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Fregoli Syndrome: A Comprehensive Review of Clinical Features, Diagnosis, and Management

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Abstract*—Fregoli syndrome is a rare and intriguing delusional misidentification disorder characterized by the persistent belief that different individuals are, in fact, a single familiar person who repeatedly changes appearance or disguises themselves. Originally described in the early twentieth century, the syndrome represents a profound disturbance in identity recognition, familiarity processing, and belief evaluation. Although uncommon, Fregoli syndrome holds significant clinical importance due to its strong association with schizophrenia, mood disorders with psychotic features, neurodegenerative diseases, traumatic brain injury, and other neurological conditions. Clinically, the syndrome is marked by intense paranoia, persecutory ideation, heightened emotional salience, and, in some cases, aggressive behavior toward misidentified individuals. Patients often display firm conviction in their beliefs, despite clear contradictory evidence, reflecting impairments in reality testing and metacognitive reasoning. Neuropsychiatric models suggest that Fregoli syndrome arises from abnormal familiarity attribution combined with deficits in belief evaluation, particularly involving frontotemporal and limbic brain networks. Hyperfamiliarity responses, impaired executive control, and disrupted self–other distinction play central roles in the development and maintenance of the delusion. Diagnosis remains primarily clinical and requires careful differentiation from other delusional misidentification syndromes, such as Capgras syndrome, as well as from primary psychotic disorders. Management focuses on treating the underlying psychiatric or neurological condition, with antipsychotic medications forming the cornerstone of therapy. Adjunctive psychological interventions, environmental stabilization, and risk management strategies are essential components of care. Given its potential for significant distress and safety concerns, early identification and multidisciplinary management are critical. This review aims to provide a comprehensive overview of the clinical features, diagnostic considerations, neurocognitive mechanisms, and management strategies associated with Fregoli syndrome, highlighting its relevance within contemporary neuropsychiatry.*

***Keywords*—** *Fregoli syndrome; Delusional misidentification; Hyperfamiliarity; Psychosis; Neuropsychiatry; Belief formation.*

I. Introduction

Fregoli syndrome is a rare and intriguing delusional misidentification disorder characterized by the persistent belief that different people are, in fact, a single familiar individual who changes appearance or is disguised. First described in the early twentieth century, the syndrome derives its name from Leopoldo Fregoli, an Italian stage actor renowned for rapid costume changes, reflecting the core phenomenology of mistaken identity through perceived transformation. Although uncommon, Fregoli syndrome holds significant importance within neuropsychiatry due to its implications for understanding face recognition, self–other differentiation, and delusional belief formation. Clinically, Fregoli syndrome is most often observed in the context of schizophrenia, mood disorders with psychotic features, and organic brain diseases. It has also been reported following traumatic brain injury, stroke, neurodegenerative disorders, epilepsy, and encephalitic conditions. The syndrome frequently presents alongside paranoia, ideas of reference, emotional dysregulation, and aggressive behavior, thereby posing substantial diagnostic and management challenges in both psychiatric and neurological settings.[1, 2]

From a cognitive neuropsychiatric perspective, Fregoli syndrome represents a disorder of familiarity attribution and belief evaluation. Patients typically exhibit heightened or inappropriate feelings of familiarity toward strangers, which are then rationalized through delusional explanations. This aberrant familiarity response, coupled with impaired reality testing, results in a fixed and distressing belief system resistant to contradictory evidence. Unlike other misidentification syndromes, Fregoli syndrome is characterized by hyperfamiliarity rather than loss of recognition, offering a unique window into the neural mechanisms of face processing and emotional salience. This review aims to provide a comprehensive overview of Fregoli syndrome, focusing on its clinical features, diagnostic considerations, neurobiological underpinnings, and current management strategies. By integrating psychiatric, neurological, and cognitive models, the article seeks to enhance clinical recognition and promote evidence-informed therapeutic approaches.

II. Historical Background and Conceptual Evolution

Fregoli syndrome was first formally described in the early twentieth century as part of a broader group of delusional misidentification syndromes. Early psychiatric literature recognized these syndromes as disturbances of identity perception rather than simple memory deficits. The defining feature of Fregoli syndrome the belief that multiple individuals are actually a single familiar person in disguise distinguished it from other misidentification phenomena and prompted interest in the psychological mechanisms underlying familiarity and recognition.[2]

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Initially, Fregoli syndrome was conceptualized within a psychodynamic framework, where persecutory anxiety and unresolved interpersonal conflicts were thought to drive misidentification beliefs. These early interpretations emphasized symbolic meanings, viewing the "disguised persecutor" as an external projection of internal fears. However, such explanations failed to account for the frequent association of the syndrome with structural brain pathology and neurological illness.[3]

III. Epidemiology and Clinical Context

Fregoli syndrome is considered one of the rarest forms of delusional misidentification disorders, with its true prevalence remaining uncertain due to underreporting and frequent misdiagnosis. Most available data derive from case reports and small clinical series rather than large epidemiological studies. The syndrome occurs across a wide age range, though it is more commonly reported in adults, particularly those with established psychiatric or neurological conditions. There is no consistent evidence of strong sex predilection, although some reports suggest a slightly higher occurrence in males, possibly reflecting greater exposure to traumatic brain injury or neurodegenerative conditions. Clinically, Fregoli syndrome is most often encountered in tertiary psychiatric and neurological care settings, where patients present with complex symptom profiles. It rarely occurs as an isolated phenomenon and is typically embedded within broader psychotic or organic syndromes. The delusional belief often emerges during acute exacerbations of illness, periods of severe affective disturbance, or following neurological insult. In many cases, the syndrome develops abruptly, particularly in organic brain disease, while in primary psychiatric disorders it may evolve gradually alongside worsening psychosis.

The clinical context of Fregoli syndrome is frequently marked by heightened emotional arousal and perceived threat. Patients commonly identify the "disguised" individual as someone familiar and emotionally significant, such as a family member, caregiver, or authority figure. This familiarity is often accompanied by persecutory interpretations, leading to fear, mistrust, and occasionally violent behavior. As a result, the syndrome poses substantial risks in inpatient and community settings. From a diagnostic standpoint, Fregoli syndrome is often overshadowed by the primary disorder, leading clinicians to overlook its distinct phenomenology. Accurate recognition requires careful phenomenological assessment and an awareness of delusional misidentification syndromes. Understanding the epidemiological and clinical context of Fregoli syndrome is essential for timely diagnosis, appropriate risk management, and effective therapeutic intervention.[3 – 6]

IV. Core Clinical Features and Symptomatology

Fregoli syndrome is defined by a fixed delusional belief that multiple individuals are actually a single known person who assumes different appearances or disguises. Unlike other misidentification syndromes characterized by loss of familiarity, Fregoli syndrome involves excessive or inappropriate familiarity toward strangers. This hyperfamiliarity forms the experiential foundation upon which the delusion is constructed. The misidentification typically involves individuals encountered in everyday settings, such as hospital staff, neighbors, or passersby. Patients may insist that these individuals share the same voice, mannerisms, gaze, or "essence" as the familiar persecutor, despite clear physical differences. The delusion is usually systematized and internally coherent, with patients providing elaborate explanations to justify the perceived transformations. Emotionally, Fregoli syndrome is strongly associated with paranoia, anxiety, and irritability. Many patients believe that the disguised individual is following, monitoring, or attempting to harm them. This persecutory dimension distinguishes Fregoli syndrome from benign misidentification experiences and significantly contributes to distress and behavioral dysregulation. Aggression toward the misidentified individual is a well-documented clinical concern.

Cognitive features include impaired reality testing, rigid belief maintenance, and resistance to contradictory evidence. Insight is typically absent, and attempts at logical correction often intensify the delusion. Additional psychotic symptoms such as hallucinations, thought disorder, and ideas of reference may coexist, particularly in schizophrenia-spectrum disorders. Behaviorally, patients may exhibit avoidance, hypervigilance, or confrontational behaviors directed at the perceived persecutor. In hospital settings, this may manifest as refusal of care, mistrust of clinicians, or repeated demands for staff changes. Recognizing these core features is critical for differentiating Fregoli syndrome from generalized paranoia and for implementing targeted management strategies.[7, 8]

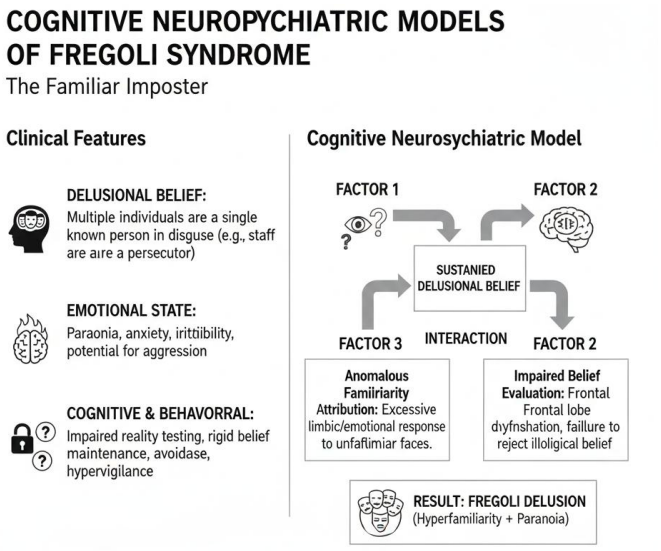

Figure 1. Mechanisms of Fregoli Delusion: From Anomalous Familiarity to Fixed Belief

With advances in neuropsychiatry, the conceptualization of Fregoli syndrome shifted toward neurobiological and cognitive models. Case reports linking the syndrome to frontal and temporal lobe lesions challenged purely psychological explanations and supported an organic basis. The emergence of cognitive neuropsychiatry further refined understanding by

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framing Fregoli syndrome as a disorder of familiarity attribution and belief evaluation rather than face perception alone (Figure 1). Over time, Fregoli syndrome came to be understood as part of a spectrum of delusional misidentification disorders, each reflecting distinct disruptions in the interaction between perceptual processing, emotional salience, and higher-order reasoning. This evolution marked a critical transition from descriptive psychopathology to mechanistic explanations, positioning Fregoli syndrome as a valuable model for studying delusions, self–other representation, and abnormal belief formation.[9 – 12]

V. Psychiatric and Neurological Associations

Fregoli syndrome most commonly occurs in association with severe psychiatric disorders, particularly schizophrenia and related psychotic spectrum conditions. Within schizophrenia, the syndrome is frequently embedded in a broader delusional system characterized by paranoia, ideas of reference, and disorganized thinking. The misidentification belief often reinforces persecutory themes, contributing to heightened emotional distress and behavioral instability. Mood disorders with psychotic features, especially severe depressive episodes, have also been linked to Fregoli syndrome, where nihilistic or guilt-based delusions may coexist with misidentification phenomena. Beyond primary psychiatric illnesses, Fregoli syndrome has strong associations with neurological disorders.[13, 14] Structural brain lesions, particularly involving the frontal and temporal lobes, are frequently reported in affected individuals. Traumatic brain injury is a notable precipitating factor, especially when damage disrupts frontotemporal networks responsible for identity processing and emotional regulation. Cerebrovascular accidents, particularly right hemispheric strokes, have also been implicated, often leading to abrupt onset of misidentification beliefs. Neurodegenerative diseases represent another important clinical context. Fregoli syndrome has been described in patients with dementia, including Alzheimer's disease and dementia with Lewy bodies, where progressive cognitive decline alters familiarity processing and belief evaluation. Epilepsy, especially temporal lobe epilepsy, has been associated with transient or persistent misidentification delusions, highlighting the role of abnormal neural excitability in identity perception. Inflammatory and autoimmune encephalitic conditions further support an organic contribution to the syndrome. The frequent coexistence of psychiatric and neurological pathology underscores the need for an integrated neuropsychiatric approach. Fregoli syndrome should be viewed not as a standalone diagnosis, but as a syndrome reflecting disruption across multiple cognitive and neural systems.

VI. Cognitive Neuropsychiatric Models

Cognitive neuropsychiatry provides a robust framework for understanding Fregoli syndrome as a disorder of belief formation rather than a simple perceptual deficit. Central to this perspective is the idea that misidentification arises from abnormal familiarity responses combined with impaired belief evaluation mechanisms. Patients experience an excessive sense of familiarity toward unfamiliar individuals, which the brain attempts to explain through delusional reasoning. The two-factor model of delusion formation is particularly influential in explaining Fregoli syndrome. The first factor involves an anomalous experience, such as inappropriate emotional familiarity triggered by a stranger's face. The second factor is a deficit in belief evaluation, typically linked to frontal lobe dysfunction, which prevents the individual from rejecting implausible explanations. Together, these factors allow the delusional belief to form and persist despite contradictory evidence. Alternative models emphasize disrupted self–other differentiation and embodied cognition. According to these views, Fregoli syndrome reflects a breakdown in the boundaries between internal representations of familiar individuals and external perceptual input. Heightened emotional salience, often driven by paranoia or anxiety, further amplifies this misattribution process. Aberrant salience attribution causes neutral stimuli to be perceived as personally significant, reinforcing the delusion.

Importantly, cognitive models explain why Fregoli syndrome is often accompanied by emotional intensity and threat perception. The misidentification belief serves as a rationalizing narrative that organizes abnormal experiences into a coherent, though false, explanatory framework. These models bridge phenomenology and neurobiology, offering clinically relevant insights into assessment and treatment planning.[15, 16]

VII. Neurobiological and Neuroanatomical Correlates

Neurobiological models of Fregoli syndrome emphasize dysfunction within distributed neural networks involved in face recognition, emotional processing, and belief evaluation. Rather than arising from isolated brain regions, the syndrome reflects impaired integration between perceptual and affective systems. Neuroanatomical evidence consistently implicates frontotemporal circuitry, particularly structures responsible for assigning emotional significance to perceived stimuli.

The temporal lobes play a central role in facial recognition and familiarity processing. Abnormal activation within these regions can generate exaggerated familiarity responses to unfamiliar faces, forming the experiential basis of misidentification. When these distorted familiarity signals are not appropriately regulated, they contribute to erroneous identity attribution. The amygdala and related limbic structures further modulate emotional salience, often amplifying fear and paranoia associated with the perceived "disguised" individual. Frontal lobe dysfunction is critical in explaining the persistence of delusional beliefs. The prefrontal cortex is responsible for reality testing, belief updating, and inhibitory control. Impairment in these functions prevents the individual from rejecting implausible interpretations, even when confronted with contradictory evidence. This explains why patients maintain rigid convictions despite logical inconsistencies. Right hemispheric dominance has been frequently observed in cases of delusional misidentification, highlighting the importance of right-sided networks in social cognition and self–other distinction.[17 – 19] Disruption of

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connectivity between the frontal cortex and limbic regions appears particularly relevant, resulting in emotional experiences that are not adequately contextualized by higher-order reasoning. Overall, neurobiological findings support the view of Fregoli syndrome as a network disorder, where abnormal familiarity signals, emotional dysregulation, and impaired cognitive control interact to produce a stable delusional belief system.

VIII. Diagnostic Approach and Differential Diagnosis

The diagnosis of Fregoli syndrome is primarily clinical and relies on detailed phenomenological assessment. A thorough psychiatric interview is essential to elicit the specific content and structure of the misidentification belief. Clinicians must distinguish between generalized paranoia and true misidentification by carefully exploring whether the patient believes multiple individuals are actually a single familiar person in disguise. Assessment should include evaluation of insight, belief flexibility, emotional response, and associated psychotic features. Particular attention must be paid to risk assessment, as the persecutory nature of the delusion may predispose to aggression. Cognitive screening is also important, especially in older patients or those with suspected neurological disease.

Neuroimaging and neurological investigations play a supportive role, particularly when organic pathology is suspected. Brain imaging may reveal structural lesions, atrophy, or vascular changes that contribute to symptom development. Electroencephalography may be useful in cases associated with epilepsy or altered consciousness. Differential diagnosis includes other delusional misidentification syndromes, such as Capgras syndrome, intermetamorphosis, and subjective doubles. Fregoli syndrome is distinguished by hyperfamiliarity rather than loss of recognition. Conditions such as delirium, severe dissociative disorders, and culturally sanctioned beliefs should also be considered to avoid misdiagnosis. Accurate diagnosis requires interdisciplinary collaboration, integrating psychiatric, neurological, and cognitive perspectives to ensure appropriate treatment planning.

IX. Management and Treatment Strategies

The management of Fregoli syndrome requires a comprehensive and individualized approach, as the condition rarely occurs in isolation and is often embedded within complex psychiatric or neurological disorders. Treatment strategies focus primarily on addressing the underlying illness while simultaneously managing the misidentification delusion and associated behavioral risks. Early recognition and intervention are crucial to prevent escalation of paranoia, aggression, and functional deterioration.

Pharmacological treatment remains the cornerstone of management. Antipsychotic medications are commonly employed, particularly in cases associated with schizophrenia or other psychotic disorders. Both typical and atypical antipsychotics have been used, with selection guided by symptom profile, comorbidities, and tolerability. In mood disorders with psychotic features, antidepressants or mood stabilizers are often combined with antipsychotic therapy to address affective symptoms contributing to delusional intensity.

In neurologically driven cases, treatment of the underlying condition is essential. This may include antiepileptic therapy in seizure-related cases, immunotherapy in inflammatory conditions, or optimized management of neurodegenerative disease. Cognitive and behavioral strategies play a supportive role, particularly in reducing distress and improving coping mechanisms. Direct confrontation of the delusion is generally ineffective and may worsen agitation; instead, therapeutic engagement emphasizes emotional validation without reinforcing false beliefs.

Environmental modifications and risk management are critical, especially in inpatient settings. Consistency of caregivers, clear communication, and minimizing triggers can reduce misidentification episodes. In severe or treatment-resistant cases, electroconvulsive therapy has been reported to be beneficial, particularly when Fregoli syndrome occurs in the context of severe mood disorders.[20]

X. Prognosis, Ethical Considerations, and Clinical Challenges

The prognosis of Fregoli syndrome varies widely depending on the underlying etiology, severity of symptoms, and timeliness of intervention. Cases associated with acute neurological insults or mood disorders often demonstrate better outcomes, particularly when treatment is initiated promptly. In contrast, chronic psychotic disorders or progressive neurodegenerative conditions may be associated with persistent or recurrent misidentification delusions.

Ethical challenges frequently arise in the management of Fregoli syndrome due to impaired insight and heightened risk of harm. Issues related to autonomy, informed consent, and use of involuntary treatment must be navigated carefully. Clinicians must balance respect for patient rights with the need to ensure safety for both the patient and others.

Clinical challenges include diagnostic uncertainty, treatment resistance, and poor adherence to therapy. The rarity of the syndrome and limited clinician familiarity further complicate management. Multidisciplinary collaboration is essential to address the complex psychiatric, neurological, and psychosocial dimensions of the condition.[21, 22]

XI. Future Directions and Research Gaps

Despite its clinical significance, Fregoli syndrome remains under-researched. Future studies should focus on systematic epidemiological data, advanced neuroimaging techniques, and longitudinal outcomes. Greater integration of cognitive neuroscience and clinical psychiatry may yield improved diagnostic markers and targeted interventions. Development of standardized assessment tools for delusional misidentification syndromes represents another important research priority[23, 24].

XII. Conclusion

Fregoli syndrome is a rare but clinically significant delusional misidentification disorder that provides valuable insights into identity processing, familiarity attribution, and

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belief formation. Its occurrence across psychiatric and neurological conditions highlights the importance of an integrated neuropsychiatric framework. Improved recognition, thoughtful diagnostic assessment, and individualized management strategies are essential for optimizing outcomes. Continued research into the cognitive and neurobiological mechanisms underlying Fregoli syndrome will not only enhance clinical care but also deepen understanding of delusional phenomena more broadly[25].

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