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Case report

Delusional misidentification syndrome and confabulation in schizophrenia: a case report with rare symptoms

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SUMMARY

Confabulation is the unintentional production of false or distorted memories. Delusional Misidentification Syndromes (DMS) are psychopathological phenomena in which familiar people or places are believed to have been altered or replaced. Both are rare but clinically significant, and they may co-occur in schizophrenia. We present the case of a 27-year-old male with treatment-resistant schizophrenia who exhibited both confabulation and Fregoli syndrome. The patient produced shifting autobiographical narratives, including multiple identities, invented family members, and fantastical life events, alongside delusions that his father was an impostor in disguise. Neuropsychological testing revealed attentional and memory impairments with signs of frontal lobe involvement. Despite high-dose clozapine and amisulpride treatment, only partial improvement was achieved, and electroconvulsive therapy contributed to further symptom reduction. The patient's PANSS score decreased from 119 to 76 after electroconvulsive therapy, indicating a marked clinical improvement. This case highlights the complex interplay between cognitive deficits, confabulation, and delusional misidentification in schizophrenia, emphasizing the importance of comprehensive assessment and individualized treatment approaches in managing rare psychopathological presentations.

Key words: confabulation, delusional misidentification syndrome, electroconvulsive therapy, fregoli syndrome, schizophrenia

Introduction

Confabulation is the production of false or distorted memories, or personal narratives, without any deliberate intention to deceive. Patients who confabulate are typically unaware that the information they are recalling or conveying is inaccurate, and they sincerely believe it to be true. Confabulations can range from minor distortions of actual events to entirely fabricated and often elaborate or bizarre memories. Confabulation was first described by Korsakoff in 1889 in patients with amnestic alcohol-related disorders. Although previously reported in neurological disorders such as anterior communicating artery aneurysm, Alzheimer's disease, and traumatic brain injury, it has also been reported in individuals with schizophrenia [1-3]. Although it is currently often overlooked in clinical evaluation, in 1971 Kraepelin described "paraphrenia confabulans" in patients with schizophrenia who present numerous extraordinary stories or accounts, which they present as actual personal experiences [3].

Delusional Misidentification Syndromes (DMS) are rare psychopathological phenomena encountered in psychiatric and neurological conditions. The patient believes that familiar people, places, objects, or events have changed or have been duplicated. These syndromes include Capgras syndrome, intermetamorphosis syndrome; subjective doubles syndrome; and Fregoli syndrome in which the patient describes an individual, most

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often unknown to the patient, is now someone he knows "in disguise", and the individual is thought to be pursuing or persecuting him in some way [4]. They are more common in males and may accompany paranoid symptoms [5]. DMS and confabulation frequently co-occur, with patients often using confabulation to explain DMS symptoms related to their current environment or significant others [11].

In this article, we present a case of a patient diagnosed with schizophrenia who simultaneously exhibited symptoms of confabulation and DMS. The co-occurrence of these two rare phenomena makes this case valuable for understanding the relationship between Fregoli syndrome, confabulation, and schizophrenia, as well as the clinical considerations for treatment and management.

Informed consent was obtained from the patient's legal-guardian.

Case Presentation

T.D. is a 27-year-old single male who dropped out of middle school, currently unemployed and lives with his father. The patient's father was appointed as his legal guardian. The patient's biological parents divorced when he was five years old, after which he began living with his mother and stepfather. He later attended a boarding school until dropping out during middle school, after which he resided with his father. He is currently not in contact with his mother. He was admitted to our inpatient psychiatric unit in July 2023 due to emerging symptoms of suspiciousness and physically aggressive behavior toward his father. His complaints had started approximately two weeks prior. The patient believed that his father was not his real father and that he had changed his face. Since 2018, the patient has had multiple hospitalizations, initially due to substance-induced psychotic disorder. During the most recent admission, the patient was diagnosed with non-organic psychotic disorder. The patient reportedly began using synthetic cannabinoids 13 years ago and had been using methamphetamine for the past five years. At the time of his most recent admission, he had not used psychoactive substances for five months. His treatment adherence and response were considered poor based on his illness history. No known physical illness was reported. Cranial MRI and EEG results showed no pathology.

On psychiatric evaluation, he was conscious, cooperative, and oriented. His self-care was poor; he was reluctant to elaborate on his thought content. His mood was irritable, and his affect was angry. His associations were loose, and he had difficulty goal-directing his thoughts. He exhibited persecutory delusions but did not report hallucinations. His judgement and abstract thinking were impaired. He lacked insight into his condition.

In the initial interview, the patient reported that his father was not his biological parent, but a garbage collector who was following him, breaking into their house, and disguising himself by wearing his father's clothes and altering his face. In subsequent interviews, he claimed his father lived abroad, was named "Jesus Christ," or was currently in prison. Confabulatory content was documented through daily psychiatric interviews. When asked for his name at different times, he responded with other male names like "Can," "Sakıp", or sometimes his real name. He sometimes stated that when his parents divorced, both his and his father's names were changed, and he does not remember his mother's name. When asked where he was from, his answers included "Australia," "Tokat," "England – an outlaw country," "the sultan monkey country," or "sultan ocean." In other sessions, he claimed to be married, to have twelve children, and that all of them were named "Menekşe." These false autobiographical statements were evaluated as reflecting both spontaneous and provoked confabulations, while his beliefs about his father were interpreted as Fregoli Syndrome.

In subsequent psychiatric assessments, spontaneous confabulation was consistently observed. The patient also gave different answers at different times about where he lived, claiming to have houses in various countries. He also claimed to have met the examining psychiatrist during military service and to have close ties with the president, who, he said, would come to discharge him from the hospital.

The patient also underwent comprehensive neuropsychological assessment. A neuropsychological test battery was administered, including: the Mini-Mental State Examination (MMSE), Digit Span Test (for attention), the List Learning subtest from the Screen for Cognitive Impairment in Psychiatry (SCIP) for verbal memory assessment; the Visual Reproduction subtest from the Wechsler Memory Scale, the Block Design test for non-verbal memory; the Stroop Test, Clock Drawing Test, and Verbal Fluency Tests for executive function evaluation. Results revealed that the patient's attention and ability to sustain attention (5 direct and 3 reverse-scored items) were below normal limits. Verbal Fluency Test results showed that the patient was able to retain 7 out of 10 verbal items after the third trial. After 10 minutes, he could recall 4 items spontaneously and recognize 6 items upon prompting. The total learning score was calculated as 18 out of 30. Neuropsychological assessment revealed mild verbal memory impairment and moderate nonverbal memory deficits, accompanied by signs of frontal lobe involvement. Nonverbal memory impairment was observed in both encoding and retrieval phases, while visuospatial functions, construction abilities, and planning ability were preserved within normal

limits. The verbal memory deficit was consistent with a frontal-type impairment, characterized by impaired spontaneous recall but intact recognition, whereas non-verbal memory difficulties affected both recall and recognition processes.

Pharmacological treatment was initiated with 20 mg/day intramuscular haloperidol and 10 mg/day intramuscular biperiden. Based on clinical findings and history, the patient was considered to have treatment-resistant schizophrenia. Clozapine was initiated and titrated up to 500 mg/day, while haloperidol was tapered and discontinued. After one month, due to insufficient treatment response, augmentation with amisulpride was initiated and increased to 800 mg/day. As the response remained inadequate after another month, Electroconvulsive Therapy (ECT) was administered, with a total of 8 sessions. Following these interventions, the patient's confabulations, delusions, and DMS symptoms partially improved. He did not mention these symptoms spontaneously and showed no apparent preoccupation with them. His Positive and Negative Syndrome Scale (PANSS) score was 119 at admission and decreased to 76 immediately after the completion of ECT. Although the patient showed marked clinical improvement, neuropsychological reassessment was not performed after ECT. Table I presents the pharmacological treatment and associated cognitive changes. At the time of discharge, he stated that he was not married but might have experienced something like a marriage in the past, that he had no children, and that he had never traveled abroad. Given the patient's poor adherence to oral medication and limited social support, long-term institutional care was deemed appropriate. He was discharged on clozapine 500 mg/day and amisulpride 800 mg/day.

Discussion

Delusional Misidentification Syndromes (DMS) are known to present across a range of psychiatric disorders, including schizophrenia, schizophreniform disorder, brief psychotic disorder, delusional disorder, and major depressive disorder [5]. In the present case, DMS symptoms were assessed under the *delusions* criterion of schizophrenia, as defined by the DSM-5. DMS is considered a risk factor for aggression, as patients may direct violent behavior toward individuals they misidentify as persecutors, with manifestations ranging from verbal threats to, in rare cases, homicidal actions [10]. Similarly, our patient exhibited homicidal behavior toward his father, which appeared to stem from DMS-related delusions and led to his hospitalization. The risk of homicide and the forensic relevance associated with DMS are of particular importance in patients with schizophrenia. It should be noted that structured risk assessment plays a crucial role in managing psychotic patients with persecutory or misidentification delusions [14].

Two types of confabulation are described: provoked and spontaneous. Provoked confabulations occur in response to questioning and are typically plausible, while spontaneous confabulations – more commonly seen in schizophrenia – involve bizarre, implausible autobiographical claims that arise unprompted [10]. In line with previous findings, misidentification of a parent has been reported to occur more frequently in psychiatric conditions compared to organic delusional disorders, as observed in our case [16].

Four main theories have been proposed to explain the mechanism of confabulation. The gap-filling theory suggests that confabulation serves to fill in missing information or avoid embarrassment and may even occur in healthy individuals. The source monitoring theory attributes it to deficits in identifying the origin of memories, while the temporality theory explains it as an inability to suppress irrelevant, activated memory traces, leading to temporal confusion. The retrieval theory – empirically the most supported – proposes that confabulation arises from failures in memory retrieval and monitoring, whereby inaccurate information is retrieved and not recognized as false. In schizophrenia, the underlying mechanisms are thought to involve executive dysfunction, source monitoring errors, and impairments in long-term memory [2,6-8]. Evidence further suggests that confabulation in schizophrenia is more closely linked to semantic rather than episodic memory and may reflect a disorder of thought processing [7-9]. In addition, recent advances in neuroscience highlight the importance of addressing neurobiological perspectives. Brain clearance mechanisms, such as the glymphatic system, as well as network-level dysfunction, may play a role in confabulatory processes, particularly in the presence of psychiatric comorbidities [15]. Beyond the mechanisms underlying confabulation, some reports focus on how

TABLE I. *Pharmacological Timeline and Clinical Progression.*

Initial Treatment	
Haloperidol 20 mg/day, biperiden 10 mg/day	PANSS 119,severe psychotic symptoms and confabulations
Clozapine Phase	
Clozapine 500 mg/day	Partial clinical improvement; reduced disorganization and confabulatory content
Augmentation Phase	
Clozapine 500 mg/day, amisulpride 800 mg/day	Further clinical improvement; stabilization of affect and reduction of delusional intensity
ECT Phase	
8 sessions of ECT	Marked clinical improvement; PANSS 76

its content is determined. These accounts indicate that confabulations often contain misattributed fragments of real experiences and are shaped by emotionally significant factors such as personal meaning or wish-fulfillment. The content is also thought to reflect motivational needs, including the preservation of self-image or coping with anxiety through grandiose or self-enhancing themes [12].

The relationship between confabulation and delusion remains a subject of debate. Some theorists propose a shared etiology, while others argue that they arise from distinct mechanisms based on their differing characteristics and origins. Some clinicians in the past, such as Korsakoff, defined the concept of delusion broadly enough to encompass false memories. In some cases, delusions may be rooted in either true or false memories. When based on a false memory, a delusion may become indistinguishable from confabulation. These rare cases, referred to as delusional confabulations, involve bizarre, shifting narratives that grow more elaborate during clinical interviews [13].

While antipsychotics remain the first-line treatment for DMS, ECT may offer symptom reduction in treatment-resistant cases. However, DMS symptoms often persist despite therapy and are among the most frequently recurring psychotic features in schizophrenia [5,6]. The severity of confabulation has been associated with overall illness severity, and its treatment should be tailored to the underlying etiology and the individual characteristics of the patient, potentially involving antipsychotic medications and/or ECT, depending on symptom severity and treatment response [7,9]. Treatment-resistant schizophrenia (TRS) is defined as the persistence of significant psychotic symptoms and functional impairment despite adequate trials of at least two different antipsychotic medications administered at appropriate doses and durations, with verified adherence. Since the patient exhibited only partial response to clozapine, he was further evaluated as meeting criteria for ultra–treatment-resistant schizophrenia (UTRS), which denotes suboptimal or absent therapeutic response following an adequate clozapine trial [17]. In our case, multiple psychiatric hospitalizations, poor response to clozapine and amisulpride, and only partial improvement with ECT indicated a severe disease course; although full remission was not achieved, the patient's symptoms improved substantially. During follow-up evaluations, the patient did not report any DMS or confabulatory symptoms, and when asked about the previously described content, he was unable to recall those experiences

In our case, the patient's grandiose and fantastical narratives about himself and his family reflected both spontaneous and provoked confabulations. The patient's confabulations appear to be deeply intertwined with his personal history and emotional experiences. The shifting and often contradictory narratives regarding his father's identity – ranging from a non-biological figure to a man named "Jesus Christ" or a prisoner – along with his inconsistent self-identification, suggest that these confabulations may serve a psychological function. Given his disrupted family background, including parental divorce, changing caregivers, and lack of contact with his mother, the confabulated content likely reflects an attempt to cope with feelings of abandonment, instability, and identity confusion. The grandiose and fantastical elements, such as claiming to be married with twelve children all named "Menekşe", may represent wish-fulfillment or efforts to preserve a positive self-image amidst adversity. Thus, consistent with existing literature, the patient's confabulations not only incorporate distorted fragments of his real experiences but are also shaped by his emotional needs and motivational drives to manage anxiety and construct a coherent self-narrative. Moreover, childhood trauma, exemplified by parental divorce and early maternal separation, likely contributed to the development of his confabulatory and misidentification phenomena by disrupting reality-attribution processes and increasing vulnerability to aberrant salience and delusional content formation through altered dopaminergic regulation [18].

The neuropsychological profile of the patient, characterized by frontal lobe involvement and distinct verbal and nonverbal memory impairments, provides insight into the possible mechanisms underlying his confabulations. According to existing theories, confabulation can arise from deficits in memory retrieval, monitoring, and source identification, all of which are functions commonly associated with the frontal cortex. The patient's impaired spontaneous recall alongside preserved recognition suggests a disruption in retrieval processes rather than a global memory failure. This aligns with the retrieval theory, which emphasizes failures in monitoring retrieved information as a core mechanism of confabulation. Additionally, the moderate nonverbal memory deficits affecting both encoding and retrieval further indicate difficulties in properly forming and accessing accurate memory traces. In the context of schizophrenia, where executive dysfunction and source monitoring errors are prominent, the patient's confabulations likely reflect these intertwined cognitive disturbances. Moreover, the link between confabulation and semantic memory dysfunction, rather than episodic memory, may explain the patient's fabricated yet thematically consistent false memories, underscoring confabulation as a disorder of higher-order cognitive processing rather than mere memory loss.

Conclusion

Confabulation remains a challenging symptom for psychiatrists to identify and manage, particularly in complex cases involving schizophrenia and DMS. Accurate assessment requires careful verification of patient-reported information through collateral sources such as family members or caregivers. The presence of confabulation may signal a treatment-resistant course, necessitating adjustments in therapeutic strategies. Additionally, recognition of DMS during clinical evaluation is crucial, as these syndromes can heighten the risk of aggression or violence toward familiar individuals, warranting proactive risk management and tailored interventions. While much of our understanding of confabulation derives from neurological conditions, its manifestation in psychiatric disorders like schizophrenia underscores the need for further research to clarify shared and distinct underlying mechanisms.

However, this single-case study limits the generalizability of the findings and precludes causal inferences. The absence of longitudinal cognitive follow-up and functional neuroimaging also restricts the evaluation of cognitive change and underlying neural mechanisms. Future studies involving larger samples, longitudinal cognitive assessments, and multimodal neuroimaging are needed to clarify these relationships.

Overall, this case highlights the importance of comprehensive assessment and multidisciplinary treatment approaches in addressing the intertwined phenomena of confabulation and DMS to improve patient outcomes.

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Conflict of interest statement

The authors declare no conflict of interest.

Authors' contributions

HIE, NÇT, YEY, and ÖDB made the psychopathological assessment. HIE and NÇT drafted the manuscript. ÖDB revised the psychopathological assessment and the discussion. All authors approved the final version of the manuscript.

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