Review Article

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Capgras syndrome in clinical settings: an integrative review of case reports

Síndrome de Capgras em contextos clínicos: uma revisão integrativa de relatos de caso

El síndrome de Capgras en contextos clínicos: una revisión integradora de reportes de caso

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ABSTRACT:
Introduction: Delusional identification syndromes represent changes that can significantly compromise the ability to recognize familiar people, objects, or places. **Objective:** To analyze, through case reports, the therapeutic strategies employed in the management of delusional identification syndromes, with an emphasis on Capgras Syndrome, the explanatory hypotheses adopted, the associated etiological and diagnostic contexts, as well as the complementary methods used to characterize the disorder. **Method:** This is an integrative review of 11 clinical case reports, evaluated according to CARE guidelines. **Results:** The etiology was multifactorial (psychotic, neurodegenerative, traumatic). Explanatory hypotheses were based on neurocognitive and psychodynamic models. Therapeutic management was guided by the etiology, combining pharmacotherapy (antipsychotics, with clozapine in treatment-resistant cases) and psychosocial interventions. Diagnostic investigation required a multimodal approach, including neuroimaging, toxicological tests, and clinical scales. **Conclusion:** Effective management requires an integrated approach, targeting the underlying cause, combining specific treatment, psychosocial support, and ongoing investigation.

Keywords: mental disorders, mental health, Capgras syndrome

RESUMO:
Introdução: As síndromes de identificação delirante comprometem de maneira significativa a capacidade de reconhecimento de pessoas, objetos ou lugares familiares. **Objetivo:** Analisar, por meio de relatos de caso, as estratégias terapêuticas empregadas no manejo das síndromes de identificação delirante, com ênfase na Síndrome de Capgras, as hipóteses explicativas adotadas, os contextos etiológicos e diagnósticos associados, bem como os métodos complementares utilizados na caracterização do transtorno. **Método:** Trata-se de uma revisão integrativa de 11 relatos de caso, avaliados segundo as diretrizes CARE. **Resultados:** A etiologia foi multifatorial (psicótica, neurodegenerativa, traumática). As hipóteses explicativas pautaram-se em modelos neurocognitivos e psicodinâmicos. O manejo terapêutico foi orientado pela etiologia, combinando farmacoterapia (antipsicóticos, com clozapina em casos resistentes) e intervenções psicossociais. A investigação diagnóstica exigiu uma abordagem multimodal, incluindo neuroimagem, exames toxicológicos e escalas clínicas. **Conclusão:** O manejo eficaz requer uma abordagem integrada, direcionada à causa subjacente, associando tratamento específico, suporte psicossocial e investigação contínua.

Palavras-chave: transtornos mentais, saúde mental, síndrome de Capgras

RESUMEN:
Introducción: Los síndromes de identificación delirante afectan de manera significativa a la capacidad de reconocer a personas, objetos o lugares familiares. **Objetivo:** Analizar, a través de reportes de casos, las estrategias terapéuticas empleadas en el abordaje de los síndromes de identificación delirante, con énfasis en el síndrome de Capgras, las hipótesis explicativas, los contextos etiológicos y diagnósticos asociados, así como los métodos complementarios utilizados en la caracterización del trastorno. **Método:** Se trata de una revisión integrativa de 11 reportes de casos, evaluados según las directrices CARE. **Resultados:** La etiología fue multifactorial (psicótica, neurodegenerativa, traumática). Las hipótesis explicativas se basaron en modelos neurocognitivos y psicodinámicos. El manejo terapéutico se orientó según la etiología, combinando farmacoterapia (antipsicóticos, con clozapina en casos resistentes) e intervenciones psicosociales. La investigación diagnóstica requirió un enfoque multimodal, que incluyó neuroimagen, pruebas toxicológicas y escalas clínicas. **Conclusión:** El manejo eficaz requiere un enfoque integrado, dirigido a la causa subyacente, que combine tratamiento específico, apoyo psicosocial e investigación continua.

Palabras clave: trastornos mentales, salud mental, síndrome de Capgras

Introduction

Delusional identification syndromes (DIS) comprise a set of neuropsychiatric phenomena of multifactorial origin, which can manifest themselves in different ways [1]. They are predominantly characterized by disturbances in the ability to recognize familiar places, people, or objects [2].

It is possible for two or more syndromes to coexist, alternate with each other, or manifest at different times throughout an individual's life [3]. Although often associated with mental and/or neurological disorders, such as schizophrenia and dementia, these syndromes can also occur acutely, in the absence of obvious signs or prior diagnosis [4].

Among DISs, the most prominent manifestations are Capgras, Fregoli, and Intermetamorphosis delusions [5]. Capgras syndrome (CS), in particular, stands out as a disorder in which the individual believes that close people

or even themselves have been replaced by identical impostors [6]. Its initial description dates back to 1923, by psychiatrists Joseph Capgras and Jean Reboul-Lachaux, who reported the case of a woman convinced that her husband and other close people had been replaced by lookalikes [7].

CS is often associated with schizophrenia, which is the most commonly related mental disorder, but it can also occur in neurological conditions such as dementia, especially Alzheimer's disease [8]. Patients with the syndrome express an unshakeable conviction that their spouses, family members, or friends have been replaced by an identical impostor [9]. In certain cases, this delusional belief can extend to animals, objects, and even the person themselves, a condition called reverse Capgras, in which the individual believes that they themselves are an impostor [10].

Generally, DISs are investigated in isolation; however, recent evidence indicates their possible coexistence in the same individual [1]. The aggregate analysis of reports and case series published in the specialized literature points to a possible association between DISs and changes in the right hemisphere [11].

In fact, neuroimaging studies and reports of lesions have suggested the involvement of this hemisphere in DISs [12]. Investigations in this area reveal that facial recognition, including the discrimination and recognition of familiar and unfamiliar faces, depends on two neural pathways [13].

From an anatomical and functional point of view, it is postulated that there are two main circuits: a ventral pathway associated with conscious recognition of facial identity, and a dorsal pathway related to the processing of affective and emotional information associated with the face, which connects to the limbic system [14].

According to the most widely accepted neurophysiological hypothesis, CS results from a functional disconnect between visual recognition of the face (ventral pathway) and the emotional response generated when viewing it (dorsal pathway), thus underpinning the feeling of strangeness and the conviction that one is facing an impostor [11].

Given the above, the neuropsychological complexity of CS is evident. Changes in the brain circuits involved in facial identification and emotional responses highlight the intrinsic relationship between neurological and psychiatric mechanisms [15]. Nevertheless, the relative rarity and heterogeneous presentation of the syndrome, coupled with often

fragmented reports in the literature, represent an obstacle to the consolidation of diagnostic and therapeutic models.

Therefore, this study is justified by the need to synthesize the sparse evidence from case reports, which constitute a significant portion of the knowledge about this rare condition.

The present study differs from other reviews in that it is not restricted to a single aspect, such as neurological etiology. In addition, the scarcity of comprehensive publications on the subject reinforces the relevance of consolidating existing knowledge and broadening the understanding of its manifestations.

It should be noted that this is a secondary review of an analytical and interdisciplinary nature, integrating perspectives from psychiatry, neurology, and neuropsychology. Its scope is to synthesize and critically discuss the published literature, without prescribing or proposing specific clinical conduct.

Thus, the objective of this study was to analyze, through case reports, the therapeutic strategies employed in the management of CS, the explanatory hypotheses adopted, the associated etiological and diagnostic contexts, as well as the complementary methods used in the characterization of the disorder.

Methods

This is an integrative review of the literature. This design was chosen because it allows for the synthesis of evidence from different methodologies (in this case, reports and case series) and the critical and integrated analysis of the knowledge produced on the subject, with the aim of generating a comprehensive understanding of CS in its multiple aspects (clinical, etiological, therapeutic, and theoretical), regardless of their specific diagnostic classification within the spectrum of delusional syndromes.

To ensure rigor, transparency, and reproducibility in the process of identifying, selecting, and analyzing studies, this review was conducted in accordance with the recommendations of the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) [16].

The protocol was not registered with PROSPERO due to incompatibility with the platform's scope, while maintaining methodological rigor in accordance with PRISMA and using double-blind peer review.

The use of the PRISMA tool, commonly associated with systematic reviews, is justified here as a superior methodological instrument for reporting the stages of a literature review in a structured manner and minimizing selection biases, being fully applicable to integrative reviews that follow a systematic protocol.

Eligibility criteria

The review included studies published as case reports or case series that provided a clinical description of individuals diagnosed with CS, either as a primary manifestation or as a significant coexisting condition, even when part of broader presentations of delusional disorder.

The selection criteria covered studies that:

a) provided information on the therapeutic strategies employed (pharmacological, psychosocial, or electroconvulsive);

b) described explanatory hypotheses or theoretical approaches associated with delirium (such as neuropsychological, psychodynamic, or cognitive);

c) reported the suggested or confirmed clinical etiology, including psychiatric (schizophrenia, bipolar disorder) or neurological (dementia or traumatic brain injury) comorbidities;

d) presented the use of complementary diagnostic tests, such as neuroimaging (magnetic resonance imaging or computed tomography), electroencephalogram, or neuropsychological assessment;

e) reported the country of origin of the case, allowing for cultural and contextual analyses;

f) were published in any language until October 2025.

The following were excluded: studies that did not provide sufficient clinical description for analysis; literature reviews, theoretical essays, or editorials; dissertations, theses, course completion papers, and letters to the editor without original clinical case content.

Search strategy

The guiding question for this review was: “What are the therapeutic strategies, theoretical hypotheses, and clinical characteristics described in case reports on Capgras syndrome, including comorbidities, associated etiologies, and diagnostic tests used?”

To answer this question, a strategy based on the PICO model was developed, in which the Population (**P**) consisted of individuals diagnosed with CS; the Intervention (**I**) consisted of clinical, pharmacological, and/or psychosocial therapeutic strategies described in case reports; Comparator (**C**), not applicable; and Outcome (**O**), in the characterization of therapeutic management, explanatory hypotheses, associated clinical etiologies, and diagnostic resources used.

Searches were conducted in the Embase, Lilacs, PubMed/MEDLINE, and Web of Science databases, using controlled descriptors extracted from the DeCS and MeSH vocabularies, combined by Boolean operators (AND, OR).

The search strategy was: ("Capgras Syndrome"[Mesh] OR "Delusional Misidentification Syndrome") AND ("Case Reports"[Publication Type] OR "Case Study") AND ("Diagnosis"[Mesh] OR "Treatment"[Mesh] OR "Therapeutics"[Mesh] OR 'Neuroimaging'[Mesh] OR "Electroencephalography"[Mesh]).

Article selection process

The selection process was conducted using the Rayyan website [17], which was used to organize and screen the studies. Initially, duplicates were removed, followed by reading the titles and abstracts. The final selection was made based on a complete reading of the texts.

Two independent reviewers (JHA and AFG) performed the screening process blindly, based on eligibility criteria. In cases of disagreement, a joint discussion was held and, when necessary, a third reviewer (ARP) was consulted for a final decision.

For each study included, the following information was extracted: author and year of publication, country of origin, patient age and sex, associated comorbidities, presumed or confirmed etiology, explanatory hypotheses, therapeutic strategy adopted, diagnostic tests used, and reported clinical outcome.

Methodological quality assessment

The quality of the case reports included was assessed based on the CARE (Case Report Guidelines) using a structured checklist consisting of 13 items (title, abstract, introduction, timeline, physical examination, diagnostic evaluation, therapeutic intervention, follow-up, outcome, patient perspective, discussion, consent, and additional information).

Two reviewers (JHA and AFG) performed this assessment independently, assigning a dichotomous score (1 = meets; 0 = does not meet) for each item.

The total score (0–13) was converted into the following categories: Excellent (12–13 points): reports that met almost all criteria, including the patient/caregiver perspective and long-term follow-up; High (9–11 points): compliance with most criteria, with occasional shortcomings; Moderate (≤8 points): significant gaps in essential items. Disagreements were resolved by consensus.

Results

A total of 556 studies were identified after applying the search strategy, of which only 11 comprised the final sample (Figure 1). The high number of excluded studies was mainly due to the absence of detailed information required by the inclusion criteria.

The analysis revealed that the criterion most frequently not met was criterion (d), related to the description of complementary tests. Many reports, although presenting the etiology (such as schizophrenia, brief psychotic disorder, or head trauma) and therapeutic interventions, did not report the performance or results of neuroimaging exams, electroencephalograms, or formal neuropsychological assessments. In some cases, only routine tests, such as electrocardiograms or laboratory tests, were mentioned, which proved insufficient to meet the criterion.

Other studies were excluded because they did not present theoretical hypotheses or pathophysiological mechanisms to support the case of CS, limiting themselves to clinical description and conduct without articulation with neurocognitive or psychodynamic models.

Methodological assessment and general characterization of the included studies

The CARE guideline assessment, summarized in Table 1, showed a varied distribution in the methodological quality of the 11 reports included: four (36.4%) were classified as excellent, as they provided detailed descriptions of clinical developments, complementary tests, and, in some cases, included the perspective of the patient or family member.

Five (45.4%) were classified as high quality, complying with most of the guidelines. Finally, two studies (18.2%) were classified as moderate,

mainly because they reported inconclusive test results, limited follow-up, and insufficient description of outcomes.

Clinical and etiological characterization of cases
The cases analyzed, whose details are compiled in Table 2, confirm the notable heterogeneity of CS, covering a wide range of ages (15 to 74 years), genders, and clinical contexts. The etiologies presented included neurodegenerative conditions, primary psychotic conditions, substance-induced conditions, traumatic causes, and atypical presentations.

In neurodegenerative conditions, CS has been associated with dementias, such as Alzheimer's disease and Lewy body dementia, in the context of progressive cognitive decline [18 - 19]. In cases with primary psychotic conditions, there was an association with schizophrenia and schizoaffective disorder, sometimes coexisting with other delusions, such as Cotard, vampirism, and Fregoli syndromes [20, 21, 22].

Cases related to substance use highlighted prolonged cannabis use as a factor related to identification delusions [23]. In addition, cases with atypical presentations related to the sensory system have been described, such as the auditory variant, without changes in visual perception, for example [24].

Explanatory hypotheses adopted in the reports
As summarized in Table 2, the explanatory hypotheses offered by the reports were organized into two main axes, reflecting the underlying etiology.

In cases of organic etiology, neurostructural models prevailed, in which CS was attributed to alterations in perceptual integration networks, temporal-limbic lobe disconnections, and neurochemical dysfunctions associated with neurodegenerative processes or traumatic injuries [18 -19, 25].

In cases of primary or substance-induced psychosis, integrative models predominated, highlighting perceptual anomalies, hyperfamiliarity, and failures in belief monitoring [21, 22, 23, 25].

Hypotheses focused on emotional and symbolic content were central to explaining specific cases, highlighting the influence of postpartum and cultural factors [26], psychodynamic mechanisms such as failure of mentalization and projection in a case of filicide [20], the role of trauma in identity distortion [27], and the proposal of a disconnection mechanism in an exclusively auditory modality [24].

Therapeutic strategies and clinical outcomes

The therapeutic strategies used in different contexts, detailed in Table 3, varied according to the underlying etiology. In acute psychotic cases, the use of antipsychotics was associated with remission or significant improvement in psychotic symptoms [26]. In resistant cases, the use of clozapine gradually reduced some of the symptoms [21 - 22].

In addition to drug management, the use of multidisciplinary strategies, such as neuropsychological assessments and behavioral therapies, has been documented [27].

Thus, the reports reveal that clinical outcomes are directly influenced by the multifactorial nature of the syndrome and by the adequacy of therapeutic management to the unique context of each patient.

Diagnostic Approaches in the Cases Analyzed

The diagnostic investigation, as shown in Table 3, reports differences in diagnostic approaches, such as organic etiologies (neurodegenerative, traumatic) and functional (psychotic, substance-induced).

The resources used in the included reports can be categorized as:

1) neuroimaging;

2) laboratory and toxicological analyses; and

3) electrophysiological and clinical evaluation.

Structural and functional neuroimaging was a cornerstone of the study. Magnetic resonance imaging (MRI) was the most commonly used method [18, 19, 20, 23, 26, 28], with findings ranging from normal results in primary psychoses [20, 22, 24] to the identification of critical changes, such as progressive atrophy in specific areas associated with dementia [18] or post-traumatic encephalomalacia [28].

Computed tomography (CT) was also used, mainly to identify established lesions [21, 25, 28]. Functional imaging techniques, such as positron emission tomography (PET) and single photon emission computed tomography (SPECT), were decisive in confirming metabolic patterns compatible with Alzheimer's disease [18] or revealing atypical perfusion [19].

Laboratory and toxicological analyses were universal for the exclusion of systemic causes. Screening for cannabinoids in urine was fundamental for establishing etiology related to substance use [23, 25, 28].

Blood tests were generally normal [20, 21, 22], and specialized investigations, such as cerebrospinal fluid analysis and autoantibody testing, were reserved for complex neurodegenerative cases [18] or to rule out encephalitis [24]. Hematological monitoring was documented during clozapine use [21 - 22].

In addition, electrophysiological and clinical evaluation complemented the process. Electroencephalogram (EEG) was used to rule out epileptiform activity, usually without abnormalities [19 - 20].

Symptom quantification was performed using validated scales: the PANSS (Positive and Negative Syndrome Scale) measured psychotic improvement [21 - 22], and the Mini-Mental State Examination (MMSE) monitored cognitive decline in dementia syndromes [18 - 19].

Discussion

The results of this integrative review highlighted the complexity and multifactorial nature of DISs, with a focus on CS.

Analysis of the case reports demonstrated a widely heterogeneous clinical profile, ranging from adolescents with substance-induced psychosis [22, 25] to elderly individuals with neurodegenerative processes [18 - 19], which corroborates the transdiagnostic nature of the syndrome, previously highlighted in reviews of delusional phenomena [29 - 30].

With regard to explanatory hypotheses, a polarization between neurocognitive and psychodynamic models was observed.

Cases with organic etiology were consistently explained by dysfunctions or disconnections in specific brain circuits. For example, posterior cortical atrophy was correlated with delirium in a case of Alzheimer's disease [18], aligning with the literature that points to this region as a critical nucleus for visuoperceptive integration [31].

Similarly, the disconnection between temporal and limbic pathways proposed to explain post-traumatic CS reinforces the classic model of affective-cognitive disconnection, originally proposed for prosopagnosia [28].

However, purely neurostructural models were insufficient to explain cases such as post-traumatic Fregoli syndrome [27] or filicide in the context of schizoaffective disorder [20], in which psychological factors such as trauma and projection mechanisms were central.

This duality suggests that an integrative model, which considers both neural and psychological vulnerabilities, is necessary to understand the genesis of misidentification delusions [32].

Diagnostic investigation proved to be essentially multimodal, with structural neuroimaging serving as a central pillar for ruling out or confirming organic etiologies.

MRI was the most widely used method [18 - 19, 20, 23, 26, 28], and its normality in cases of primary psychosis [20, 22, 24] proved to be as valuable a finding as the identification of specific lesions in organic cases [18, 28]. This exclusionary approach is essential and follows the recommendations for the evaluation of a first psychotic episode.

However, the identification of findings such as enlarged ventricles [27] or thickening of the cerebellar lobules [23] raises questions about the clinical significance of nonspecific abnormalities.

Future longitudinal studies with advanced neuroimaging are needed to determine whether such findings represent markers of vulnerability or epiphenomena [33].

Analysis of the reports confirms the absence of a standardized protocol for CS, reflecting its transdiagnostic nature. Direct extrapolation of therapeutic responses from isolated reports is risky, given the uniqueness of each etiological, pharmacological, and psychosocial context.

Although antipsychotics, particularly clozapine, have shown efficacy in psychotic conditions [21 - 22, 27 - 28], their indication should be guided by the treatment of the primary condition, rather than by the delusional syndrome itself.

Remission in cases such as postpartum psychosis [26] or after simple psychosocial interventions [24] highlights the decisive importance of contextual factors, which are often overlooked in reports focusing solely on pharmacotherapy.

The heterogeneity of cases and the lack of detailed description of non-pharmacological interventions in most studies [20, 25 - 26, 28] expose a gap in the literature and a risk of inappropriate generalization, which can lead to ineffective clinical practices.

Therefore, optimal management should be individualized, multimodal, and always integrated with care for the underlying disease.

The analysis of outcomes revealed that they are deeply influenced by the underlying disease. While patients with psychosis showed complete remission of delusions [22, 25 – 26], those with neurodegenerative diseases exhibited a progressive course, with the disappearance of Capgras delusion over the years, accompanying the advancement of dementia and global cognitive decline [18].

This finding highlights that, in neurodegenerative conditions, delirium may be a transient symptom in a continuum of decline, and its management should be integrated into the care of the primary condition.

The methodological quality rating using CARE criteria allowed us to stratify the robustness of the evidence from the 11 included studies. The four reports rated as "excellent" [19, 23, 25 - 26] constitute the most solid core for inferences about clinical evolution and therapeutic response, due to their detailed description and use of validated scales.

The five studies of "high" quality [18 - 19, 23 - 24, 26] complement this basis with well-structured reports, although with some specific limitations, such as the absence of the patient's perspective.

The two "moderate" studies [20, 27], on the other hand, call for caution in interpreting specific findings, such as the limited therapeutic description in the forensic report [20] and the inconclusive tests in the case associated with trauma [27], which make their evidence on the effectiveness of specific interventions less substantive.

Therefore, conclusions about etiological heterogeneity and the plausibility of theoretical models are supported by the set of studies included.

However, inferences about the comparative effectiveness of specific therapeutic interventions should critically consider methodological quality, prioritizing evidence from reports with greater descriptive completeness (classified as high and excellent quality).

Study limitations

The interpretation of the results of this study should take into account some important limitations. The final number of studies included was relatively small, reflecting the rarity of the phenomenon (Capgras) and the application of strict eligibility criteria, which prioritized reports with detailed clinical and theoretical descriptions. Although this strategy ensured greater analytical depth, it inevitably restricted the sample size.

Another critical factor is the high clinical and etiological heterogeneity of the cases analyzed, which range from neurodegenerative conditions to primary and substance-induced psychoses.

This diversity, while illustrating the transdiagnostic nature of the syndrome, prevents direct comparisons, aggregate quantitative analyses, and, consequently, the generalization of findings.

In addition, it is recognized in the literature that case reports, as a source of evidence, are potentially subject to publication bias. Cases with particularly atypical presentations or notable outcomes may be overrepresented, which may influence the synthesized perception of the clinical picture and therapeutic response.

Finally, the descriptive and uncontrolled nature of the case reports, which constitute the entirety of the evidence summarized here, does not allow for causal inferences or robust assessments of comparative effectiveness.

Therefore, the conclusions of this review should be interpreted as an analytical and critical synthesis of the available literature, serving to map the complexity of the phenomenon and indicate directions for future research, but not to establish definitive clinical conduct.

Conclusion

The present study suggests that CS constitutes a heterogeneous clinical manifestation, present in various psychiatric, neurological, and substance-induced contexts, whose management tends to follow the diagnosis and treatment of the underlying etiology.

Furthermore, it was observed that there is no universal therapeutic strategy, and individualized, multimodal treatment is necessary.

Reports suggest an association between the use of atypical antipsychotics and improvement in psychotic symptoms, whereas in neurodegenerative conditions, the focus is on managing the underlying disease. Psychosocial

interventions are frequently described as relevant for functional improvement and long-term stabilization.

Thus, CS can be interpreted as a clinical marker of a disruption in the integration between perception and affect. This perspective reinforces its transdiagnostic nature and suggests that its clinical relevance lies in indicating underlying neuropsychological dysfunctions, which cut across different clinical conditions and point to alterations in the systems of recognition, affective processing, and belief formation.

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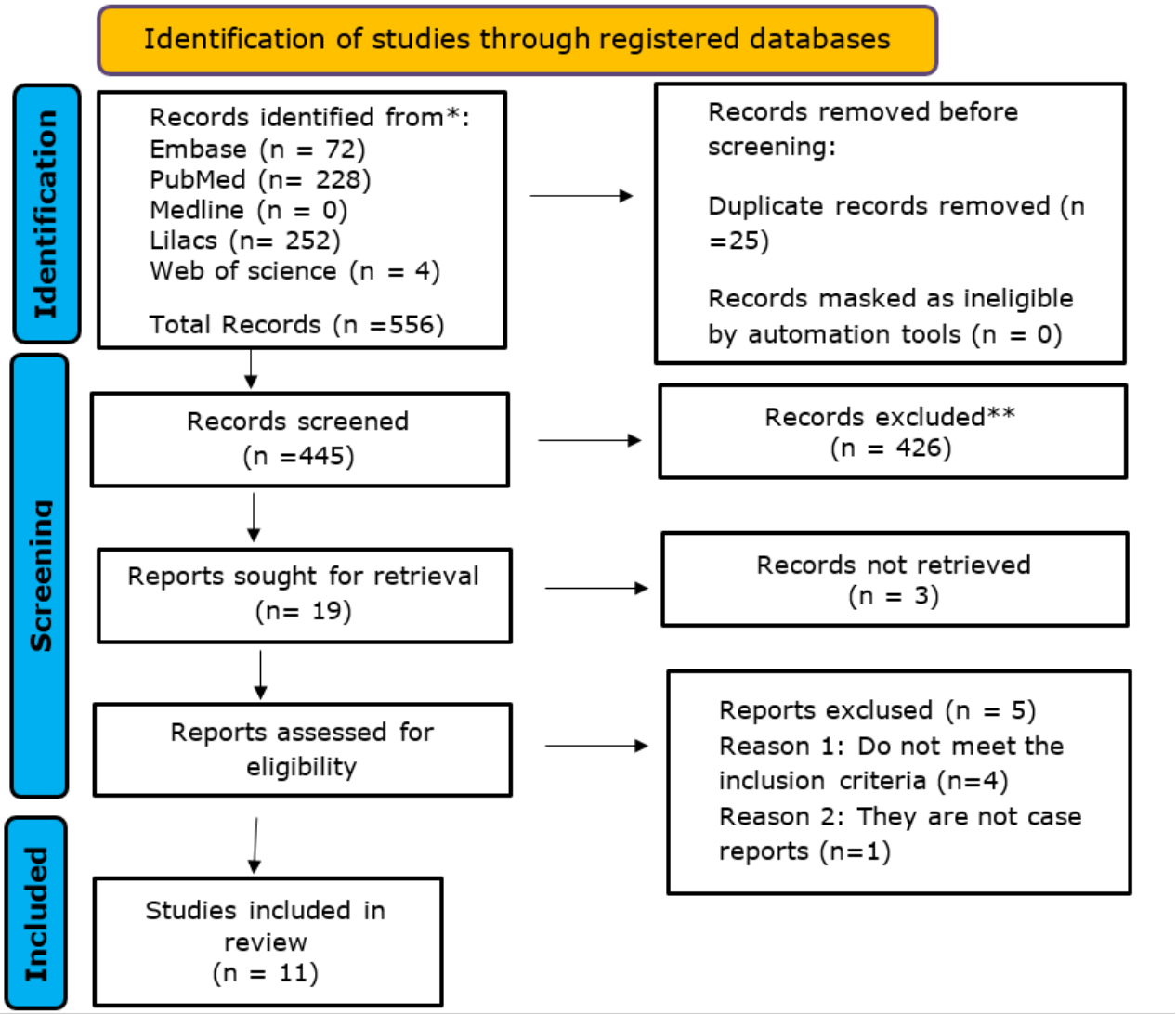

Figure 1. PRISMA Flow Diagram
Source: The authors

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Table 1. Evaluation of studies according to CARE (CAse REport) guidelines

#N	Authors and Year	CARE Compliance	CARE Classification
01	Schroeter et al. (2020) [18]	Exceptional longitudinal follow-up (8 years). Includes the caregiver's perspective (spouse). Uses a comprehensive battery of tests to correlate clinical presentation and findings.	Discharge
02	Watanabe et al. (2024) [19]	Detailed clinical timeline. Describes diagnostic tests and the use of clinical scales for monitoring.	Excellent
03	Ben Ammar et al. (2021) [20]	Contextualizes the case within a forensic context.	Moderate
04	Revilla et al. (2021) [21]	Describes a longitudinal clinical history (13 years). Uses a validated scale (PANSS).	Discharge
05	O'Brien et al. (2023) [22]	Describes a complex clinical presentation. Uses a validated scale (PANSS) to measure outcomes.	Discharge
06	Castro et al. (2022) [23]	Presents a detailed clinical timeline in table form. Clear temporal relationship between exposure (cannabis) and symptoms.	Excellent
07	Shaw et al. (2024) [24]	Adequately describes an atypical clinical presentation. Discusses the differential diagnosis.	Discharge
08	Quarenta et al. (2022) [25]	Follows recommended guidelines for investigation. Includes the patient's perspective. Outcome and follow-up are described.	Excellent
09	Connors et al. (2024) [26]	Clear and comprehensive structure; patient's perspective after recovery; 3-year follow-up	Excellent
10	Ghannadi et al. (2024) [27]	Moderate structure; history of trauma; inconclusive imaging studies; limited follow-up.	Moderate
11	Gramling et al. (2024) [28]	Clear chronological report. Describes appropriate diagnostic workup and therapeutic intervention with outcome.	Discharge

Source: The authors.

Table 2. Description of articles included in the integrative review

#N	Authors and Year	Country of origin of the case	Objective	Sample group	Adopted explanatory hypothesis
01	Schroeter et al. (2020) [18]	Germany	To identify neural correlates of SC in Posterior Cortical Atrophy (PCA) using advanced neuroimaging.	Woman, 57 years old, with PCA/Alzheimer's disease.	Neuroanatomical/Structural: Atrophy in specific regions of the posterior cingulate gyrus and middle frontal gyrus.
02	Watanabe et al. (2024) [19]	Japan	To describe SC as a presenting symptom of Dementia with Lewy Bodies (DLB).	Woman, 74 years old, with DLB.	Neurodegenerative: SC is a symptom indicative of DLB.
03	Ben Ammar et al. (2021) [20]	Tunísia	To analyze a case of filicide motivated by SC in schizoaffective disorder.	Woman, 35 years old, with schizoaffective disorder.	Psychodynamic/Violence Risk: SC as a specific risk factor for homicidal impulses toward the delusion's target.
04	Revilla et al. (2021) [21]	Peru	To report the rare coexistence of Cotard and Capgras syndromes in treatment-resistant schizophrenia.	Man, 23 years old, with treatment-resistant schizophrenia.	Inferential: Perceptual anomaly followed by abnormal rationalization; Capgras as an externalization of negative events.
05	O'Brien et al. (2023) [22]	Ireland	To report the coexistence of Capgras and vampirism delusions in treatment-resistant schizophrenia and response to clozapine.	Boy, 15 years old, with schizophrenia.	Integrative (Sensory-Inferential): Impairment in sensory processing combined with failure in belief evaluation.
06	Castro et al. (2022) [23]	Colômbia	To report SC triggered by cannabis use.	Man, 28 years old, with chronic cannabis use.	Organic/Substance-Induced: Cannabinoid use as the likely trigger.

#N	Authors and Year	Country of origin of the case	Objective	Sample group	Adopted explanatory hypothesis
07	Shaw et al. (2024) [24]	United Kingdom	To describe an exclusively auditory variant of SC in a first psychotic episode.	Woman, 70 years old, experiencing a first psychotic episode.	Multimodal Neuropsychological: Deficits in non-visual sensory pathways (auditory) may cause the syndrome.
08	Quarenta et al. (2022) [25]	Portugal	To describe a case of "Subjective Doubles Syndrome" in a first psychotic episode with cannabis use.	Man, 30 years old, experiencing a first psychotic episode.	Neurofunctional: Hyperactivity of the perirhinal cortex (hyperfamiliarity) and frontal dysfunction.
09	Connors et al. (2024) [26]	Austrália	To report a case of Fregoli Delusion in postpartum psychosis to discuss its genesis.	Woman, 30 years old, with postpartum psychosis.	Cognitive/Belief Model: Primary paranoia leading to hyperactivity of "identity nodes."
10	Ghannadi et al. (2024) [27]	Irã	To report a complex case of Fregoli Delusion associated with trauma and substance use.	Man, 23 years old.	Psychodynamic/Trauma: Trauma as a significant contributing factor to perceptual and delusional alterations.
11	Gramling et al. (2024) [28]	EUA	To highlight the association of SC with Traumatic Brain Injury (TBI) and response to olanzapine.	Man, 39 years old, with a history of TBI.	Neurostructural: Disconnection between the temporal lobe and limbic system due to injury (frontal encephalomalacia).

Source: The authors.
Caption: #N: Article identification number in the review. **DLB:** Dementia with Lewy Bodies. **PCA**: Posterior Cortical Atrophy. **CS:** Capgras Syndrome**. TBI:** Traumatic Brain Injury**.**

Table 3. Analysis of characteristics and interventions in clinical cases

#N	Authors and Year	Associated Comorbidities	Primary Etiology	Diagnostic Tests Used	Therapeutic Approach	Clinical Outcome
01	Schroeter et al. (2020) [18]	Posterior Cortical Atrophy (PCA)/Alzheimer's; Balint's Syndrome; Depression.	Neurodegenerative Organic (Alzheimer's Disease – PCA variant).	MRI: Progressive atrophy. PET: Typical Alzheimer's pattern. Lumbar puncture: Abnormal. MMSE: 24 → 4	Galantamine + Quetiapine + Valproate (for seizures) + Rehabilitation.	Dementia progression. Capgras delusion resolved after 8 years.
02	Watanabe et al. (2024) [19]	Dementia with Lewy Bodies (DLB); Primary Sjögren's Syndrome; Burning Mouth Syndrome.	Neurodegenerative Organic (DLB).	MRI/EEG: Normal. SPECT: Atypical hypoperfusion. Cognitive assessment (MMSE/HDSR).	Pharmacological: Rivastigmine + Amitriptyline + Mirtazapine.	Good prognosis for pain. DLB with mild progression.
03	Ben Ammar et al. (2021) [20]	Schizoaffective Disorder; Chronic Delusions; Non-adherence.	Psychiatric (Schizoaffective Disorder).	MRI, EEG, biological tests: All normal.	(The focus of the article was forensic evaluation, not subsequent treatment).	Patient deemed legally non-responsible. Case illustrates violence risk in SIDs.
04	Revilla et al. (2021) [21]	Treatment-resistant schizophrenia; concomitant Cotard syndrome.	Psychiatric (Schizophrenia).	Brain CT scan: Normal. PANSS: 125 → 51.	Previous failure: Multiple antipsychotics + ECT. Success: Clozapine (650 mg/day).	Significant improvement in symptoms. Marked reduction in the PANSS score.
05	O'Brien et al. (2023) [22]	Treatment-resistant schizophrenia; mild	Psychiatric (Schizophrenia).	Brain MRI: Normal. PANSS: 146 (on admission) → 45.	Previous failure: Risperidone, Olanzapine. Success: Clozapine (550	Complete resolution of delusions. Improvement in social functioning.

#N	Authors and Year	Associated Comorbidities	Primary Etiology	Diagnostic Tests Used	Therapeutic Approach	Clinical Outcome
		intellectual disability.			mg/day) + multidisciplinary therapy	
06	Castro et al. (2022) [23]	Chronic cannabis use (8 years); aggressive behavior.	Organic/Substance-related (Cannabis use).	Toxicology: THC positive. MRI: Mild prominence of the cerebellar folia.	Clozapine (500 mg/day) + Valproic acid + Pipotiazine (IM).	Complete resolution in 2 months. Discharged with follow-up.
07	Shaw et al. (2024) [24]	First episode of psychosis. Advanced age (70 years).	Psychiatric (Functional) – Psychosis. Trigger: isolation during the COVID-19 pandemic (auditory/telephone-only contact with her husband).	Neuroimaging and extensive laboratory workup (including autoantibodies): No abnormalities.	Psychotropic medication (not specified). Diagnostic workup to rule out an organic cause.	Resolution of the delusion after in-person contact.
08	Quarenta et al. (2022) [25]	PEP; Cannabis use disorder (daily use, 10 years).	Psychiatric/Substance-related (Cannabis-induced psychosis).	Toxicology: THC positive. Brain CT scan: Normal.	Paliperidone (9 mg) + Lorazepam + psychosocial interventions (TCC, family support).	Complete remission in 7 days. Adherence issues during follow-up..
09	Connors et al. (2024) [26]	Psychiatric/neurological disorders; perinatal period.	Psychiatric/presumed organic (e.g., dementia, traumatic brain injury, substance use).	Neuroimaging (MRI, CT), EEG, neuropsychological testing..	Pharmacological: Quetiapine (up to 400 mg/day). Psychosocial: Admission to a perinatal unit, psychoeducation.	Complete and sustained remission (>3 years). Functional adaptation to motherhood.

#N	Authors and Year	Associated Comorbidities	Primary Etiology	Diagnostic Tests Used	Therapeutic Approach	Clinical Outcome
10	Ghannadi et al. (2024) [27]	Severe trauma history (kidnapping, abuse). Substance use disorder (in remission). Suicide attempts.	Psychiatric (Fregoli delusion). Factors: trauma and neurodevelopmental anomaly..	Neuroimaging: Enlarged ventricles (nonspecific developmental anomaly).	Integrated: Risperidone (antipsychotic) + trauma-focused CBT.	Aim: remission and improved quality of life. Ongoing monitoring required.
11	Gramling et al. (2024) [28]	History of traumatic brain injury; cannabis use; family history of schizophrenia.	Organic (encephalomalacia due to traumatic brain injury). Trigger: cannabis.	CT scan: Left frontal encephalomalacia. Urine toxicology: Positive for cannabinoids.	Olanzapine (15 mg/day).	Significant improvement within 3 days. Discharged for outpatient follow-up.

Source: The authors.
Caption: DLB: Dementia with Lewy Bodies. **DISs:** Delusional Identification Syndrome. **ECT:** Electroconvulsive Therapy. **IM:** Intramuscular. **MMSE:** Mini-Mental State Examination. **PANSS:** Positive and Negative Syndrome Scale. **PCA:** Posterior Cortical Atrophy. **FEP:** First Psychotic Episode. **CBT:** Cognitive Behavioral Therapy. **TBI:** Traumatic Brain Injury. **EEG:** Electroencephalogram. **PET:** Positron Emission Tomography. **MRI:** Magnetic Resonance Imaging. **SPECT:** Single Photon Emission Computed Tomography. **CT:** Computed Tomography