Delusional Misidentification Syndromes Following Acquired Brain Injury: A Scoping Review of Clinical Manifestations, Neuroanatomical Bases, and Treatment Approaches

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Abstract

Objective: Delusional misidentification syndromes (DMS) are rare psychopathological phenomena characterised by fixed false beliefs about the identity of familiar others (e.g., Capgras, Fregoli) or the self (e.g., Cotard's) which can present significant clinical challenges, including risks to patient safety and distress for family members. Although most commonly described in psychiatric and neurodegenerative contexts, DMS may also occur following acquired brain injury (ABI), providing a valuable window into the neurocognitive mechanisms underlying familiarity processing, self/other representation, and belief evaluation, as well as informing clinical management of these complex presentations. This review aimed to scope the available literature on the development of DMS following ABI.

Method: A scoping review was conducted across PsycINFO, CINAHL, and MEDLINE from inception to February 2026. From 173 initial records, 34 studies were eventually included.

Results: Most studies were single-case reports, with only one observational cohort. Eighteen reported Capgras as the primary presentation following ABI, while four reported Fregoli, and six Cotard's. Additional studies described mixed, variable, or longitudinal presentations. Neuroanatomical findings most consistently implicated right-hemisphere fronto–temporo–parietal networks with prominent frontal involvement. Treatments were inconsistently reported and outcomes varied from complete remission to chronic persistence.

Conclusions: DMS following ABI are rare but clinically significant phenomena, with Capgras syndrome representing the most frequent presentation. Converging evidence supports a network-level involvement of right-hemisphere frontal-temporal-insular areas underpinning familiarity processing, self-representation, and belief evaluation. Further exploration of DMS

following ABI is warranted to support inform diagnosis, risk assessment, clinical management, interventions, and communication with affected individuals.

Keywords

DMS, Capgras Syndrome, Cotard's Syndrome, Fregoli Syndrome, Misidentification, Delusions, Brain Injury.

Introduction

Delusional misidentification syndromes (DMS) are a group of rare psychopathological phenomena in which a person holds a false belief about the identity of someone, themselves or places (Grover et al., 2025). These syndromes are typically secondary to an underlying medical, neurological or psychiatric disorder (Darby & Prasad, 2016; Devinsky, 2009). They are more commonly described in psychiatric and neurodegenerative settings than in the context of acquired brain injury (ABI; Pandis et al., 2019).

DMS are not recognised as discrete diagnostic entities within either Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR; American Psychiatric Association, 2022) or International Classification of Diseases 11th Revision (ICD-11; World Health Organization, 2022) and are instead conceptualised as forms of delusional content occurring within broader psychiatric and neurocognitive disorders. This diagnostic ambiguity has contributed to a fragmented literature and highlights the need for synthesis of DMS presentations following ABI. Individuals affected by DMS generally hold a fixed, false belief that the identity of a familiar person, place, object or even themselves has been changed, duplicated, or otherwise altered (Klein & Hirachan, 2014). Their presentation exhibits difficulties at the intersection of perception, recognition, affective familiarity, and belief evaluation, where a correctly perceived stimulus fails to evoke the normal sense of emotional familiarity, leading to the formation of a delusion explanation (Ellis & Young, 1990; Devinsky, 2009; Hirstein & Ramachandran, 1997).

It is important to note that DMS can co-occur (Ventriglio et al., 2020) and can in fact often evolve into one another (Pąchalska, 2019). Therefore, they are thought not to reflect unique syndromes or be indicative of a specific condition (Grover et al., 2025). However, when DMS primarily concern the recognition of persons and self-identity, they are often referred to

as ‘person centred’ or ‘self-related’ misidentification syndromes. These are believed to implicate social cognitive and affective processing networks (Ventriglio et al., 2020) and include delusions such as Capgras, Fregoli, Cotard’s, intermetamorphosis, and subjective doubles, which are described below. Table 1 provides a general summary of the core phenomenological and distinguishing features of each type of DMS.

Capgras Syndrome

Capgras syndrome is characterised by the delusion that a familiar person has been replaced by an identical imposter (Capgras & Reboul-L’Achaux, 1923). It was first described by the French psychiatrist Joseph Capgras and his colleague Jean Reboul-Lachaux in a woman characterised by preserved facial recognition but the loss of emotional familiarity. It is considered a relatively uncommon phenomenon in general psychiatric populations, with prevalence estimates ranging between 1.3% to 2.5% within psychiatric populations (Huang et al., 1999; Tamam et al., 2003). Higher rates have been reported in more specific samples, particularly among individuals experiencing first-episode psychosis, where Capgras syndrome has been identified in up to 14.1% of cases, especially within acute schizophreniform and brief psychotic presentations (Salvatore et al., 2014). A high level of variability was also reported in a large retrospective study of 25,000 clinical case records which identified 84 individuals with Capgras syndrome distributed across several diagnostic categories, including schizophrenia, bipolar and depressive disorders, Alzheimer’s disease, schizoaffective disorder, and other delusional disorders (Bell et al., 2017). In neurological populations, and dementia in particular, Capgras syndrome appears to be more common than previously recognised, with a systematic review and meta-analysis of Alzheimer’s disease cohorts estimating a pooled prevalence of approximately 6% among individuals with Alzheimer’s disease (Pereira & De Oliveira, 2019).

A limited evidence base makes the effective management of Capgras syndrome challenging, with treatment typically combining psychological support with antipsychotic medication (Barrelle & Luauté, 2018). The research indicates that pharmacological interventions should be directed at the core features of the underlying condition (Khouzam, 2002).

Fregoli Syndrome

Named after Italian actor Leopoldo Fregoli, who was famous for his rapid on-stage costume changes, Fregoli syndrome involves the belief that different people are, in fact, a single person who is changing appearance or disguising themselves (Courbon & Fail, 1927). It was first described by French authors Paul Courbon and Georges Fail in a woman who believed that two known actresses were following and persecuting her by appearing as various people in disguise. Robust epidemiological estimates for Fregoli syndrome in psychiatric populations are limited, and the literature is dominated by case reports rather than case-finding studies. Case-level meta-analytic work on Fregoli syndrome further supports that a meaningful minority of reported cases occur in the context of neurological disorders, including neurodegenerative disease, but this approach does not yield population prevalence figures (Teixeira-Dias et al., 2023). As with Capgras, there is no consensus on the treatment of Fregoli syndrome, however, a recent meta-analysis of 89 patients presenting with Fregoli syndrome with either primary or secondary psychosis found the most common treatment to be exclusively antipsychotic medication (Teixeira-Dias et al., 2023).

Cotard's Syndrome

Cotard's syndrome is marked by the delusion that one is dead, does not exist, or has lost their internal organs or blood (Cotard, 1880). Cotard's is a nihilistic delusional disorder and, unlike Capgras and Fregoli, the focus of the misidentification in on the self rather than

others. It was fist described by French neurologist and psychiatrist Jules Cotard's in the late 19th century and is considered especially rare, with prevalence estimates ranging between 0.62% in psychiatric patients and 0.11% in neurological populations (Ramirez-Bermudez et al., 2010). In a study of older geriatric patients in Hong Kong, Cotard's syndrome was identified in the 0.57% of a 349-patient sample, suggesting it is uncommon even in specialist dementia settings (Chiu, 1995). Due to the profound sense depersonalisation and derealisation which characterises it, Cotard's is often associated with suicidal ideation, despite individuals believing that they cannot die as they are already dead (Debruyne et al., 2009; Dieguez, 2017). There is no clear consensus on optimal treatment of Cotard's delusion. However, emerging evidence suggests the use of atypical antipsychotics either as monotherapy or in combination with mood stabilisers or antidepressants (Couto & Moreira Gonçalves, 2021), with some reports specifically supporting the use of olanzapine and fluoxetine (Fusick et al., 2024).

Intermetamorphosis Syndrome

Intermetamorphosis syndrome is the belief that both familiar people as well as strangers in an environment have exchanged identities while maintaining their original physical appearance (Courbon & Tusques, 1932). Unlike Capgras syndrome, in which a familiar person is replaced by an imposter, intermetamorphosis involves the conviction that individuals transform into other known people, adopting their identity, personality, and intentions. The syndrome was first described by French psychiatrists Courbon and Tusques in the early 20th century and is considered exceptionally rare. Robust prevalence estimates are not available; however, reviews of published cases suggest that intermetamorphosis accounts for only a small minority of reported DMS (Förstl et al., 1991). Intermetamorphosis has been described in both psychiatric and neurological contexts, most commonly in association with schizophrenia-spectrum disorders, dementia, and ABI, and frequently co-occurs with or evolves alongside

other delusional misidentification phenomena (Christodoulou et al., 2009) with no current consensus on treatment (Förstl et al., 1991).

Subjective Doubles Syndrome

The syndrome of subjective doubles is characterised by the belief that one or more doubles of the self-exist, typically perceived as physically identical but psychologically or morally altered. The syndrome was first described by Christodoulou (1978), who reported a patient with a diagnosis of schizophrenia who believed that another person existed who was an exact double of himself, sharing his physical appearance but acting independently and often malevolently. In psychiatric populations, the syndrome of subjective doubles is considered exceptionally rare, and robust prevalence estimates are not available. Narrative reviews of delusional misidentification in dementia report occasional cases in Alzheimer's disease and other neurodegenerative conditions, typically in the context of advanced cognitive impairment and prominent neuropsychiatric symptoms (Cipriani et al., 2013). However, syndrome-specific prevalence data in dementia populations are lacking.

Reverse subjective doubles syndrome, introduced by Silva et al. (1989), represents a related but distinct phenomenon in which the individual believes that they themselves are duplicated, or that their own identity, thoughts, or actions are attributed to another person. This variant reflects a more profound disruption of self-representation and agency, with misidentification directed inward rather than toward an external double. As with subjective doubles, reverse subjective doubles syndrome is documented almost exclusively through isolated case reports, precluding meaningful prevalence estimates. Similarly, evidence regarding treatment is limited to isolated case reports, most commonly involving antipsychotic medications (Arturo Silva et al., 1994; Gonzalez et al., 2025).

Other Delusional Misidentification Syndromes

Reduplicative paramnesia was first describe by Pick (1903) and is characterised by the fixed belief that a place (or less commonly a person or object) has been duplicated or exists simultaneously in multiple locations. Dysmetropsia refers to a perceptual disturbance in which the size, distance, or spatial relationships of objects or one's own body are distorted (e.g., micropsia or macropsia). The syndrome was first described by Lippman (1952) and later named by Todd (1995).

Both reduplicative paramnesia and dysmetropsia have been reported in the context of neurological conditions, including ABI and dementia, but they remain uncommon and are largely described through case reports and small case series, limiting reliable prevalence estimates in both neurodegenerative and psychiatric populations (Diamantaras et al., 2023; Mastria et al., 2016). Treatment is not standardised and is typically informed by the underlying condition, with no consensus on specific pharmacological or non-pharmacological interventions.

DMS Following Brain Injury

Although DMS have been most extensively studied in psychiatric and neurodegenerative populations, they can also emerge following ABI, which suggests a link between specific brain regions and the emergence of such delusional disorders (Young et al., 1992). This occurrence provides a valuable opportunity to investigate the neurocognitive mechanisms underpinning these phenomena, as injury-related disruption to perceptual, affective, mnemonic, and belief-evaluation systems such as the dual routes of face recognition may contribute directly to their development.

First introduced by Bauer (1984), the of dual routes model of face recognition identifies two distinct visual pathways: a ventral one connecting the visual cortex via the inferior

longitudinal fasciculus to the temporal lobes, and a dorsal one linking the visual cortex to the limbic system through the inferior parietal lobule. According to this model, damage to these pathways may result in DMS. Additionally, the dual-process model of recognition – which posits that memory recognition abilities rely on both recollection and familiarity processes (Gardiner & Richardson-Klavehn, 2000) – aligns with Devinsky's (2009) hypothesis that dysfunction in familiarity processing, potentially arising from bilateral frontal and right temporo-parietal lesions, contributes to the development of delusions.

Researchers have suggested that DMS arising in the context of ABI result from a combination of neuropsychological impairments and deficits in belief evaluation systems (Darby et al., 2017; Darby & Prasad, 2016). According to Coltheart (2010), DMS emerge from a two-factor model. The first factor involves a neuropsychological anomaly that prompts the initial formation of the delusional belief – in Capgras delusion, for instance, the absence of autonomic responses to familiar faces generates the belief that a loved one has been replaced by an imposter (Ellis et al., 1997; Hirstein & Ramachandran, 1997). Similarly, mirrored-self misidentification may stem from impairments such as mirror agnosia or a mismatch in face processing systems (Breen et al., 2000). The second factor, critical for the delusional belief to persist, is an impairment in the cognitive system responsible for hypothesis evaluation. This deficit prevents the individual from rejecting the delusional belief despite contradicting evidence. For example, when Capgras, Fregoli, and Cotard's patients are presented with clear evidence contrary to their delusions, they fail to revise their belief (Coltheart, 2010). Neuroimaging studies have implicated the right lateral prefrontal cortex (RLPFC) as a key region associated with hypothesis evaluation, and disruptions in this area have shown to be linked to delusional severity (Corlett et al., 2007; Fletcher et al., 2001).

Furthermore, while the specific content of delusions is generally shaped by the nature of the associated neuropsychological impairments, the generalised impairment in belief

evaluation remains consistent across cases. This may help explain why patients with similar neuropsychological deficits may not always develop the same delusions, as both factors need to be present. For example, patients with face-processing deficits do not always experience DMS unless the evaluation system is also impaired (Ellis et al., 1997; Tranel et al., 1995). Thus, delusions are hypothesised to result from the interplay of specific neuropsychological impairments and a deficit in belief evaluation systems (Coltheart, 2007; Coltheart et al., 2007).

Despite strong theoretical grounds for the emergence of DMS following ABI, the empirical literature remains sparse, fragmented, and dominated by single-case reports spanning multiple disciplines and diagnostic traditions. Existing reports vary widely in phenomenological focus, terminology, and methodological quality, and no synthesis has yet systematically mapped the range of DMS presentations, aetiologies, neuroanatomical correlates, and outcomes following ABI. As a result, the prevalence, mechanisms, and clinical implications of post-ABI DMS remain poorly characterised. Understanding these conditions better through research can improve diagnostic accuracy, leading to earlier and more effective interventions. Additionally, by exploring effective treatments and management strategies, research can enhance the quality of life for those affected by these conditions and inform clinicians on best practices, ultimately contributing to better patient outcomes.

Although previous reviews have examined DMS across mixed-aetiologies, (e.g., Teixeira-Dias et al., 2023), no review has specifically synthesised evidence relating to ABI. Thus, the present study aimed to scope the existing literature on the clinical manifestations, neuroanatomical bases, and treatment approaches in individuals who developed Capgras, Fregoli, and Cotard's delusions following ABI.

Methods

Methodological Approach

A scoping review approach was adopted for this study (Arksey & O'Malley, 2005a). Scoping reviews are used as an efficient method to explore and summarise bodies of literature which are still in their infancy and are characterised by paucity of studies (Arksey & O'Malley, 2005a) and was thus utilised to complete a rigorous and transparent process that mirrors the credibility of a systematic review methodology (Centre for Reviews and Dissemination, 2008). The five-stage process outlined in the latest guidance by the Joanna Briggs institute (Peters et al., 2020) was utilised in order to ensure methodological robustness. These include (1) identifying a research question, (2) identifying potentially relevant studies (3) selecting studies to include, (4) charting the data found and (5) ordering, summarising, and describing the results

Identifying the Research Question

The following research questions guided this review: i) what are the clinical manifestations of person-centred misidentification syndromes following ABI? ii) what are the treatments for these within the ABI population? iii) which brain regions are impacted in individuals with an ABI presenting with person centred misidentification syndromes?

In this review, ABI was defined as a non-degenerative insult to the brain occurring after birth that results in disruption to normal brain function. This includes traumatic brain injury (arising from external force) and non-traumatic causes such as cerebrovascular events, anoxic injury, and infectious or inflammatory conditions (e.g., encephalitis). Congenital, neurodevelopmental, and progressive neurodegenerative conditions are excluded (Goldman et al., 2022).

This review also specifically focused on person-centred and self-related DMS as these phenomena provide a unique window into disturbances of identity, affective familiarity, and social cognition. In contrast, place-centred misidentifications are more closely associated with spatial and topographical processing (Darby & Prasad, 2016; Weinstein, 1994). Restricting the review in this way allowed for a more coherent examination of the cognitive and neural mechanisms underpinning identity-related misidentification.

Identifying Relevant Studies

The Preferred Reporting Items for Systematic Reviews and Meta-Analyses for Scoping Reviews (PRISMA-ScR) statement was adopted to guide the identification of relevant studies. Systematic searches were conducted across the databases PsycINFO, CINAHL, and MEDLINE from inception to February 2026, using the following keywords: “Capgras delusion” or “Capgras Syndrome”, or “Cotards Syndrome” or “Cotards delusion”, or “Fregoli syndrome” or “Fregoli delusion” or “Delusional misidentification syndrome” AND “Acquired Brain Injury” or “Traumatic brain injury” or “head injury” or “TBI” or “stroke” or “anoxic”. Hand-searches were also carried out across reference lists of relevant citations.

Any study published in English and enrolling an individual or a population presenting with person-centred misidentification syndromes (e.g., Capgras, Fregoli or Cotard’s delusion), or another DMS following an ABI was considered eligible for inclusion. No age or geographical limits were set. Studies that reported on an individual or population with a presentation that was not a DMS following ABI, systematic reviews, commentaries, conference proceedings, and letters were excluded. Included studies were also limited to publications in English due to feasibility constraints and the absence of resources for translation.

Study Selection

The initial search yielded 173 citations. The lead author screened the titles and abstracts for eligibility and removed duplicates manually. Two reviewers then screened 104 articles and then read 64 full texts and considered these for inclusion. In cases where abstracts and titles appeared to meet the inclusion criteria, but the full text version could not be retrieved, the authors were contacted. The rationale for any exclusions at full text review was recorded. In total, 34 articles were eventually included in the review, 33 of which were single case studies, and one reported on a case series from a population based observational study. Although qualitative studies were not excluded a priori, no eligible investigations adopting a qualitative methodology were identified by the searchers.

Of the included studies, 18 reported individuals with Capgras, three with Fregoli, six with Cotard's, two reported cases of Capgras with comorbid Cotard's, one reported on Capgras and reduplicative paramnesia, one reported on a case of Cotard's with comorbid dysmetropsia, one reported on a case of reduplicative paramnesia of the self, one reported on a case of intermetamorphosis and one reported on a case that progressed from Capgras and Fregoli to Cotard's syndrome all following an ABI. Figure 1 illustrates the PRISMA-ScR flow chart.

Data Charting

Data charting was undertaken to systematically extract and organise key information from the included studies. A standardised data charting form was developed in advance, informed by the review objective and refined iteratively during the initial stages of the review. Relevant data, including study characteristics, participant details, methodological features, and key findings were systematically entered into a structured table. This can also be viewed as a 'narrative review' of the data that were found (Arksey & O'Malley, 2005b; Pawson, 2002).

To enhance rigour, accuracy, and consistency, the data charting was conducted by one reviewer and independently reviewed by a second reviewer. Any discrepancies were discussed and resolved through consensus and the involvement of the third reviewer if required.

Quality Assessment

Since a formal quality appraisal is not typically performed in a scoping review (Pham et al., 2014), this was not included in the present review. However, efforts were made to highlight methodological and clinical limitations whenever present.

Results

A summary of the demographic characteristics, aetiologies, symptom duration, latency to onset, neuroanatomical findings, treatment approaches, and outcomes across included studies is presented in Table 2. The narrative synthesis below focuses primarily on the phenomenological features and clinically salient aspects of each DMS presentation. The full details of the included studies and their results are provided as Supplementary File (Table S1).

Clinical Manifestations

Capgras

Eighteen studies reported Capgras syndrome following ABI. Presentations occurred following traumatic, vascular, autoimmune, and infectious causes, with symptom onset ranging from the acute post-injury period to several years later and duration varying from transient episodes to chronic symptoms (Alexander et al., 1979; Ali, 2023; Ardila & Rosselli, 2018; Edelstyn et al., 2001; Fils & Stewart, 2011; Garcha et al., 2018; Gramling et al., 2024; Hirstein & Ramachandran, 1997; Johnstone et al., 2020; Jones et al., 2016; Koda et al., 2021; Lozano-Cuervo et al., 2020; Mattioli et al., 1999; Navarro, 2020; O'Connor et al., 1996; Pena-Salazar

et al., 2014; Staton et al., 1982; Weston & Whitlock, 1971). Key study characteristics are summarised in Table 2.

The phenomenology of Capgras syndrome most commonly involved the misidentification of close family members, although several reports described broader or atypical forms of misidentification. The core delusional belief across all cases was that the familiar person "looked identical but was not the same person", frequently described as an impostor, double, or replacement. Patients often emphasised a lack of emotional familiarity, stating that the person "felt different", "was not really my wife", or "was pretending to be someone else" (Fils & Stewart, 2011; Mattioli et al., 1999; Hirstein & Ramachandran, 1997). In several reports, patients demonstrated logical elaboration of the delusion, offering detailed explanations (e.g., impersonation for theft, substitution following death, conspiracies), consistent with intact reasoning processes operating on a false premise (Jones et al., 2016; O'Connor et al., 1996; Weston & Whitlock, 1971). Emotional responses varied widely. Some patients showed marked paranoia, agitation, or hostility toward the misidentified individual (Alexander et al., 1979; Gramling et al., 2024; Lozano-Cuervo et al., 2020), whereas others described the belief with relative emotional neutrality (Edelstyn et al., 2001; Navarro et al., 2020).

Capgras and Reduplicative Paramnesia

One case report (Neto et al., 2016) described concurrent Capgras syndrome and reduplicative paramnesia in the context of limbic encephalitis secondary to diffuse large B-cell lymphoma. The presentation involved progressive cognitive decline, behavioural change, temporal lobe seizure activity, and persistent delusional beliefs. Capgras phenomenology was evident in the belief that the patient's wife had been replaced by a perfect double, while reduplicative paramnesia involved the belief that his house and a major city avenue existed

simultaneously in two identical versions. The presentation emerged within a broader cognitive and behavioural syndrome rather than as an isolated psychiatric symptom.

Fregoli

Three studies reported Fregoli syndrome following TBI or stroke. Symptom duration varied considerably and ranged from approximately one month to over a year (Box et al., 1999; Feinberg et al., 1999; Kakegawa et al., 2020), although precise estimates were often difficult to establish due to limited follow-up and inconsistent reporting. Key study characteristics are summarised in Table 2.

Across studies, delusional content primarily involved misidentification of other people, typically reflecting hyperfamiliar misidentification. Box et al. (1999) described persistent misidentification of another inpatient as her mother, with behavioural enactment (e.g., repeatedly approaching the patient and attempting to get into bed with her). Feinberg et al. (1999) reported multiple Fregoli-type misidentifications involving familiar others (e.g., asserting that unfamiliar individuals were family members or acquaintances despite discrepancies) and included one self-misidentification within the enumerated series of misidentification events. Kakegawa et al. (2020) reported that the number of misidentified people ranged from one to dozens, and patients often misidentified staff or other patients as acquaintances or famous people and in some cases, misidentification also drew on auditory information (e.g., voices or coughing) as well as visual cues.

Emotional responses were variably described. Feinberg et al. (1999) noted periods of irritability during evaluation but do not provide a consistent affective response linked specifically to misidentification episodes and Box et al. (1999) described preoccupation with delusional ideas and readiness to act on them, but affective response to the misidentification itself was not consistently quantified.

Cotard's

Six case reports described Cotard's syndrome following traumatic, vascular, and seizure-related ABI (Biran, 2019; McCormick & Simberlund, 2020; Mendez, 2023; Ricardo et al., 2021; Torrisi et al., 2019; Young et al., 1992). Symptom duration ranged from days to several months, and latency to onset varied from the acute post-injury period to several months following ABI. Key characteristics are summarised in Table 2.

Across all cases, the core delusional content involved nihilistic beliefs about the self, most commonly the conviction of being dead, no longer existing, or having lost bodily integrity. In Young et al. (1992), the patient believed he had died and was in hell. Ricardo et al. (2021) described beliefs of being dead alongside confabulations, while Torrisi et al. (2019) reported delusions involving repeated death, rebirth, or loss of body parts. In Mendez (2023), the delusion was more focal, with the patient insisting that the left side of his body was dead, consistent with partial or somatic Cotard's phenomenology.

Unlike Capgras or Fregoli syndromes, delusions were self-referential in all cases, rather than centred on misidentification of others. Emotional responses varied across cases. Several patients exhibited marked distress, agitation, fear, or behavioural dysregulation, particularly when delusional beliefs were challenged (Torrisi et al., 2019; Ricardo et al., 2021). Others demonstrated emotional blunting or reduced affective reactivity, reporting nihilistic beliefs with limited overt distress (Young et al., 1992).

Comorbid Capgras and Cotard's

Two case reports described comorbid Capgras and Cotard's syndromes following severe ABI (Butler, 2000; Sottile et al., 2015; see Table 2 for key characteristics). Phenomenologically, the Capgras component involved paranoid misidentification of close family in both cases. In Butler (2000), the patient repeatedly examined his father's face and

accused him of being a “criminal” double who had taken his father’s place. In Sottile et al. (2015), psychological/psychiatric evaluation identified paranoia towards family attributable to Capgras syndrome.

The Cotard’s component was self-referential nihilistic belief. Butler (2000) reports repeated statements that the patient reported that he was dead and detained in Hell, alongside intense depersonalisation/derealisation and incorporation of nightmare material into delusional narrative. Sottile et al. (2015) describe Cotard’s phenomenology as the patient experiencing the smell of her own decomposition. Emotional response was prominent in both cases. Butler (2000) documents morning presentations of perplexity and fear, agitation/restlessness, intermittent hostility, and accusatory persecutory themes. Sottile et al. (2015) report the patient was afraid to sleep and showed a paranoid attitude towards family.

Cotard’s and Dysmetropsia

One case report (Parks et al., 2014) described transient Cotard’s syndrome occurring alongside dysmetropsia during a prolonged migraine aura. The patient experienced persistent visual perceptual disturbance over two weeks and a brief delusion of being dead lasting approximately 60 minutes.

Reduplicative Paramnesia of the Self

One case report (Dasarathy et al., 2009) described reduplicative paramnesia of the self following ABI in a patient with a pre-existing diagnosis of schizophrenia. The patient developed the belief that another person looked exactly like her and could be mistaken for her, with distress focused on the perceived double taking her place.

Intermetamorphosis

One case report (Silva et al., 1993) described intermetamorphosis following gunshot-related ABI. The patient developed paranoid misidentification beliefs involving perceived

physical and psychological transformation of others, including the belief that his wife was an alien. This was associated with significant risk, including threats toward his wife.

Progression from Capgras and Fregoli to Cotard's syndrome

One longitudinal case report (Pąchalska, 2019) described progression from Capgras- and Fregoli-type misidentification phenomena to Cotard's syndrome over several years following severe ABI. The case was characterised by profound autobiographical memory and identity disturbance, followed by increasing misidentification phenomena and later nihilistic beliefs.

Neuroanatomical Bases

Capgras

All studies attempted to investigate potential neural correlates of Capgras syndrome using structural or functional imaging, although implicated regions varied. Among the 18 Capgras cases, frontal lobe involvement was identified in 14 cases (78%), most commonly affecting bilateral fronto-basal, orbito-frontal, or dorso-lateral prefrontal regions.

Temporal lobe involvement was reported in 12 cases (67%), most frequently involving the right temporal lobe or temporoparietal junction. This included right temporal and temporoparietal damage (Alexander et al., 1979; Staton et al., 1982), right occipito-temporal infarction (Navarro et al., 2020), right antero-temporal and temporal pole involvement (Jones et al., 2016), and limbic-temporal network abnormalities (Hirstein & Ramachandran, 1997; Lozano-Cuervo et al., 2020).

Parietal regions were implicated in several cases, including right temporoparietal contusions (O'Connor et al., 1996), parieto-occipital junction involvement (Staton et al., 1982), and posterior cortical hypometabolism (Lozano-Cuervo et al., 2020). Subcortical structures were highlighted in multiple cases, including left thalamic lacunar infarction with frontal

hypoperfusion (Peña-Salazar et al., 2014), basal ganglia involvement (Edelstyn et al., 2001), and moyamoya-related frontal hypoperfusion (Koda et al., 2021).

Overall, these findings suggest that Capgras syndrome following ABI most commonly arises from disruption to right-lateralised frontal-temporal networks, often with parietal or subcortical contributions, consistent with models emphasising impaired integration between face recognition and affective familiarity systems (Darby et al., 2017).

Capgras and Reduplicative Paramnesia

Structural and functional neuroimaging revealed abnormalities consistent with limbic system involvement. Brain MRI demonstrated T2/FLAIR hyperintensities in the left temporal pole and left mesial temporal region, while FDG-PET imaging showed hypermetabolism in the left mesial temporal and peri-insular cortex, alongside severe and diffuse hypometabolism in frontal, parietal, and lateral temporal cortices (Neto et al., 2016). Electroencephalography revealed diffuse slow-wave activity without epileptiform discharges. The pattern of imaging findings was consistent with disruption of frontal-temporal-limbic networks.

Fregoli

Across the three papers, frontal-temporal systems were implicated, though the nature of evidence differed. Feinberg et al. (1999) reported clear structural injury with a large right frontal contusion and smaller left temporoparietal contusions with oedema/encephalomalacia. Box et al. (1999) reported pathology involving the right ventro-medial and polar frontal cortex, with some involvement of the left ventro-medial and right dorso-lateral regions, alongside a small infarct in the right parietal corona radiata, following severe head injury and neurosurgery. Kakegawa et al. (2020) identified right hemispheric lesions in all 10 stroke cases, and voxel-based lesion-symptom mapping showed overlapping lesion sites associated with Fregoli

syndrome involving the right insula, inferior frontal lobe, anterior temporal lobe, and subcortical limbic system, described as areas connected by the uncinate fasciculus.

Cotard's

Across all six cases, Cotard's syndrome was associated with predominantly right-hemisphere pathology, frequently involving fronto-temporo-parietal networks. Young et al. (1992) described contusions affecting the right temporo-parietal cortex, with additional bilateral frontal involvement. Torrisi et al. (2019) reported lesions involving the right temporal lobe, insula, and basal ganglia, with functional imaging demonstrating disrupted connectivity in right frontal, parietal, and insular regions. Ricardo et al. (2021) similarly identified right frontal, parietal, and occipital lesions following subarachnoid haemorrhage and delayed cerebral ischaemia.

Subcortical involvement was noted in several cases. Torrisi et al. (2019) and Ricardo et al. (2021) implicated basal ganglia and internal capsule structures, while Mendez (2023) demonstrated right-hemisphere parietal, occipital, posterior frontal, and posterior temporal involvement with corresponding EEG abnormalities and FDG-PET hypermetabolism consistent with seizure-related dysfunction. Although McCormick et al. (2020) did not report neuroimaging, prominent executive and attentional deficits were described, suggesting potential frontal network involvement.

Comorbid Capgras and Cotard's

Neuroanatomical correlates were described in both papers. Butler (2000) reports initial CT findings of haematomas in the right thalamus and left basal ganglia, bilateral intraventricular haemorrhage, and pituitary stalk shearing injury; EEG later showed mild diffuse slowing without focal features. Sottile et al. (2015) report MRI evidence of extensive temporoparietal ischaemia, with subsequent CT showing a large hypodense area in the

temporoparietal cortex. The authors interpret the presentation using a disconnection account, describing Capgras as involving disconnection between face recognition systems and limbic processing, and Cotard's as broader disconnection between sensory areas and limbic systems.

Cotard's and Dysmetropsia

Neuroimaging two weeks after symptom onset suggested right temporal–parietal–occipital cortical involvement, with changes interpreted as consistent with cortical infarction/cortical laminar necrosis. However, a follow-up MR scan 18 months later showed no evidence of prior stroke, raising uncertainty about whether the episode represented true migrainous infarction versus transient aura-related imaging changes.

Reduplicative Paramnesia of the Self

Injury description emphasised bilateral frontal contusions (contra-coup) and the clinical impression of frontal lobe dysfunction, consistent with the authors' framing of reduplicative phenomena in the context of ABI superimposed on psychiatric vulnerability (Dasarathy et al., (2009).

Intermetamorphosis

Head CT demonstrated brain loss of the right frontal lobe and right lateral ventricular enlargement, with prominent sulci bilaterally. Neuropsychological testing showed severe impairment on the Benton Facial Recognition Test and deficits on NCSE subtests (construction and memory), supporting right hemisphere pathology and facial-recognition processing difficulties associated with misidentification (Silva et al., 1993).

Treatment Approaches

Capgras

Treatment details were reported in 11 studies, of which 10 employed pharmacological approaches (Ardila & Rosselli, 2018; Edelstyn et al., 2001; Fils & Stewart, 2011; Gramling et al., 2024; Koda et al., 2021; Mattioli et al., 1999; O'Connor et al., 1996; Peña-Salazar et al., 2014; Ali et al., 2023; Lozano-Cuervo et al., 2020). Typical and atypical antipsychotics (e.g., risperidone, haloperidol, quetiapine, olanzapine, oxypertine) were most frequently used. Adjunctive treatments included SSRIs and SARIs (e.g., sertraline, trazodone), benzodiazepines (e.g., clonazepam), beta-blockers (propranolol), and immunotherapy in autoimmune cases. One study reported resolution following stroke management alone without psychiatric medication (Garcha et al., 2018).

Treatment outcomes varied widely. Complete remission occurred in three cases (17%), including resolution following quetiapine (Koda et al., 2021), stroke management (Garcha et al., 2018), and immunotherapy for autoimmune encephalitis (Lozano-Cuervo et al., 2020). Partial improvement was reported in approximately 39% of cases, while persistent or chronic symptoms were described in 44%, either despite treatment or in the absence of intervention (Alexander et al., 1979; Staton et al., 1982; Jones et al., 2016; Johnstone et al., 2020; Weston & Whitlock, 1971). Overall, outcomes highlight marked heterogeneity and a substantial risk of chronicity following ABI-associated Capgras syndrome.

Capgras and Reduplicative Paramnesia

Despite immunotherapy and multiple psychotropic medications, no meaningful improvement in cognitive, behavioural, or delusional symptoms was reported (Neto et al., 2016).

Fregoli

Treatment reporting varied. Box et al. (1999) reported commencement of risperidone (6 mg daily) and later olanzapine (5 mg daily) during the period when the misidentification belief was present; the Fregoli belief subsided over approximately one month and there was no evidence of delusional beliefs by three months post-injury. Feinberg et al. (1999) did not report a specific treatment for the misidentification symptoms, and outcome was described in terms of presence/recurrence of misidentification events during admission rather than clear resolution. Kakegawa et al. (2020) monitored clinical features and duration during inpatient rehabilitation but did not report specific psychiatric treatment for Fregoli and persistence was described as at least 1.5 months (up to 17 months during observation).

Cotard's

Treatment details were inconsistently reported. Pharmacological interventions were described in four studies. Torrisi et al. (2019) reported resolution of Cotard-like delusions following treatment with risperidone and escitalopram, while Ricardo et al. (2021) described partial improvement with clozapine and valproate, although confabulation and anosognosia persisted. Mendez (2023) reported gradual resolution of Cotard's symptoms with antiepileptic medication (levetiracetam and brivaracetam), without primary antipsychotic treatment, highlighting seizure control as the principal intervention. Young et al. (1992) did not report specific psychiatric pharmacological treatment, with symptoms resolving gradually alongside neurological recovery and rehabilitation. Two studies did not provide sufficient detail regarding treatment or response (McCormick et al., 2020; Biran et al., 2019). Overall, outcomes ranged from complete remission to partial or fluctuating improvement, underscoring the heterogeneity of Cotard's syndrome following ABI and the absence of standardised treatment approaches.

Comorbid Capgras and Cotard's

Both reported cases (Sottile et al, 2015; Butler, 2000) responded favourably to treatment, with substantial reduction or complete resolution of delusional symptoms following pharmacological and psychological intervention.

Cotard's and Dysmetropsia

Management focused on vascular risk reduction and aspirin therapy. Visual symptoms improved gradually and no psychiatric treatment for Cotard's symptoms was reported (Parkes, et al., 2014).

Reduplicative Paramnesia of the Self

Treatment involved depot antipsychotic medication and behavioural improvement was reported, although the specific outcome of the reduplicative delusion remained unclear. (Dasarathy et al., 2009).

Intermetamorphosis

Antipsychotic treatment was associated with reduction in psychotic symptoms and no recurrence of the misidentification delusion (Silva et al., 1993).

Progression from Capgras and Fregoli to Cotard's

Management consisted primarily of long-term neuropsychological rehabilitation. Despite ongoing intervention, misidentification and nihilistic symptoms were reported to worsen over time (Pąchalska, 2019).

Discussion

This scoping review synthesised the sparse but growing literature on delusional misidentification syndromes (DMS) following ABI. The review followed Joanna Briggs Institute guidance and PRISMA-ScR-informed identification procedures to map available

evidence on delusional misidentification phenomena arising after ABI. Database searches (PsycINFO, CINAHL, and MEDLINE) from inception to February 2026 identified 173 records. After removal of duplicates, 104 records were screened, 75 reports were retrieved, and 64 full-text reports were assessed for eligibility, resulting in 34 included studies. The evidence base was overwhelmingly comprised of single-case reports, with one observational, population-based stroke study describing multiple cases of Fregoli syndrome (Kakegawa et al., 2020). Eighteen studies reported Capgras syndrome as the primary DMS following ABI, one study reported Capgras with concurrent reduplicative paramnesia (Neto et al., 2016), three studies reported Fregoli syndrome (two single-case reports and one stroke cohort study; Box et al., 1999; Feinberg et al., 1999; Kakegawa et al., 2020), six studies reported Cotard's syndrome as the primary presentation (Biran, 2019; McCormick & Simberlund, 2020; Mendez, 2023; Ricardo et al., 2021; Torrisi et al., 2019; Young et al., 1992), and two studies reported comorbid Capgras and Cotard's delusions (Butler, 2000; Sottile et al., 2015). The remaining reports comprised rarer DMS variants and trajectories: Cotard's with dysmetropsia (Parks, 2014), reduplicative paramnesia of the self (Dasarathy et al., 2009), intermetamorphosis (Silva et al., 1993), and longitudinal progression from Capgras and Fregoli to Cotard's syndrome (Pąchalska, 2019). Collectively, the findings suggest that DMS remain uncommon after ABI and may be under-recognised in clinical settings or inconsistently labelled across disciplines.

Across included studies, DMS following ABI showed marked heterogeneity in aetiology, clinical phenomenology, neuroanatomical correlates, and outcome. Capgras syndrome was most frequently reported and typically involved misidentification of close family members following traumatic or vascular injury (e.g., Alexander et al., 1979; Weston & Whitlock, 1971; Staton et al., 1982; O'Connor et al., 1996; Mattioli et al., 1999; Fils & Stewart, 2011; Jones et al., 2016; Garcha et al., 2018; Koda et al., 2021; Navarro et al., 2020; Ali et al., 2023), but was also described following autoimmune encephalitis (Lozano-Cuervo et al., 2020)

and neurocysticercosis (Ardila & Rosselli, 2018). Onset ranged from acute presentation to months or years post-injury, and duration varied from transient to chronic. Treatments were inconsistently reported and predominantly pharmacological (most commonly antipsychotics), with outcomes ranging from complete remission to persistence over years. These findings broadly reinforce the clinical impression that DMS after ABI are not uniform syndromes with a single aetiology, but rather striking, content-specific delusions that can arise in multiple neurological contexts.

Several hypotheses have been proposed to account for Capgras delusion formation, emphasising disruption to mechanisms supporting familiarity, memory integration, and belief evaluation. One influential account suggests disconnection between memory-related systems limits the integration of new information with stored representations, contributing to misidentification phenomena (Mattioli et al., 1999; Staton et al., 1982). Another proposes that disconnection involving the right temporal lobe and limbic or frontal systems contributes to DMS (Alexander et al., 1979). Moscovitch & Melo (1997) similarly argue that bilateral frontobasal lesions may disrupt retrieval mechanisms relevant to recognition and belief formation. The Capgras cases included in this review align with these models insofar as frontal involvement was commonly described and temporal involvement, often right-sided, was frequently implicated (e.g., Alexander et al., 1979; Jones et al., 2016; Mattioli et al., 1999; O'Connor et al., 1996; Staton et al., 1982; Navarro et al., 2020; Lozano-Cuervo et al., 2020). However, the diversity of reported lesion sites and mechanisms (including vascular, inflammatory, infectious, and traumatic contexts) supports a network-level interpretation rather than a single focal localisation.

Fregoli syndrome was represented by four studies, including three single-case reports following TBI (Box et al., 1999; Duggal, 2004; Feinberg et al., 1999) and one observational

stroke study reporting ten cases within a rehabilitation cohort (Kakegawa et al., 2020). Across studies, Fregoli delusions were characterised by hyperfamiliar misidentification (often of staff, other patients, or acquaintances), sometimes drawing on multi-modal cues (e.g., voice) in addition to visual information. Neuroanatomically, the stroke cohort provides the most methodologically informative evidence in the ABI literature to date, reporting consistent right-hemisphere involvement across cases and lesion overlap implicating right insula, inferior frontal lobe, anterior temporal lobe, and subcortical limbic structures. These are areas described as interconnected by the uncinate fasciculus (Kakegawa et al., 2020). The single-case reports also implicated fronto-temporal systems but varied in whether evidence was structural (Feinberg et al., 1999; Box et al., 1999) or functional/epileptiform (Duggal, 2004). These findings strengthen the plausibility of accounts emphasising impaired reality monitoring and abnormal familiarity attribution within fronto-temporo-limbic networks (Devinsky, 2009), while also indicating that Fregoli syndrome is not restricted to TBI in ABI populations.

Cotard's syndrome was reported as the primary presentation in six single-case studies spanning traumatic, vascular, and seizure-related aetiologies (Young et al., 1992; Biran, 2019; Torrisi et al., 2019; Ricardo et al., 2021; McCormick & Simberlund, 2020; Mendez et al., 2023). In these cases, nihilistic beliefs were self-referential and often accompanied by broader cognitive impairment or neuropsychiatric features (e.g., executive dysfunction, attentional disturbance), with outcomes ranging from rapid resolution to persistence alongside residual deficits. Across cases with imaging, right-hemisphere fronto-temporo-parietal and insular/subcortical involvement was prominent, and functional connectivity findings further supported distributed network disruption rather than focal localisation (Torrisi et al., 2019). Importantly, Cotard's phenomenology also appeared in additional included reports outside the Cotard-only subgroup: transient Cotard's during a syndrome of perceptual disturbance (Parks et al., 2014), co-occurring Capgras and Cotard's following ABI (Butler, 2000; Sottile et al.,

2015), and longitudinal evolution toward Cotard's in a long-term post-TBI trajectory with prior misidentification phenomena (Pąchalska, 2019). These additional papers reinforce the view that nihilistic delusions can occur both as an isolated phenomenon and within broader misidentification syndromes or evolving neuropsychiatric profiles.

Beyond Capgras, Fregoli, and Cotard's, this review identified rare but clinically salient DMS variants following ABI. Intermetamorphosis was reported after a gunshot head injury with right frontal tissue loss and severe facial recognition impairment and was associated with high-risk behaviour toward a misidentified partner (Silva et al., 1993). Reduplicative paramnesia was reported both in isolation (Dasarathy et al., 2009) and concurrently with Capgras in the context of limbic encephalitis secondary to lymphoma (Neto et al., 2016), supporting the clinical reality that misidentification phenomena can extend beyond persons to places and aspects of self. The longitudinal case describing progression from Capgras and Fregoli phenomena to Cotard's syndrome over decades (Pąchalska, 2019) further supports a spectrum conceptualisation, in which vulnerability within belief-evaluation and familiarity/self-representation systems may manifest in different DMS subtypes across time and context.

Taken together, findings across DMS following ABI indicate overlap in implicated systems despite divergent phenomenology. Across syndromes, frontal involvement was common, consistent with proposals that impaired executive control and belief evaluation contribute to the maintenance of delusional beliefs (Coltheart, 2010). Right-hemisphere network involvement, particularly fronto-temporo-insular and temporo-parietal regions, was repeatedly observed across Capgras, Fregoli, and Cotard's presentations, consistent with broader accounts emphasising right-hemisphere contributions to familiarity processing, self-referential integration, and reality monitoring (Darby & Prasad, 2016). While specific

mechanisms differ across cases (e.g., post-traumatic structural lesions, seizure-related dysfunction, inflammatory limbic involvement, vascular disconnection), the overall pattern supports interpretation of post-ABI DMS as arising from network-level disruption rather than a single lesion site.

Finally, the wide variation in latency between ABI and symptom onset highlights the importance of longitudinal follow-up. Delayed emergence, particularly in chronic or evolving neurological contexts, risks misattribution to primary psychiatric illness unless ABI history, neurocognitive profile, and neurological investigations are integrated into formulation and diagnostic reasoning.

Clinical Implications

Clinically, these findings support the need for ongoing neuropsychiatric monitoring following ABI, including beyond the acute rehabilitation window. DMS may emerge weeks, months, or years after injury, and presentations can include not only Capgras, Fregoli, and Cotard's syndromes, but rarer variants such as intermetamorphosis, and reduplicative paramnesia. Improved awareness of this broader spectrum may enhance diagnostic specificity and reduce the risk of inappropriate psychiatric labelling when delusions are content-specific and temporally linked to neurological insult.

The review also highlights the importance of multidisciplinary assessment when psychotic symptoms arise post-ABI. Given that several included reports linked DMS to identifiable neurological mechanisms (e.g., vascular lesions, seizure-related dysfunction, limbic encephalitis, infection), a combined approach incorporating neurological evaluation, neuroimaging, and neuropsychological assessment is likely to improve formulation and guide management. This is particularly relevant where delirium, mood disorder with psychotic features, dementia, or primary psychosis are plausible differentials. Enhanced interdisciplinary

communication is essential to ensure that delusional misidentification syndromes are not misattributed solely to primary psychiatric conditions, particularly in the context of neurological injury. In addition, clear and ongoing communication with families and caregivers is important to support understanding of the condition and to facilitate effective behavioural management strategies in everyday settings.

From a risk and safeguarding perspective, several DMS presentations involved close relatives and/or paranoia and behavioural enactment, and at least one intermetamorphosis report described explicit intent to harm a misidentified partner (Silva et al., 1993). Services should therefore consider carer support, safety planning, and risk assessment as integral components of care when DMS content involves family members or is associated with agitation, hostility, or command hallucinations.

Strengths and Limitations

A key strength of this review lies in its use of a scoping review methodology to synthesise a rare, heterogeneous, and methodologically limited evidence base. DMS following ABI are uncommon, clinically complex, and unlikely to be captured in large-scale studies. As such, the inclusion of single-case reports is not a weakness per se but a necessity, allowing detailed examination of phenomenology, neuroanatomical correlates, temporal course, treatment attempts, and outcomes that would otherwise remain undocumented. By systematically collating 34 studies across a broad spectrum of DMS presentations, including Capgras, Fregoli, Cotard's, mixed and longitudinal presentations, and rarer variants such as intermetamorphosis and reverse subjective double, this review provides the most comprehensive mapping of post-ABI DMS to date.

An additional strength is the structured differentiation between primary DMS presentations and mixed or evolving syndromes. Rather than collapsing all misidentification

phenomena into a single category, the review preserves phenomenological distinctions while also demonstrating overlap at the level of neural systems and cognitive mechanisms. The inclusion of a population-based observational stroke study using lesion-symptom mapping represents a notable methodological advance within this literature, strengthening inferences about right-hemisphere frontal-temporal-insular network involvement beyond what is possible from isolated case reports alone.

Nevertheless, several limitations must be acknowledged. First, the overwhelming reliance on single-case designs limits generalisability and precludes statistical synthesis or causal inference. Case reports are inherently vulnerable to selection bias, inconsistent reporting, and subjective interpretation of symptoms, particularly when diagnostic frameworks evolve over time or differ across disciplines. Second, heterogeneity in how DMS were defined and operationalised across studies complicates direct comparison. Some reports provided rich phenomenological detail and systematic assessment, while others relied on narrative description without formal psychiatric or neuropsychological evaluation, increasing the risk of misclassification.

Third, neuroimaging and cognitive data were inconsistently reported. Although many studies identified frontal and right-hemisphere involvement, variation in imaging modality, timing, and analytical approach limited precise localisation or network-level comparison across cases. Similarly, neuropsychological assessment was often partial or absent, despite frequent reference to executive dysfunction, attentional impairment, or face-recognition difficulties. Fourth, treatment reporting was uneven and largely descriptive, with limited information on dosing, duration, adherence, or long-term outcome, restricting conclusions about therapeutic efficacy. Finally, delayed onset and fluctuating symptom trajectories raise the possibility that some DMS following ABI remain unrecognised or underreported, particularly when symptoms

emerge outside acute or rehabilitation settings. Finally, the restriction to English-language studies may have introduced language bias and limited the inclusion of relevant international literature.

Future Research

Future research on DMS following ABI would benefit from greater methodological consistency and systematic investigation. Establishing clearer operational definitions of DMS subtypes, alongside explicit differentiation from delirium, affective psychosis, neurodegenerative conditions, and primary psychiatric disorders, would improve diagnostic clarity and facilitate comparison across studies. Given the content-specific nature of these delusions, careful phenomenological description should remain central to future reporting.

Standardised documentation of key clinical variables, including latency to onset, duration of symptoms, affective response, behavioural enactment, risk to self or others, and functional impact, would substantially strengthen the evidence base. The frequent co-occurrence of executive dysfunction, attentional impairment, and impaired insight across cases suggests that comprehensive neuropsychological assessment should be considered essential rather than optional in future reports, particularly where belief evaluation or familiarity processing is implicated.

There is also a clear need for research designs that extend beyond single-case descriptions. The inclusion of a stroke cohort study employing lesion–symptom mapping demonstrates the value of larger samples and network-based analytical approaches in elucidating shared and syndrome-specific neural mechanisms. Prospective case series, multicentre registries, and collaborative databases could enable examination of variability in presentation, treatment response, and longitudinal outcome, while accommodating the rarity of these syndromes. The adoption of qualitative methods would also be beneficial to explore

people's subjective experiences of being affected by DMS following ABI. Finally, treatment research remains underdeveloped. Future work should aim to document intervention strategies more systematically, including pharmacological, neurological, psychological, and rehabilitative approaches, with attention to both short- and long-term outcomes. Such efforts would support the development of evidence-informed clinical guidance for managing these complex and often distressing post-ABI presentations.

Conclusion

DMS following ABI represent rare but clinically significant disturbances in familiarity, identity, and belief evaluation. This scoping review identified 34 studies, largely single-case reports, describing Capgras syndrome as the most frequently reported primary presentation, alongside smaller bodies of evidence for Fregoli and Cotard's syndromes and a range of rarer DMS variants (including intermetamorphosis, reverse subjective double, and reduplicative paramnesia). Across reports, implicated mechanisms converged on disruption to frontal and right-hemisphere fronto-temporo-insular and temporo-parietal systems, supporting models that conceptualise DMS as network-level disorders of familiarity attribution and belief evaluation rather than outcomes of single focal lesions. Recognising the breadth of post-ABI DMS phenomenology may improve diagnostic accuracy, inform safeguarding and carer support, and guide future research toward more systematic, mechanistically informed investigations.

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Tables

Table 1

Core phenomenological and distinguishing features of delusional misidentification syndromes

Syndrome	Core Belief	Target of Misidentification	Typical Emotional Response	Distinguishing Features
Capgras Syndrome	A familiar person has been replaced by an identical impostor	Others (usually close family member)	Variable: paranoia, fear, hostility, or neutral affect	Preserved perceptual recognition but loss of emotional familiarity - often elaborated explanations (e.g., conspiracy, replacement)
Fregoli Syndrome	Different people are actually a single known person in disguise	Other (strangers misidentified as familiar individuals)	Variable - may include suspicion or persecutory beliefs	Hyperfamiliarity - misidentification often extends to multiple individuals and may involve multimodal cues (e.g., voice)
Cotard's Syndrome	The self is dead, does not exist, or has lost bodily integrity	Self	Often distress, nihilism, or emotional blunting	Self-referential delusion - frequently associated with depersonalisation and derealisation
Intermetamorphosis	People transform into other known individuals while retaining physical appearance	Others	Often paranoia or threat-related affect	Misidentification involves perceived transformation of identity, personality, and intent
Subjective Doubles	A duplicate of the self exists as a separate individual	Self (externalised double)	Variable - may include paranoia or hostility toward the double	Double is perceived as physically identical but psychologically distinct

Reverse Subjective Doubles	The self is duplicated or transformed into another identity	Self	Often associated with disturbed agency or identity	Misidentification directed inward - disruption of self-boundaries
Reduplicative Paramnesia (self-related)	A place or self is duplicated and exists in multiple locations or forms (for this review only self-duplication was included)	Self or environment	Variable	Often co-occurs with other DMS - linked to spatial and contextual disorientation

Table 2

Summary of clinical, neuroanatomical, and treatment-related findings across DMS following acquired brain injury

Domain	Capgras	Fregoli	Cotard's	Other DMS Variants
Typical Aetiology	Predominantly TBI and cerebrovascular events. Also autoimmune and infectious causes	TBI and stroke	TBI, stroke, seizure-related conditions	Heterogeneous (TBI, stroke, rare neurological conditions)
Phenomenology	Hypofamiliar misidentification (loss of emotional familiarity)	Hyperfamiliar misidentification (false familiarity)	Nihilistic self-referential delusion	Mixed or atypical misidentification involving self/others
Target of Delusion	Familiar others (e.g., spouse, parent)	Strangers misidentified as familiar individuals	Self	Variable (self, others, environment)
Cognitive Profile	Often relatively preserved global cognition with deficits inferred in familiarity processing and belief evaluation	Variable - may include confabulation or disorganisation	Variable - may include executive and attentional deficits	Poorly characterised - often inferred rather than formally assessed
Executive Function / Belief Evaluation	Frequently inferred impairment in belief evaluation systems - rarely formally assessed	Limited formal assessment - often inferred from behaviour	Suggested impairment in belief evaluation and self-referential processing	Sparse reporting - largely inferred
Neuroanatomical Correlates	Predominantly right frontal and temporal involvement. Frequent fronto-temporo-parietal network disruption	Right hemisphere predominance - insula, frontal, temporal, and limbic regions implicated	Right-hemisphere fronto-temporo-parietal networks and subcortical involvement common	Variable but broadly consistent with right-hemisphere and network-level disruption

Time to Onset	Highly variable (acute to years post-injury)	Variable (acute to delayed but sometimes in rehabilitation phase)	Typically acute to subacute post-injury	Variable
Duration	Ranges from transient to chronic persistence	Often transient but may persist	Variable - may resolve or persist	Poorly characterised
Pharmacological Treatment	Commonly antipsychotics (e.g., risperidone, olanzapine) with adjunctive medications used	Antipsychotics reported in some cases	Antipsychotics, antidepressants, or antiepileptics depending on aetiology	Variable - no consistent approach
Non-Pharmacological Approaches	Rarely reported - includes reassurance, environmental modification, caregiver strategies	Limited reporting	Limited reporting	Very limited evidence
Outcomes	Mixed - remission, partial improvement, or chronic symptoms	Often improvement or resolution, though persistence reported	Mixed outcomes with some complete resolution reported	Highly variable
Key Interpretation	Disrupted integration of perceptual recognition and affective familiarity	Aberrant assignment of familiarity to unfamiliar stimuli	Disruption of self-representation and belief systems	Reflect broader disruption of identity and belief networks
Neuropsychological tests	Heterogeneous - included executive function, memory, attention, and facial recognition measures where reported.	Sparse reporting - primarily clinical observation and neuropsychological assessment.	Variable - included executive, attentional, and broader cognitive assessment.	Limited and heterogeneous - included facial recognition and executive function measures in isolated cases.

DMS = Delusional misidentification syndrome; TBI = Traumatic brain injury;

Figures

Figure 1

Prisma-Scr Flow Diagram