Capgras Syndrome: Introduction, Overview, and Literature Review

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Abstract

Capgras Syndrome (CS) is a delusional disorder, one of the Delusional Misidentification Syndromes (DMSs), in which the patient believes that an 'imposter' has taken the place of one of their loved ones. This, at instances, results in violence and aggression towards the supposed imposter. In this paper, the aim is to see to it that the syndrome receives the attention that it deserves and that the most recent advances in the research related to the syndrome are illustrated in a concise manner.

Keywords: Neuropsychiatry; Psychiatry; Neurology; Capgras' Syndrome; Delusional Misidentification Syndromes; DMS; Jean Marie Joseph Capgras; Dr. Capgras; Imposter Syndrome; Prosopagnosia

1. Introduction

Imagine the following, you wake up, only to find yourself surrounded by people who pretend to be your loved ones; even worse, they look, act, and talk in the same manner as your loved ones. Now imagine that these imposters dare not admit to their crime. Furthermore, you are unable to either prove your theory (remember, you have no proof, but your belief cannot be shaken), or 'shake' the belief. This is the unfortunate situation in which patients with Capgras Syndrome find themselves (1–4).

Capgras' Syndrome (CS) is one of the 'delusional misidentification syndromes (DMS)' (5) which are 'a group of disorders, characterized by patients mistaking the identity of people they know, although they recognize them physically' (5). This phenomenon extends to include non-human objects (5,6). Capgras' Syndrome (CS) is the most common DMS we know of (5).

CS is one of 4 sub-types of DMS, the other three being Fregoli syndrome, intermetamorphosis syndrome, and subjective doubles syndrome (5,7,8). Each of these alters the manner in which objects are identified. The author is currently working on review articles on the other 3; they are expected to be published soon following this publication.

DMS are also known as monothematic delusions (9) as they, for the most part, tend to relate to a certain topic

(2).
The delusional nature of the misidentification in general, as described by Karakasi and colleagues (2), is a 'rigid, unwavering, obsessive belief experienced in delusional definiteness, resentment and suspicion'.

2. Objectives:

1) To see to it that readers are familiarized with Capgras Syndrome
2) To see to it that this motivates researchers in the field of neuropsychiatry to further research the topic
3) To see to it that the topic is better mentioned and further illuminated in the literature associated with the topic.

3. Justification/Rationale:

The author noticed a lack of mentioning of Capgras Syndrome in modern psychiatry textbooks; ergo, he thought it be appropriate that he write a paper in which he discusses the topic in a clear, and simplified manner so as to see to it that the syndrome receives the attention it deserves.

4. Research Methodology

The author entered the entirety of the aforementioned keywords into a plethora of internet-based search engines, then he downloaded all available and relevant papers, after which he read through them and summarized their findings in the aforementioned manner.

5. Historical perspective:

As beautifully articulated by Karakasi and colleagues 'Jean Marie Joseph Capgras (1873–1950) was the first to describe the phenomenon, while in 1923, Jean Reboul-Lechaux reported an incident that he designated as "l'illusion des sosies" (the illusion of doubles).' (2) (10). Reboul Lachous, another French psychiatrist also played a role in the description of the syndrome (4).
Born in 1873 in a small village in France (3,11), little did anyone know that little Jean Marie Joseph Capgras, henceforth referred to only as either Capgras or Dr. Capgras, would grow to make extraordinarily rich contributions to the field of Psychiatry.
Dr. Capgras showed signs of intellectual prowess early on in his life as a student at Montauban lycee (11). This is, in addition, to the plethora of knowledge he must have received from his father's library (11). As per Luaute's 1986 paper,

'It was under the influence of one of his cousins, Dr Pecharman of the Asiles d'alienes de la Seine, that he [in reference to Dr. Capgras] then went into Psychiatry and ca me to Paris where he took the 1898 internship ex am for the Asiles d'alienes de la Seine and was awarded first place. He spent his internship at the Asile de

Ville-Evrard and later at the Clinique de l'Asile Ste Anne where he studied under lojJroy. He also took advantage of these years of training in Paris to study with lules Falret and with Valentin Magnan, the most eminent among the alienists of the period.' (11)

Another instance in which the character of Dr. Capgras shows in Luaute's writing is in the following excerpt from the aforementioned paper:

'From the time spent with Capgras and from his teaching, Professor H.F. Ellenberger learned the principle that everything must be verified. We know how well this great historian of Psychiatry was able to employ this principle and later to present it as one of the basic principles which must govern all historical research.' (11)

Weirdly enough, despite being internationally known for his unfathomable contributions to psychiatry and having a syndrome named after him, it seems as if this isn't the cause of fame for Dr. Capgras in his native France. As per Luaute, 'Joseph Capgras is much less weIl known in France for the syndrome which bears his name than as the co-author with Paul Serieux of a masterful work published in 1909 entitled 'Les Folies raisonnantes' ('Reasoning Madness') and sub-titled 'Le Delire d'interpretation'' (11).
In 1923, Capgras first defined the syndrome (henceforth either referred to as either Capgras' Syndrome or simply CS) that now holds his name (5,12).

6. Pathogenesis:

There exists a notion within the field of psychodynamics that CS is a 'psychotic' response to 'an unbearable ambivalence or regression to either an archaic way of functioning or to an earlier stage of development' (5) (13). However, in the process of data collection for this paper, I have found myself unable to come across empirical, credible literature to support this notion.

In the latter half of the 20th century, Ellis and Young worked tirelessly towards understanding CS, they attempted to use the advances made in the field of facial recognition to attribute CS to 'a change in the normal affective response' (5) that becomes manifest once familiar faces are presented (14,15).

This technology then evolved to include other senses, such as the olfactory and auditory senses (5,16). This has resulted in the manifestation of a thought process involving a duality of lanes (the dual lane hypothesis), a covered lane and an open lane which Barrelle and Luauté explain beautifully in their 2018 paper (5).
Despite these advancements, the dual lane hypothesis fails to explain the delusional nature of CS (5). This is due to it being rather unclear whether or not the emotional response is associated with CS (17).
Three hypotheses abound attempting to explain the delusional nature of CS through the introduction of a second factor accounting for the nature have arisen (5). Those being:

i) A failure of the so-called "system of evaluation of convictions" (18), or as I prefer to call it, the 'conviction-evaluation system'.
ii) A manifestation of abnormalities of salience (19)
iii) "in abductive inference processes" (5,20)

A rather peculiar 1997 case report by Hirstein and Ramachandran (21) brought into question, at least in part, our understanding of the underlying pathology bringing CS into being. In that report, Hirstein and Ramachandran illustrated the case of a patient whose CS only manifested in telephone calls to the supposed 'imposter'.
An analysis of 9 cases in Taiwan showed rather clearly that organic illness is a major consideration when

discussing CS (22), despite the evidence, presented in this paper, and in a 1999 paper (1) for CS being primarily psychiatric as opposed to organic in nature.

7. Aetiology:

'CS is distinguished by its delusional mechanism: it is neither a hallucination – the object is present – 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.' (5)

Lest we forget the value of advances in DMS to the field of neuroscience, it is important that we note that the advances in DMS have created the underlying foundation upon which we improved our understanding of normal human cognition (5,23).
A plethora of proposed etiologies exist attempting to explain Capgras' Syndrome; the following is a list of proposed etiologies behind CS:

1) Frontal Lobe damage resulting in a disruption in familiarities, combined with right hemisphere damage, resulting in visual recognition disturbances (3,24)
2) 'Diminished function' of the right hemisphere solely (2)
3) A, yet unclearly articulated, form of prosopagnosia (2,3) (defined as the 'Inability to recognize familiar faces that is not due to impaired visual acuity or level of consciousness.') (25)
4) An occurrence that is secondary to an Oedipus or Electra complex (3)
5) A possible result of 'repressed' feelings? (3)
6) Secondary to brain pathology that results in loss of connection between the parts responsible for vision and those responsible for processing the visual information (3)
7) Guilt regarding sexual dysfunction (3)
8) 'Reduplicative paramnesia and other delusional misidentification syndromes (which believe a location has been relocated or duplicated) are similar to Capgras syndrome.' (3)
9) Secondary to heroin dependence (26)
10) Dopamine deficiency/impaired emotional response (2)
11) Secondary to Moyamoya Disease (MMD) (27)
12) Secondary to Early-Onset Alzheimer Disease (EOAD) (12)
13) Secondary to schizophrenia/schizoaffective disorders (12)

Despite CS being previously associated with purely psychiatric disorders, more recent studies seem to suggest that CS may, at instances, present in non-psychiatric disorders/conditions; this is suggestive of either an organic cause (12) or multiple etiologies.
Despite the above, it seems as if primary psychiatric disorders are primarily to blame for CS as opposed to morphological abnormalities of the brain (28).
It seems as if there exists a significant association between neurodegenerative illnesses and CS (5). However, as aforementioned, CS far more commonly manifests itself as a result of a primary psychiatric illness (28). CS almost never manifests itself solely (5).
Despite this relatively unclear etiology, CS is the most commonly presenting and the best studied DMS (5).
I tend to agree with the notion presented by Barrelle and Luauté, that being that it is of great importance that we make it clear that despite the notion that CS is, for the most part, the direct result of a primary psychiatric

illness; there exists a considerable number of individuals who present with CS do so as a result of a, usually curable, underlying organic illness (5).

In so far as the relationship between CS and DMS in general is concerned, it is rather documented that CS frequently is associated with other DMS (5); in fact, DMS usually evolve from one another (6). As per Barrelle and Luauté 'Common to all DMS is the delusional negation of identity of objects having affective importance for the patient, and these objects are limited in number' (5) (29).

A 2007 paper established an association between CS and neurodegenerative illness, most specifically Lewy Body disease (30). Furthermore, the same paper also makes it clear that this is usually associated with older age, meanwhile, younger aged patients who present with CS usually do as a result of primary psychiatric illness (30).

CS rarely manifests in children (3).

8. Imaging findings:

'an increasingly common clinical conundrum: is an abnormality identified on MRI causally related to the patient's symptoms or an incidental finding? Answering this question is particularly difficult for symptoms such as Capgras delusion, which can be due to lesions in multiple different brain locations, but can also be due to primary psychiatric disease.' (28)

In 2017, Darby and Fox (28) published an article in which they discussed a case reported by Ferguson and colleagues (31). The report showed a 'small area of gliosis in the left frontal periventricular white matter of indeterminate age.' (28).

Furthermore, there is credible reason to ascertain the significance of these findings. That being the fact that prior to the publication of this case report by Ferguson and colleagues (31), Darby and colleagues (32) published a paper in which they discussed the findings associated with 16 lesions in association with 'delusional misidentifications'. The cause behind our claim to the significance of the findings by Ferguson and colleagues is due to the reported findings being of 'a different connectivity profile' (28) than the aforementioned 16 lesions. This gives rise to a thought in which we believe that 'an alternate neuroanatomical substrate for delusional misidentifications' (28) may have manifested itself in such an instance. The former paper by Darby and colleagues (32) illustrates areas in which dysfunction would result in a delusional misidentification (such as CS).

As per Wacholtz's findings in the late 90s, lesions specific to CS seem to be located in the temporal and frontal lobes of the right hemisphere rather commonly (33).

In so far as DMS in general is concerned, patients who exhibited DMS following focal neurological injuries were, for the most part, injured in the right hemisphere, specifically the frontal aspect of the right hemisphere (5,34,35)

9. Clinical Presentation + Epidemiology:

First and foremost, it is of the upmost importance that it is made clear that CS is a "hypo-identification" as opposed to other DMS where "hyper-identification" is the mainstay/presenting symptom (5,36). As per Barrelle and Luauté:

'This distinction between hyper- and hypo-identification is used as the basis for the most widely used classification of the DMS. Other phenomena of hyper-identification and duplication of persons

have been described in recent years. They only justify inclusion in the DMS group if they take place in the presence of the misidentified object (a necessary characteristic owing to the description of the original case by Capgras), which excludes entities where duplication is the result of various delusional, imaginative, or hallucinatory mechanisms.' (5)

In summary, the epidemiology of CS is rather unclear (5), however, there exists some data in so far as the epidemiology of CS is concerned. The information is as follows:
Data reported by Barrelle and Luauté (5) and Salvatore and colleagues (9) show some rather intriguing data in relation to CS, that being that CS was of greatest incidence among research participants who presented with a schizophreniform disorder (50%), a 'brief psychotic disorder' (35%), or an unspecified psychosis (24%); it was moderate for a major depressive episode (15%), schizophrenia (11%), or a delusional disorder (11%) (5,9).
Early data seems to indicate a greater incidence of CS amongst females, at an approximately 2:1 ratio (37).
The association between CS and Lewy body dementia seems rather staggering at approximately 25% (38) and approximately 10% at 'Alzheimer-type dementia' (39). However, it is of great importance that we note that identification disorders (such as CS) are a rather common incident in neurodegenerative illnesses (5), another fact of importance is that DMS are a rather rare manifestation of other types of dementia, such as Parkinson's disease (5).
CS can be seen in patients with late-onset Alzheimer Disease (AD); this occurs in ranges of ~10-15% of patients with late-onset AD (12). In those patients with AD, the mean age of onset of CS is between 72-82 years (12).
Of course, as is understood in the field of medicine, this is not always the case; Ng and colleagues reported a case involving a 50-year old male who presented with CS secondary to early-onset AD (EOAD) (12).
Approximately 33% of DS cases seem to be a manifestation of an organic illness, or, at the very least, seem to have an organic component to them, usually neurodegenerative illness (40).
Patients with CS usually attempt to justify their delusion through a detail they somehow manage to 'catch' the 'imposter' missing or manifesting that the 'original' version would never commit/miss, etc.; this justification is sometimes physical or metaphysical (e.g. a personality trait) (5,36,41). This is usually manifest through the sense of vision; however, other senses (e.g.: auditory) may manifest themselves (5,16).
The clinical presentation is usually expected to vary depending on the underlying etiology (5), for example, in patients who manifest CS in a neurological context, the syndrome usually exhibits without 'affective manifestations' (e.g.: violence towards the imposter) (5) (42).
It is of importance that the treating team always keeps in mind the plethora of definitions for the clinical presentation (the differential diagnoses); a good example of when this manifested itself as rather necessary was in the case reported by Ng and colleagues (12) in which, if it weren't for the dedication and focus of the treating group, the case could've easily been diagnosed as late-onset schizophrenia, especially given the similarities that made themselves manifest (12,43).
As aforementioned, it is also possible for CS to be a manifestation of moyamoya disease, however, this, so far, seems to be a rather rare occurrence (27). Approximately 14% of patients with moyamoya disease primarily present with symptoms of psychiatric illness (12), and 66% of MMD patients first present with cognitive impairment (44).
Approximately speaking, 43% of patients suffer from an organic illness of sorts (e.g.: dementia, traumatic brain injury TBI, cerebrovascular illnesses, etc..) (12,35,45–48).
The initial presentation of CS is usually alarming enough for the patient to present to a physician rather rapidly. However, there are instances, like the one reported by Shah and colleagues (49) in which the presentation starts (in this case 4 years PTA), but is relatively unalarming, and increases in intensity some time following that (as to be expected, in a case not receiving medical attention) (in this case only 5 months

PTA) (49). So, it is of significance that this is articulated.
As per Pereira and Oliveira (50) '[discussing phenomenon affecting the world's ageing population] dementias have the most significant impact on the elderly', the leading cause of dementia is Alzheimer's disease (AD) (51). This is significant as psychosis presents in 42-84% of AD cases, significantly manifesting as DMS (52).

10. Treatment:

Aker and colleagues (4) published a case report in which they used olanzapine 10 mg on a drug-naive patient who they diagnosed with Capgras' Syndrome. This was followed by a weekly observation of the patient. Unfortunately, it is unclear in the abstract of their paper (the only accessible part of their paper) whether or not the treatment was effective. A paper out of India also lists antipsychotics as a treatment option for patients who present with Capgras' Syndrome secondary to schizophrenia.
In a paper out of Slovenia, published in 2018, the authors used 'daily directly supervised methadone treatment' to treat a patient who presented with Capgras' Syndrome following heoin dependence (26).
Of course, there exists the general, well-known, rule in Medicine in which we address the underlying cause of whatever illness manifests itself in our practices whenever discussing treatment. I believe it is of the upmost importance that we remember this notion whenever cases of CS make themselves manifest.
DMS in general have no written guidelines that aid HealthCare Workers (HCWs) in the management of CS or any other DMS (5). However, proper understanding of the underlying etiology significantly aids in the management of the outcome.
CS presenting secondary to curable neurological disorders seem to manifest a higher rate of remission as opposed to CS occurring as a result of primary psychiatric illness (5) (53).
General accepted notions seems to suggest that the drug therapy of choice for CS is with neuroleptics (42). Pimozide seems rather favourable (5,54,55) as a treatment option.
Other treatment options that have piqued interest in research professionals of the field include mirtazapine and electroconvulsive therapy ECT (5,56,57).
There is currently a floating theory of using CBT or hypnosis as a treatment option for CS (5).
For the most part, it seems as if treatment of the underlying cause results in the notion of misidentification subsiding (2), despite this not always being the case (2).
Psychotherapy plays a cruicial role, as it significantly aids in the amelioration of the patient's relationship with their respective family. Theories and hypotheses pertaining to the field of psychoanalysis seem to articulate that the emotions felt by the patient regarding the people with whom he is confronted (fear, anger) are mirrored and 're-manifest' themselves whenever the patient is interacting with the 'imposters'; ergo, it seems rather logical to assume, based on this knowledge, that psychotherapy may greatly aid the patient in rejecting the notions without the feeling of guilt (2,58–61).

11. Recent advances

The 21st century has been witness to a plethora of, until that point, hitherto undreamt of advances. These advances have become rather manifest in the field of neuroscience, especially in our understanding of DMS. We are now at a point where we can now define and study DMS in general and CS in particular through the lens of neurological facial recognition (the way by which we recognize faces). This is because of the unfathomable recent advances in our understanding of this from the neuroscientific perspective (14).
These advances have resulted in the incorporation of other senses (olfactory, tactile, etc.) into the trans-modal

system that became manifest as a result (16). However, as aforementioned, this system of interpretation fails to fully explain the syndrome.
As per Barrelle and Luauté 'Taking into account the delusional character of the disorder, some dual-factor models have been proposed, where the primary perception anomaly – loss of emotional familiarity – is associated with a secondary defect in one or several processes of the information treatment chain' (5,18,20)

12. Conclusion(s):

'These disorders have a practical interest for clinicians as much as an epistemological interest. Violent behavior has often been reported in patients suffering from a DMS: the hostility manifested towards "imposters" ranges from verbal or physical aggression to homicide, with some cases of parricide and infanticide' (5)

These instances (62) make it of the upmost importance that we act in a manner which assures that not only do we understand the syndrome (CS) and DMS in general better, but that we reach a point in which the public better understands the syndrome and its manifestation as well as understand how it is that we can predict these instances and, ergo, act in a manner that would prevent them from becoming manifest.
As of this writing, it seems rather clear that a multidisciplinary approach has manifested itself as a necessity in managing CS (2).

13. Recommendations:

1) Increased funding directed towards the study of DMS to see to it that our understanding of the topic is increased.
2) The introduction of CS into the educational system so as to pique the interest of young researchers and, ergo, aid in the advancement of our understanding of CS. This, in turn, will result in improvement of management of the syndrome.

14. Abbreviations (in alphabetical order):

AD = Alzheimer Disease
CS = Capgras' Syndrome
DMS = Delusional Misidentification Syndrome
EOAD = Early-Onset Alzheimer Disease
HCW = Health Care Worker
MMD = MoyaMoya Disease
PTA = Prior To Admission

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