizarre belief. The second deficit leads to the persistence of that abnormal perception as a delusion resistant to reasoning, also related to the right anterior cortex of the second deficit [9, 30, 32, 36].

6. Cognitive domains in Capgras delusion: memory, executive impairment, and confabulation

Neuropsychological deficits in patient with CS were reported across multiple cognitive domains, including memory, executive functioning, and visuospatial processing. These studies suggest that memory was statistically more likely to be impaired than other cognitive domains. Therefore, the memory may be playing an important role in the development of these delusions [32]. The existence of confabulations may have a role in prognosis and predicted significantly longer delusion duration, once more supporting the importance of memory impairment in patients with CS [32]. To mention a little more about the confabulation, some authors are focusing on confabulation in these patients because they are thought to be confabulation and delusion are closely related. When asked how they can explain their beliefs, Capgras patients will often confabulate. Confabulation is a kind of false memory that occurs when patients produce stories that fill in gaps in their memories, whereas a delusion is a mental state, typically thought of as a belief. Confabulation and delusion cannot be completely the same [37, 38]. Some researchers suggested that CS comes out when right hemisphere dysfunction causes a memory disconnection that leads to a failure to put new information together with representations about a significant individual and to keep in reserved over time [17]. Against all of these, although many patients have subtle deficits in face recognition and memory for faces, they do not have difficulty in recognizing faces in everyday life [1, 2]. CS is distinguished by its delusional mechanism: it is neither a hallucination nor an illusion—the object is correctly recognized in its appearance. CS is not a memory disorder. The person is correctly recognized; people are memorized [7]. Language deficits may not be absent, because of the right hemispherical dominance of the lesions [32].

7. Neuroanatomical and neuropsychological impairments in Capgras syndrome

In 1971 a case of Capgras was described in a young man following a head injury, with no previous history of psychiatric disorder. Since then, many patients with CS have undergone more thorough neurological investigations [29]. Identification disorders like CS are very frequent in neurodegenerative diseases [7]. Regarding the organic conditions that occur in Capgras delusion, this appears mainly in various types of dementia like Alzheimer, Lewy bodies, and Parkinson [39]. The prevalence of CS in Lewy body dementia may be as high as 25% and 10% in Alzheimer-type dementia. Identification disorders are much rarer in other types of dementia, especially those associated with Parkinson's disease [7]. Nearly half of the cases in CS were associated with neurocognitive disorders, such as delirium, traumatic brain encephalopathy, cerebrovascular disease, dementia, meningioma, encephalitis, and multiple sclerosis [21, 22]. Although there is usually a delay in the presentation of Capgras delusion after cerebral events, there are also such cases of immediate presentation [31]. Psychotic disorders with CS tend to present in the late teens and early twenties. It reflects the long mean duration of the delusion in the functional group [26]. Those with neurological disorder associated with the onset of the delusion had a mean age of 60, in keeping with their presentation in middle to late adulthood, especially as Capgras delusion in dementia tends to occur in the later stages [26]. Therefore all individuals with Capgras should be examined for organic pathology [9]. In a literature review of patients with CS who had associated organic factors, there are several single case reports in patients with Capgras delusion which

suggest structural and metabolic anomalies in mostly right-sided frontal, temporal, or parietal brain regions. But most of CS patients had bilateral lesions although, for those with unilateral lesions, right hemisphere lesions were much more likely [30]. Some studies give emphasis to the presence of two lesion sites, one in right frontal and the other in right temporal cortex [30]. The identity of the imposter is significantly associated with the reported underlying etiology. Capgras' delusion is reportedly due to functional psychiatric disorder, which is more likely to view their parent as an imposter, whereas the spouse is involved in those with suspected neurological etiology. There may be mentioned two reasons. The first one is may be because of the different mean age for the groups. The age of onset of Capgras delusion is different between those with organic disorders and those with neurological disorders [26]. The other reason is about Capgras delusion's feature. Capgras delusion is the phenomenon mostly specific to close relatives. This supports the role of intimacy [9, 26]. Selectivity for familiar persons is essential, though sometimes relative, and the syndrome can extend to persons who are simply known or famous [7]. Against this, the frequency with which strangers and multiple imposters are implicated in all cases of Capgras delusion can be up to 39% [26]. Multiple imposters are significantly more likely to occur in functional cases, while the involvement of inanimate objects would seem to suggest organic etiology [26]. The neuropsychological findings discussed may lead to some account of the possible mechanisms by which an abnormal experience may be generated in a subset of Capgras patients, but some researchers do not think in itself account for the formation of delusional belief [40]. Consequently, the explanation may offer a useful, helpful analysis of a certain step in the pathology of the CS in a subgroup of more neurological patients but could be unlikely to enlighten about delusions more generally or those with Capgras in the context of a functional psychosis such as schizophrenia or bipolar disorder [40].

8. Neuropsychological assessment for Capgras syndrome

The term CS does not demonstrate a well-defined mental disorder. Over the years various studies have suggested psychodynamic and neurophysiological interpretations for CS, and various aetiologies have been recommended for the condition's development [15, 17, 28].

Although frequently seen in psychotic cases, Capgras has also been associated with neurological disorders suggesting that the syndrome has an organic basis [14]. According to a study, CS patients were classified into groups according to whether or not they had evidence of neurological disorder. Some of the patients identified as having no neurological lesion might be found to have organic brain disease with more sophisticated imaging techniques or at post-mortem evaluation [41]. In another study, approximately one in five of patients with CS presented with organic mental disorders [1, 2]. Multiple hypotheses have been put forth regarding the underlying pathophysiology of CS. Some areas of the brain are responsible for the etiology of this disease. Results of structural and neuroimaging studies of CS provide support for an organic etiology [17]. Multiple studies and reports have remarked on CS in the setting of various neurological and neurodegenerative diseases [42]. There is a study that found more widespread bilateral frontal and temporal cortex atrophy in schizophrenia patients with CS than schizophrenia patients without the syndrome by using computerized tomography (CT) [17]. Likewise other studies using CT found global brain atrophy in combination with right hemisphere lesions in patients with dementia. There is also reported that positron emission tomography [PET] demonstrated

abnormal brain glucose metabolism in paralimbic structures and temporal lobes of patients with Alzheimer's dementia comorbid with CS and other subcategories of delusional misidentification syndromes [17]. Numerous neuropsychological researches support an association between CS and right frontal and temporal lobe abnormalities, and also many study reports indicate that patients with CS tend to have inferior scores on neuropsychological tests of frontal lobe function [17]. Even though less well documented, regions of the prefrontal cortex are also associated within facial processing: projections from the face processing areas in the right ventromedial occipitotemporal regions to the ventromedial prefrontal cortex via the uncinate fasciculus as well as limbic-thalamic pathways are well established [34].

9. The association between Capgras delusion and schizophrenia

Some people with schizophrenia exhibit this syndrome, but it is not related directly to schizophrenia itself; there are people with schizophrenia who do not exhibit CS, as well as people with CS who do not exhibit schizophrenia. The mean age of schizophrenic patients with Capgras syndrome is older than the age at which schizophrenia alone is usually expected to occur. When brain abnormalities of people with schizophrenia affect certain areas, CS and schizophrenia will occur concurrently. Capgras delusion and schizophrenia seem to be statistically related, at least in the case of the paranoid subtype of schizophrenia. It has been claimed that right hemisphere damage is a characteristic of schizophrenia; perhaps the imperfect evaluation of beliefs, which we have suggested, occurs as an outcome of damage to a particular area of the right frontal lobe, which is necessary for the occurrence even of the persecutory and grandiose delusions that are common in paranoid schizophrenia. Accounting the association of Capgras delusion with paranoid schizophrenia, the same neuropsychological deterioration of belief assessment is required for both, and in cases of patients with persecutory or grandiose delusions where the neuropathology also has affected the track from face recognition to the autonomic nervous system, Capgras delusion will also be existing [43, 44]. CS in paranoid schizophrenia may improve with successful treatment. But recurrence of illness may be accompanied by a return of delusional material [44].

10. Differential diagnosis of primary and secondary Capgras delusions

It is important to note that the Capgras delusion can be either a primary condition that is part of a 'mental illness' or a secondary condition that is the direct result of an organic disease of the brain. Also, the primary and secondary versions differ significantly in their presentation. In primary Capgras syndrome, the patient is more likely to be furious or violent toward the imposter. In secondary CS, the imposters do not change over time. This is different from the situation in schizophrenia where the delusions can vary [45]. The mean age of onset of the delusion was earlier in primary Capgras (mean age 32 years) than in secondary Capgras (mean age 48.5 years). Primary cases are more likely to have a subtle onset which evolved gradually, whereas secondary cases are more likely to have sudden-onset delusions. Primary cases show associated psychotic symptoms, particularly paranoid thought, whereas psychotic symptoms are not very often of the secondary cases. The patients with CS without apparent organic cerebral dysfunction were more likely to have experienced other psychiatric symptoms prior to the onset of the Capgras delusion than those with organic cerebral dysfunction [41]. Patients

with neurological impairments were more likely to regard the misidentification as benign or as due to illusory, whereas patients without evidence of neurological basis were more likely to appraise the delusions as being threatening [41]. Thus, hostility and violence are seen much more frequently in those patients diagnosed as schizophrenic than in other patients [46].

11. The role of Capgras syndrome in violence

Acting on delusions is a crucial clinical issue. There is a positive relationship between delusions and serious violent acts. Although the pathway from delusions to violent outcomes is not direct, the risk is greatly increased when symptoms are acute, especially at the time of initial presentation and if not treated [19]. And the risk also can be changed according to the etiology of delusions. There is a requirement to be concerned about the patient's tendency for violence and to evaluate for it thoroughly in Capgras delusion [45]. In patients with CS of an organic nature, violence may be associated with few or no affective manifestations (e.g., hostility, aggression, and auditory hallucinations), and may not be associated with paranoid elements [7]. Delusional symptoms in CS such as persecutory thoughts, threat-control symptoms, command auditory and/or visual hallucinations, and hallucinations of threatening content have all demonstrated to be significant predictors of violence act and aggressive behaviour [28]. If the patients are married, divorced, or separated, the most frequent doubles are the spouse. If the patients are single, the most frequent doubles are the siblings [19]. It should be noted that healthcare professionals may become the objects of delusional misidentification [42]. Because the double is usually assumed to have malicious, the CS could be characterized by hostility toward misidentified objects, and, later, it can lead to physical harm to others [19]. The assault associated with CS, the tendency to violence, cannot be attributed purely to the delusion's existence. Other factors are presumably to affect the possibility of violent act. A significantly higher tendency for interpersonal violence are men disclosed among male subjects, average age at 40 years old, with a history of aggressive behaviour and substance abuse; social withdrawal prior to the violent act is common, and the violence is usually well planned [19, 21, 22]. Persecutory paranoid motivations have been implicated as a key factor in acts of violence toward family members who constitute the majority of victims in CS [28]. Physical violence was expressed by 58.2% of patients with CS and 62.5% of patients with CS engaged in acts of interpersonal violence toward their close family members and caregivers [21, 22]. Mothers and spouses were the most frequently attacked group of relatives, respectively. Also, it was found that 1 of 10 Capgras patients attempted homicide [21, 22]. Most of the perpetrators were males suffering from mental disorders without organic association. A higher incidence of self-harm and suicide attempts, which is about 1 in 10 of patients with CS, was detected among females even in patients with psychiatric disorders and in patients with neurodegenerative disorders [21, 22]. Although in the usual cases, the misidentified object is a person, hence justifying the title of delusional identification of people, the CS is not restricted to person misidentification but can also involve other living or lifeless objects [7, 19]. Physical violence against objects was also common, such as setting fire to one else's estate [21, 22].

12. Differential diagnosis of Capgras syndrome

The differential diagnosis of patients who suffered CS is crucial. It is substantial to rule out the presence of brain disease in every patient with Capgras delusion [45].

Many patients with CS also present with medical illnesses of organic etiologies, associated with delusional misidentification; these patients may respond well to treatment of the medical condition underlying the onset of CS [47]. The syndrome should be differentiated from the quite common false recognitions which occur in confusional states and the transient misidentifications encountered in mania [8]. For this purpose, ending with a complete mental status examination, as well as thorough testing of cognition, is important. Neuropsychological testing and neuroimaging are often indicated. Clinicians should clarify the nature of the underlying psychiatric illness [45].

13. Key features and biomarker of Capgras syndrome

Low platelet monoamine oxidase (MAO) activity is a biochemical abnormality which is present in some psychiatric disease. Some authors suggested that the low platelet MAO activity might be proposed as a potential biochemical marker of CS. It is also thought that reduced *monoamine oxidase* activity in primary psychiatric patients with CS may give a piece of information to the pathogenetic mechanism underlying the reported cases of CS in organic patients without a primary behavioral disorder. However, study results show that platelet *monoamine oxidase* activity in patients with delusional misidentifications did not differ notably from that of schizophrenia and nonpsychiatric controls [41, 48].

The key features currently considered to be critical to the development of the Capgras delusion are as follows:

- There is an abnormal perceptual experience that is a prerequisite for the delusion.
- This perceptual experience is accompanied by a paranoid which leads to misattribution of the abnormal perceptual experience.
- The loss of normal response to known faces occurs in the context of more generalized derealization-depersonalization [41].

14. Treatment considerations

Delusion in CS should be treated timely because it can cause a dangerous condition [42]. However, there are no guidelines to assist clinicians to care for patients presenting with CS in selecting complementary examinations to be performed or in selecting treatment [7]. Likewise, the symptoms of DMS are very refractory to treatment despite various interventions including psychotherapy and pharmacotherapy approaches [19]. The CS like other DMSs is known to develop similar to the comorbid disorder that they accompany, disappearing after remission even though it is not unusual for them to continue after the disappearance of the comorbid disorder [7]. Thus, treatment of the underlying neurological or psychiatric conditions may not lead to remission of CS [27]. The presence of depersonalization, derealization or visual-perceptual disturbances, and other comorbidities may influence the treatment of CS [47]. The syndrome has been linked to dopaminergic overactivity, and serotonin abnormality has been implicated in some but not all studies. Similarly, reduced platelet monoamine oxidase activity has been noted by some but not by others [17]. According to the results of the case studies in the literature, CS patients are sometimes responsive to typical and atypical antipsychotics such as olanzapine, risperidone, quetiapine, sulpiride, trifluoperazine, and pimozide [19]. Pharmacological treatment of

CS is based on antipsychotics, antidepressants, anticonvulsants, and benzodiazepines considering patient needs and characteristics, but no control trials are available [49]. In the literature, there have been reported cases with a diagnosis of organic or functional delusional disorder associated with CS whose DMS responded well to pimozide that is well known for the treatment of monosymptomatic delusional disorders [49]. Experience with the new generation of atypical antipsychotics for the treatment of CS is quite limited. Although for patients manifesting any psychotic disorder, atypical antipsychotics are usually recommended because of the reduced risk of adverse effects [6, 47]. A crucial point of a case report is the positive outcome in response to antipsychotic medication [olanzapine] [49]. The combination of antipsychotic drug therapy and selective serotonin reuptake inhibitor (SSRI) may produce a positive outcome in patients with CS [15].

A case report also suggested the use of clonazepam which is benzodiazepine. In this case report, in addition to the antipsychotic properties of clonazepam, its anticonvulsant properties were also utilized in CS patient with the suggestion of some researches that found an over 90% incidence of electroencephalographic abnormalities in CS patients [26]. According to the results of the case studies, it showed a positive outcome in a patient with CS after treatment with mirtazapine that is also a serotonin 2A receptor antagonist, which could potentially afford its antipsychotic effects resulting in significantly decreasing the symptoms of CS [19, 27].

With patients who have progressive dementia, such as dementia with Lewy bodies, in which misidentification syndromes are common occurred, cholinesterase inhibitors have demonstrated benefit to reduce psychiatric symptoms [6].

Electroconvulsive therapy (ECT) has been reported to benefit either alone or in conjunction with antipsychotics, mood stabilizer, or antidepressant medication in patients with CS. It has been suggested that ECT provides permanent effective control of CS [42, 47, 49].

Psychotherapy may be beneficial in the treatment of selected patients with CS in order to reform the patient's relationship with his family. The psychoanalytic theories show that the emotions which the patient experiences in regard to the people with whom he is confronted are transferred to the imposters, and therefore, in this way from a safe delusional distance, the patient gives himself to refuse them without guilt, sometimes manifesting an aggressive behaviour toward them [21, 22]. It has been shown that group psychotherapy may also be beneficial by becoming less prone to feel hostile toward others, thereby weakening the delusional misidentification process for psychotic patients with DMS [40]. Cognitive behavioural therapy (CBT) may be a utilized form of psychotherapy intervention in some cases by assisting the patient to overcome the delusional beliefs [21, 22].

It is quite common in cases of delusion for his/her family members of the deluded person to be concerned about the delusion and to try to get rid of it by constantly challenging it [43]. It may be beneficial to know that just as an impairment in the interpersonal relationship between the patient and the object may occur before the onset of the delusion, an amelioration in this relationship is an essential factor in the amelioration of symptoms. Therefore treatment must include helping the partner or person implicated to gain insight and perhaps change their attitude toward the patient [29].

15. Conclusion

CS is a different neuropsychiatric symptom of interest to researchers over the past century. No approved questionnaires focus on CS. While noting that


the Capgras syndrome has no formal place in recognized diagnostic systems, it should be emphasized that this is of significance. It is crucial to keep them in mind as a possibility and to pursue any possible clues. Capgras delusion is a complex psychopathological phenomenon that presents in a wide range of psychiatric and neurological disorders with differing patterns dependent on the main etiology. Misidentifications in CS are fixed false beliefs and, therefore, represent true delusions. Even if when patients are confronted over and over with the illogical nature of the delusion, they keep their beliefs. Specific questions and interventions may assist to clinicians in successfully identifying patients with CS. In a series of interviews with these patients, some focus on identifying CS, rather than a single interview which is likely to increase the detection of the delusional misidentification. The clinician should always be mindful of the risk of aggression and homicide in CS.

Author details

Aslı Enzel Koc and Cicek Hocaoglu*
Medical School, Recep Tayyip Erdogan University, Rize, Turkey

*Address all correspondence to: cicekh@gmail.com

IntechOpen

© 2020 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. 

References

- [1] Breen N, Caine D, Coltheart M. Mirrored-self misidentification: Two cases of focal onset dementia. *Neurocase*. 2001;7(3):239-254. DOI: 10.1093/neucas/7.3.239
- [2] Feinberg TE, Roane D. Self-representation in delusional misidentification and confabulated "others". *Cortex*. 2017;87:118-128. DOI: 10.1016/j.cortex.2016.07.014
- [3] Alam A. A Brief Overview of the Capgras Delusion Brooklyn College. Available from: https://www.academia.edu/33990249/Brief_Overview_of_Capgras_Delusion
- [4] Lykouras L, Typaldou M, Mourtzouchou P. Neuropsychological relationships in paranoid schizophrenia with and without delusional misidentification syndromes: A comparative study. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*. 2008;32:1445-1448
- [5] Feinberg TE, Roane DM. Delusional misidentification. *Psychiatric Clinics of North America*. 2005;28(3):665-683. DOI: 10.1016/j.psc.2005.05.002
- [6] Barrelle A, Luauté JP. Capgras syndrome and other delusional misidentification syndromes. *Frontiers of Neurology and Neuroscience*. 2018;42:35-43. DOI: 10.1159/000475680
- [7] Christodoulou GN. The syndrome of Capgras. *The British Journal of Psychiatry*. 1977;130(6):556-564. DOI: 10.1192/bjp.130.6.556
- [8] Hirstein W, Ramachandran VS. Capgras syndrome: A novel probe for understanding the neural representation of the identity and familiarity of persons. *Proceedings. Biological Sciences*. 1997;264(1380):437-444. DOI: 10.1098/rspb.1997.0062
- [9] Currell EA, Werbeloff N, Hayes JF, Bell V. Cognitive neuropsychiatric analysis of an additional large Capgras delusion case series. *Cognitive Neuropsychiatry*. 2019;24(2):123-134. DOI: 10.1080/13546805.2019.1584098
- [10] Rodrigues ACT, Banzato CEM. Delusional misidentification syndrome: Why such nosologic challenge remains intractable. *Psychopathology*. 2006;39(6):296-302. DOI: 10.1159/000095777
- [11] Antonio V, Iris B, Fabiana R, Federica C, Vishal B. Delusion, possession and religion. *Nordic Journal of Psychiatry*. 2018 Sep;72(Suppl 1):S13-S15. DOI: 10.1080/08039488.2018.1525639
- [12] Gaines AD. Culture-specific delusions. *Psychiatric Clinics of North America*. 1995;18(2):281-301. DOI: 10.1016/s0193-953x(18)30055-8
- [13] Salvatore P, Bhuvaneswar C, Tohen M, Khalsa HM, Maggini C, Baldessarini RJ. Capgras' syndrome in first-episode psychotic disorders. *Psychopathology*. 2014;47(4):261-269. DOI: 10.1159/000357813
- [14] Mazzone L, Armando M, De Crescenzo F, Demaria F, Valeri G, Vicari S. Clinical picture and treatment implication in a child with Capgras syndrome: A case report. *Journal of Medical Case Reports*. 2012;6:406. DOI: 10.1186/1752-1947-6-406
- [15] Thiel CM, Studte S, Hildebrandt H, Huster R, Weerda R. When a loved one feels unfamiliar: A case study on the neural basis of Capgras delusion. *Cortex*. 2014;52:75-85. DOI: 10.1016/j.cortex.2013.11.011
- [16] Bourget D, Whitehurst L. Capgras syndrome: A review of the neurophysiological correlates and presenting clinical features in cases

involving physical violence. The Canadian Journal of Psychiatry. 2004;**49**(11):719-725. DOI: 10.1177/070674370404901102

[17] Edelstyn NMJ, Oyebode F, Barrett K. The delusions of Capgras and intermetamorphosis in a patient with right-hemisphere white-matter pathology. Psychopathology. 2001;**34**(6):299-304. DOI: 10.1159/000049328

[18] Karakasi MV, Markopoulou M, Alexandri M, Douzenis A, Pavlidis P. In fear of the most loved ones. A comprehensive review on Capgras misidentification phenomenon and case report involving attempted murder under Capgras syndrome in a relapse of a schizophrenia spectrum disorder. Journal of Forensic and Legal Medicine. 2019;**66**:8-24. DOI: 10.1016/j.jflm.2019.05.019

[19] De Pauw KW, Szulecka TK. Dangerous delusions, violence and the misidentification syndromes. The British Journal of Psychiatry. 1988;**152**:91-96

[20] Edelstyn NMJ, Oyebode F. A review of the phenomenology and cognitive neuropsychological origins of the Capgras syndrome. International Journal of Geriatric Psychiatry. 1999;**14**(1):48-59. DOI: 10.1002/(sici)1099-1166(199901)14:1<48::aid-gps891>3.0.co;2-0

[21] Jariwala PC, Shah ND, Dave KR, Mehta RY. Delusional misidentification syndromes in patients of paranoid schizophrenia: Case series and review. Telangana Journal of Psychiatry. 2018;**3**(2):125-129

[22] De Pauw KW. Psychodynamic approaches to the capgras delusion: A critical historical review. Psycho-pathology. 1994;**27**(3-5):154-160. DOI: 10.1159/000284864

[23] Silva A, Leong GB, Weinstock R, Sharma KK, Klein RL. Delusional misidentification syndromes and dangerousness. Psychopathology. 1994;**27**(3-5):215-219. DOI: 10.1159/000284872

[24] Pandis C, Agrawal N, Poole N. Capgras' delusion - A systematic review of 255 published cases. Psycho-pathology. 2019;**52**:161-173. DOI: 10.1159/000500474

[25] Khouzam HR. Capgras syndrome responding to the antidepressant mirtazapine. Comprehensive Therapy. 2002;**28**(3):238-240. DOI: 10.1007/s12019-002-0033-9

[26] Sinkman A. The syndrome of Capgras. Psychiatry: Interpersonal and Biological Processes. 2008;**71**(4):371-378. DOI: 10.1521/psyc.2008.71.4.371

[27] Carabellese F, Rocca G, Candelli C, Catanesi R. Mental illness, violence and delusional misidentifications: The role of Capgras' syndrome in matricide. Journal of Forensic and Legal Medicine. 2014;**21**:9-13. DOI: 10.1016/j.jflm.2013.10.012

[28] Albonico A, Barton J. Progress in perceptual research: The case of prosopagnosia. F1000Research. 2019;**8**:765. DOI: 10.12688/f1000research.18492.1

[29] Enoch MD, Ball HN. Uncommon Psychiatric Syndromes. 4th ed. Arnold; Hodder Arnold Publication, CRC Press; 2001

[30] Darby R, Prasad S. Lesion-related delusional misidentification syndromes: A comprehensive review of reported cases. The Journal of Neuropsychiatry and Clinical Neurosciences. 2016;**28**(3):217-222. DOI: 10.1176/appi.neuropsych.15100376

[31] Ellis HD, Young AW. Accounting for delusional misidentifications.

The British Journal of Psychiatry.
 1990;**157**:239-248

[32] Brighetti G, Bonifacci P, Borlimi R, Ottaviani C. "Far from the heart far from the eye": Evidence from the Capgras delusion. *Cognitive Neuropsychiatry*. 2007;**12**(3):189-197. DOI: 10.1080/13546800600892183

[33] Garcha M, Sivakumar K, Leary M, Yacoub HA. Transient Capgras syndrome secondary to bilateral ischemic stroke. *Cognitive and Behavioral Neurology*. 2018;**31**(2):96-98. DOI: 10.1097/wnn.0000000000000152

[34] Coltheart M, Langdon R, McKay R. Delusional belief. *Annual Reviews of Psychology*. 2011;**62**:271-298

[35] Hirstein W. The misidentification syndromes as mindreading disorders. *Cognitive Neuropsychiatry*. 2010;**15**(1-3):233-260. DOI: 10.1080/13546800903414891

[36] Estrada-Bellmann I, Ulloa-Escobar Y, Barbosa-Flores SL, Pech-George RE, González-Treviño R, Conde-Gómez L, et al. Capgras syndrome as a psychiatric manifestation in Parkinson's disease: A case report and literature review. *Medicina Universitaria*. 2015;**17**(66):49-52. DOI: 10.1016/j.rmu.2014.07.002

[37] Bortolotti L, Broome MR. If you didn't care, you wouldn't notice: recognition and estrangement in psychopathology, Philosophy, Psychiatry, & Psychology. 2007;**14**(1):39-42

[38] Ullrich S, Keers R, Shaw J, Doyle M, Coid JW. Acting on delusions: The role of negative affect in the pathway towards serious violence. *The Journal of Forensic Psychiatry & Psychology*. 2018;**29**(5):691-704. DOI: 10.1080/14789949.2018.1434227

[39] Cipriani G, Vedovello M, Ulivi M, Lucetti C, Di Fiorino A, Nuti A. Delusional

misidentification syndromes and dementia. *American Journal of Alzheimer's Disease & Other Dementias*. 2013;**28**(7):671-678. DOI: 10.1177/1533317513506103

[40] Spiegel DR, Laroia R, Samuels D. A possible case of Capgras syndrome after a right anterior cerebral artery cerebrovascular accident treated successfully with mirtazapine. *The Journal of Neuropsychiatry and Clinical Neurosciences*. 2008;**20**(4):494

[41] Breen N, Caine D, Coltheart M, Hendy J, Roberts C. Towards an understanding of delusions of misidentification: Four case studies. *Mind and Language*. 2000;**15**(1):74-110. DOI: 10.1111/1468-0017.00124

[42] Silva JA, Leong GB, Miller AL. Delusional misidentification syndromes. *CNS Drugs*. 1996;**5**(2):89-102. DOI: 10.2165/00023210-199605020-00002

[43] Coltheart M, Langdon R, McKay R. Schizophrenia and monothematic delusions. *Schizophrenia Bulletin*. 2007;**33**(3):642-647. DOI: 10.1093/schbul/sbm017

[44] Merrin EL. The Capgras phenomenon. *Archives of General Psychiatry*. 1976;**33**(8):965. DOI: 10.1001/archpsyc.1976.01770080083008

[45] Sharpless B. Unusual and Rare Psychological Disorders: A Handbook for Clinical Practice and Research. New York: Oxford University Press; 2017

[46] Jovic Z. Delusional misidentification syndromes. *Jefferson Journal of Psychiatry*. 1992;**10**:4

[47] Joseph AB. Delusional misidentification of the Capgras and intermetamorphosis types responding to clorazepate. *Acta Psychiatrica Scandinavica*. 1987;**75**(3):330-332. DOI: 10.1111/j.1600-0447.1987.tb02797.x

[48] Barton JL, Chaparala S, Barton ES, Jackson IV, Davis LG. Delusional misidentification and platelet monoamine oxidase. *Biological Psychiatry*. 1980;**15**(/2):275

[49] Torales J, Rodríguez H, Arce A, Moreno M, Riego V, Chávez E, et al. Olanzapine in the treatment of Capgras syndrome: A case report. *International Journal of Culture and Mental Health*. 2013;**7**(2):224-228. DOI: 10.1080/17542863.2013.775782