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Simultaneous Onset of Schizophrenia in Two Sisters: A Case Report

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Abstract

Introduction: Family, adoption, and twin studies have consistently demonstrated a strong heritable component of schizophrenia, with a significantly increased relative risk among first-degree relatives of patients with schizophrenia. The simultaneous onset of schizophrenia among family members is uncommon but clinically significant.

Case Presentation: Two sisters aged 32 and 25 years, with no psychiatric history, were admitted for severe aggression and psychotic symptoms that began after family stressors. Both developed grandiose, persecutory, and bizarre delusions, including a prophetic identity, supernatural pregnancies, vast inheritances, and Capgras syndrome, in which they believed their parents were impostors. The younger sister also experienced auditory and visual hallucinations. Physical examination and laboratory findings were normal. Despite treatment with risperidone, haloperidol, and six electroconvulsive therapy sessions, no improvement was observed until clozapine was initiated, leading to a marked reduction in delusions and aggression. Both sisters were discharged on clozapine 150 mg/day and maintained remission at the six-month follow-up.

Conclusions: Genetic predisposition and shared stressors may have contributed to schizophrenia in two sisters. Similar symptoms and persistent psychosis after separation supported independent diagnoses rather than an induced psychosis. This case highlights the importance of careful diagnosis, early identification of treatment resistance, and prompt initiation of clozapine in refractory cases. Familial clusters of psychosis illustrate how biological vulnerability may be exacerbated by isolation and enmeshment.

Keywords: Shared Psychotic Disorder, Schizophrenia, Sibling Relationships

1. Introduction

Schizophrenia is a chronic, debilitating psychotic disorder that imposes a substantial burden on individuals, families, and health care systems. Global estimates suggest that the lifetime prevalence of psychotic disorders is approximately 0.7%, and the point prevalence of schizophrenia is relatively low but clinically significant. Despite its low prevalence, the social, economic, and clinical impact of this disorder remains profound (1).

Family, adoption, and twin studies have consistently demonstrated a strong heritable component, with a significantly increased relative risk among first-degree relatives. These findings have been corroborated by large-scale genome-wide association studies that have identified more than 100 loci associated with schizophrenia. However, the broad phenotypic variability, polygenic architecture, and influence of environmental factors, such as migration, urbanization, and social stressors, indicate that schizophrenia arises from a complex interaction between genetic vulnerability and environmental exposures (2, 3).

The simultaneous onset of psychosis among family members is uncommon but clinically and pathophysiologically important. It may represent a spectrum ranging from independent familial co-occurrence to shared psychotic disorder, also known as folie à deux or folie à trois. In the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR), shared psychotic disorder is no longer recognized as an independent diagnosis. Instead, it is subsumed under "Other Specified Schizophrenia Spectrum and Other Psychotic Disorder," with examples including delusional symptoms occurring in a close associate of an individual with psychosis (4, 5). Based on

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admissions to psychiatric hospitals, the incidence rate of shared psychotic disorder has been reported to range from 1.7% to 2.6% (6). Prevalence varies across age groups and sexes; however, approximately 90% of cases involve couples, siblings, or parent-child pairs. Schizophrenia is likely the most common diagnosis in the primary case, whereas other diagnoses include delusional disorder and mood disorder with psychotic features (7).

Differentiating between the simultaneous onset of independent schizophrenia in two sisters and the transmission of delusional beliefs within a close relationship is essential for etiologic understanding, treatment planning, and prognosis. The present report describes two sisters who presented with symptoms consistent with schizophrenia at approximately the same time. It focuses on the temporal sequence of symptom onset, familial and environmental risk factors, paraclinical findings, differential diagnoses, particularly the distinction from folie à deux, and treatment considerations, including separation, antipsychotic medication management, and family-based follow-up.

2. Case Presentation

Two sisters, aged 32 and 25 years, with no prior history of psychiatric illness, substance use, or head trauma, were brought by their parents to the emergency department of a psychiatric hospital. In both sisters, psychotic symptoms began approximately two years before admission, following family stressors, including inheritance disputes and the elder sister's broken engagement. Symptoms gradually intensified over time, leading to increasing verbal and physical aggression toward family members.

The elder sister had persecutory and grandiose delusions, severely impaired judgment, and poor insight. Her thought process was tangential, with prominent functional decline and marked impairment in interpersonal and social functioning. The younger sister also showed poor insight, prominent delusional beliefs, and multimodal hallucinations, including auditory, visual, and tactile phenomena. Her affect was restricted, and her daily functioning was significantly compromised. Both sisters presented with a euthymic mood.

Both sisters exhibited grandiose and persecutory delusions. They claimed prophetic status and reported supernatural pregnancies with multiple fetuses, asserting that they had the ability to deliver numerous children in a single labor. Additional fixed false beliefs involved possession of vast inherited properties, the existence of unseen husbands who were inaccessible to others, and a conspiracy theory alleging that the Jewish community was attempting to abduct Muslim children while falsifying their hospital laboratory results. Both sisters exhibited a Capgras-like delusion, insisting that their parents had been replaced by impostors, which fueled escalating aggression and led to the parents' expulsion from the home. The younger sister also reported multimodal visual, auditory, and tactile hallucinations involving her purported husband.

The father had a mild intellectual developmental disorder without psychotic symptoms. The family was socially isolated. The sisters were exceptionally close and emotionally enmeshed. The elder sister was college educated. The younger sister had limited schooling, had been divorced for five years, and had a child living with her ex-husband.

Initial physical and laboratory examinations, including toxicology testing and brain magnetic resonance imaging, revealed no abnormalities.

The diagnosis of schizophrenia in each sister was based on DSM-5-TR criteria, including prominent delusions, hallucinations, disorganized behavior, functional decline, and persistence of symptoms over time. In the elder sister, the clinical picture was dominated by fixed persecutory, grandiose, and bizarre delusions with marked behavioral disorganization and functional impairment. In the younger sister, schizophrenia was supported by similar delusional content, multimodal hallucinations, impaired insight, and significant dysfunction.

Following admission, the sisters were separated into different wards to evaluate the possibility of induced or shared psychosis; however, psychotic symptoms persisted in both patients without meaningful improvement. Sequential trials of risperidone and haloperidol were administered. Risperidone was administered for 4 weeks, with gradual dose escalation to 12 mg/day, followed by the addition of haloperidol for 4 weeks at up to 10 mg/day, without meaningful clinical improvement.

Despite adequate trials of antipsychotic medications, both patients continued to exhibit severe psychotic symptoms, behavioral disorganization, and episodes of aggression toward family members. Given the severity of the clinical presentation and the limited response to pharmacologic treatment, electroconvulsive therapy (ECT) was initiated as an adjunctive intervention. However, after six ECT sessions, no meaningful clinical improvement was observed, and treatment-resistant psychosis was considered. Consequently, clozapine therapy was initiated.

Following the lack of meaningful response to previous antipsychotic trials and ECT, clozapine

treatment was initiated in both patients. Clozapine was started at a low dose of 12.5 - 25 mg/day and gradually titrated over several days to minimize adverse effects. The dosage was progressively increased under close clinical monitoring until a maintenance dose of 150 mg/day was achieved in each patient, resulting in clinically meaningful improvement in psychotic symptoms and behavioral stabilization. The final dose was determined based on therapeutic response and tolerability. Both sisters demonstrated meaningful improvement, although the younger sister's hallucinations and agitation appeared to respond earlier than the elder sister's predominantly delusional symptoms.

Regular hematologic monitoring was conducted in accordance with standard clozapine safety protocols, including routine complete blood count assessments to monitor for neutropenia or agranulocytosis.

At the six-month follow-up, the older sister achieved partial remission, marked by reduced psychotic symptoms and aggression, improved behavior, and restored daily functioning, with intermittent delusions. The younger sister experienced complete remission, characterized by the absence of hallucinations, a significant reduction in delusions, and functional improvement.

Therapeutic response was assessed through longitudinal clinical evaluation by the treating psychiatric team rather than through a formal structured rating scale. Clinical improvement was determined based on reductions in delusional intensity, resolution of aggressive behavior, decreased hallucinations, improved behavioral organization, and better overall functioning during hospitalization and follow-up.

Throughout the treatment course, both patients were carefully monitored for potential adverse effects related to pharmacologic therapy and ECT. No serious adverse events were observed during treatment with risperidone, haloperidol, or ECT. Clozapine was generally well tolerated, and no clinically significant hematologic or cardiovascular complications were detected during treatment or follow-up.

3. Discussion

In the present case, several clinical features were clearly observed, including the simultaneous onset of psychotic symptoms in two sisters, complex delusional beliefs, behavioral disorganization, aggression, and persistence of symptoms despite separation. These observations suggest a severe psychotic disorder consistent with schizophrenia-spectrum pathology.

Several mechanisms may help explain this unusual presentation. Genetic vulnerability, shared environmental stressors, and the sisters' close interpersonal relationship may have contributed to the emergence and parallel development of psychotic symptoms. However, these factors should be interpreted as potential explanatory hypotheses rather than definitive causal mechanisms.

When psychosis occurs in genetically related individuals, distinguishing shared psychotic disorder, or folie à deux, from independent schizophrenia is challenging. Both patients were diagnosed with schizophrenia based on DSM-5-TR criteria, including persistent delusions, positive symptoms, disorganization, and functional decline, after alternative diagnoses were considered.

The differential diagnoses considered included shared psychotic disorder, mood disorder with psychotic features, delusional disorder, substance-induced psychosis, and neurologic causes. Shared psychotic disorder was considered less likely because symptoms remained persistent and equally severe after separation. Mood disorder with psychotic features was considered less likely because of the absence of a clear mood episode. Delusional disorder was considered less likely because of the presence of hallucinations, disorganization, and functional impairment. Substance-induced psychosis was unlikely given the negative history and toxicology results. Neurologic causes were also considered less likely because of unremarkable magnetic resonance imaging findings and other investigations and the absence of neurological signs.

Historically, the term folie à deux described shared psychotic phenomena in closely related individuals. In earlier diagnostic systems, this condition was also referred to as shared psychotic disorder. However, in contemporary classifications such as DSM-5-TR, this entity is no longer considered a distinct diagnostic category and is generally conceptualized within the spectrum of psychotic disorders or as part of other specified schizophrenia spectrum and other psychotic disorders. In the present report, both patients were diagnosed with schizophrenia according to DSM-5-TR criteria. Concepts of shared or induced psychosis are discussed only as differential considerations in the context of simultaneous symptom onset.

This aligns with findings indicating that schizophrenia is the most frequent diagnosis in primary cases of shared psychotic phenomena (7). The sisters' shared onset, linked to psychosocial stressors such as

inheritance disputes and relational conflicts, suggests an interaction between genetic predisposition and environmental triggers in their schizophrenia. Although schizophrenia has a strong heritable component, with substantially increased risk among first-degree relatives and heritability estimates of 70% - 80% (2, 5), environmental factors, including social isolation and family dynamics, influence the expression of genetic vulnerability. In this instance, the family's isolation and the sisters' close bond may have amplified psychotic themes, while shared stressors acted as triggers.

Clinically, both sisters exhibited grandiose and persecutory delusions, supernatural beliefs, and Capgras syndrome involving misidentification of their parents. The younger sister's multimodal hallucinations further support an underlying psychotic disorder rather than suggestibility-based shared delusions. Capgras syndrome has been described in familial psychotic disorders and is considered an indicator of more severe thought disorganization and underlying schizophrenia rather than secondary delusional contagion (8).

A notable similarity exists between the present case and that reported by Knezevic et al. (8). In both reports, the affected individuals were sisters who shared a close emotional bond and developed psychosis within the same household. Each case involved Capgras-type misidentification delusions directed toward family members, leading to aggression and severe family conflict. Both studies emphasized that the persistence of symptoms after separation and the complexity of delusional content supported a diagnosis of independent schizophrenia rather than an induced psychotic disorder (8).

The lack of response to conventional antipsychotics and ECT underscores the severity and treatment resistance often observed in familial schizophrenia (9). Clozapine administration produced marked improvement in both cases, consistent with its documented efficacy in refractory psychosis. These outcomes highlight the importance of early recognition of treatment resistance and timely initiation of clozapine to prevent chronicity and functional decline.

Historical evidence, such as the report by Solomon and Bliss (10) of concurrent schizophrenia onset in monozygotic twins, suggests a strong genetic influence on the timing and expression of psychosis, even in the absence of interpersonal factors. This case of non-twin siblings with simultaneous psychosis mirrors these findings, suggesting that shared genetic predisposition, amplified by shared psychosocial stressors, can synchronize illness expression.

The clinical presentation highlights how shared stressors and emotional interdependence can amplify risk in genetically vulnerable individuals. Effective treatment should combine medication with family psychoeducation, reduced expressed emotion, and sustained social support to prevent relapse and the mutual reinforcement of psychotic beliefs.

In summary, this case supports the conceptualization of schizophrenia as a disorder arising from the intersection of genetic vulnerability and psychosocial adversity. The simultaneous onset in siblings reflects both shared biological risk and environmental synchrony. Differentiation from folie à deux is crucial, as the persistence of psychotic symptoms following separation and the favorable response to clozapine support an independent and biologically driven psychotic process rather than an induced delusional state.

3.1. Limitations

This report has several limitations that should be acknowledged. First, as a case report involving two patients from the same family, the findings cannot be generalized to broader populations. Second, symptom severity and treatment response were assessed primarily through clinical evaluation rather than standardized rating scales, which may limit the objectivity of outcome assessment. Third, although the simultaneous onset of psychotic symptoms in the two sisters raises questions regarding shared environmental or genetic vulnerability, the design of this report does not allow causal conclusions to be drawn. Finally, additional biological, genetic, or longitudinal data were not available, which restricts deeper exploration of potential etiological mechanisms.

Footnotes

AI Use Disclosure: The authors declare that no generative AI tools were used in the creation of this article.

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