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Fregoli Syndrome: A Rare Delusional Misidentification Syndrome

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ABSTRACT

Fregoli syndrome is a rare delusional misidentification syndrome (DMS), in which a patient believes that one or more familiar persons are disguised as different people. That is, although other persons appear in their normal visual form, the patient is convinced that they are someone familiar in disguise. This syndrome was first described by Courbon & Fail in the year 1927. This article mainly reviews the etiology, neurobiology/neuropathology, differential diagnosis, treatment, gaps & limitations in existing literature, recent findings and future directions of research in the aspect of this syndrome. It spans both psychiatric and neurological domains. Available literature suggests that a substantial portion of cases have secondary (organic) causes, especially in older individuals or those with first episodes, and these are often associated with right hemisphere lesions. Cognitive disturbances (memory, executive function) and anomalies in emotional recognition/familiarity are common. Treatment is empirical with antipsychotics. Clinicians should maintain a high index of suspicion for organic contributors, perform thorough diagnostic workups and individualize treatment. Research remains limited, more systematic, standardized and prospective work is needed in this area.

Introduction

Fregoli syndrome is a rare delusional misidentification syndrome (DMS), in which a patient believes that one or more familiar persons are disguised as different people. That is, although other persons appear in their normal visual form, the patient is convinced that they are someone familiar in disguise. This syndrome was first described by Courbon & Fail in the year 1927 [1].

Epidemiology

As it is a rare syndrome, the estimates of incidence or prevalence are sparse, mostly derived from case reports or series. A recent meta-analysis (Teixeira-Dias et al.) identified 119 cases in the literature. Among stroke populations, a study that includes 874 consecutive patients found with 10 cases of Fregoli syndrome (≈1.1%) following stroke.

No large population based prevalence estimates are available. Demographic data suggest it can appear in adults, in both genders, though some reports hint at older age in cases with secondary/neurological etiologies.

Etiology

Etiologies can be divided into primary psychiatric disorders (schizophrenia, schizoaffective disorders, bipolar disorder, psychotic depression) and secondary/organic causes (neurological damage [stroke, traumatic brain injury, neurodegenerative disorders], epilepsy, systemic illness and sometimes even with medication effects). The meta-analysis by Teixeira-Dias et al found that about 42% of reported cases have a secondary cause [2].

Phenomenology and Clinical Features

Delusional content: The core belief is that someone familiar is disguising themselves as others (sometimes even multiple disguises & multiple persons). Often persecutory features: the "familiar person in disguise" is thought to intend harm or to be following/persecuting the patient. In primary psychosis cases, persecutory delusions are more common whereas in case of secondary psychosis, it is very less. Misidentified persons often are family members or people close to the patient and sometimes strangers are thought to be these disguised familiar persons.

Associated symptoms include hallucinations (often auditory), delusions of other types (paranoid, guilt, grandiosity), cognitive deficits (memory, executive dysfunction, working memory) and even emotional recognition deficits in some cases.

Neurobiology / Neuropathology

Right hemisphere lesions are frequent in secondary cases; particularly in frontal, temporal and insular areas. The uncinate fasciculus (white matter tract connecting temporal pole/anterior temporal areas with frontal lobe) is implicated in stroke cases. Lesion-symptom mapping in post-stroke patients with Fregoli syndrome finds involvement of this tract. Functional imaging in some case reports suggests hypoperfusion in posterior cingulate, prefrontal cortex and retrosplenial cortex (especially in comorbid cases of Capgras associated with Fregoli). Potential mechanisms proposed in literature include disturbances in familiarity recognition and reality monitoring, integration of perceptual information (face identity, appearance) with emotional/memory circuits and disconnection between areas that process identity/appearance vs those that monitor familiarity/emotional significance [3].

Differential Diagnosis

When encountering suspected Fregoli syndrome, health care professionals need to differentiate from other DMS (Capgras, intermetamorphosis, subjective doubles), psychotic disorders (delusional disorder, schizophrenia), neurological disorders (dementia, epilepsy & stroke) and medication/metabolic causes (drug-induced psychosis & metabolic derangements)

Treatment

Because of rarity, there are no randomized controlled trials specific to Fregoli syndrome. Current treatment is based on case reports, general principles and treating underlying cause. Most of the patients responded (partially or fully) to antipsychotics (both typical and atypical). Sometimes cognitive remediation, psychotherapy, possibly ECT in severe or refractory cases especially when mood component or postpartum psychosis is present.

Gaps & Limitations in Existing Literature

- Inconsistent reporting of neuropsychological testing, imaging findings; missing data points.
- Few studies with longitudinal follow up systematically tracking course and response to therapy.
- Lack of standardized diagnostic criteria/assessment tools specific to Fregoli syndrome (vs broader DMS).
- Unclear which cognitive deficits are specific vs which are general to psychosis or secondary neurological disease [4-6].

Recent Findings

Teixeira-Dias et al., provide the first individual patient meta-analysis comparing Fregoli syndrome in primary vs secondary psychosis.

Some of their key findings include:

- In secondary psychosis, more likely to be of older age and more likely to have neuro-imaging abnormalities.
- Persecutory features were observed to be much less frequent in secondary than primary.
- Right-sided lesions more common in total sample and particularly in secondary cases.
- These clarify that Fregoli syndrome is not purely a psychiatric phenomenon, and that clinicians need high suspicion for organic causes, especially in certain demographics [2,3].

Clinical Implications

When a patient presents with Fregoli delusions, workup should include neuroimaging (MRI/CT), neurological assessment, perhaps EEG, metabolic labs etc., to look for secondary etiology. Detailed neuropsychological assessment (memory, facial recognition/emotion, executive function, familiarity judgments) may help both in diagnosis and in monitoring treatment. Treatment should not only focus on suppressing psychosis with antipsychotics, but also treat reversible causes (stroke, injury, metabolic causes) [7].

Future Directions

Some areas where further research is needed:

- Standardized diagnostic criteria and assessment tools for Fregoli syndrome (within DMS).
- Prospective cohort studies with neuroimaging, cognitive profiling and longitudinal follow-up to understand course, predictors and treatment response.

- Neurobiological studies (structural and functional imaging, diffusion tensor imaging etc) focusing on connectivity (e.g. uncinate fasciculus) and emotional processing networks.
- Although case reports are helpful, systematic trials (or at least observational studies with consistent methods) comparing antipsychotics, psychotherapy, neuromodulation etc are required.

Conclusion

Fregoli syndrome is a rare but clinically important delusional misidentification syndrome. It spans both psychiatric and neurological domains. Available literature suggests that a substantial portion of cases have secondary (organic) causes, especially in older individuals or those with first episodes and these are often associated with right hemisphere lesions. Cognitive disturbances (memory, executive function) and anomalies in emotional recognition/familiarity are common. Treatment is empirical with antipsychotics. Clinicians should maintain a high index of suspicion for organic contributors, perform thorough diagnostic workups and individualize treatment. Research remains limited, more systematic, standardized and prospective work is needed in this area.

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