CASE REPORT

Psychotic Symptoms Mimicking Schizophrenia in an Adolescent with Clinically Suspected Frontal Lobe Epilepsy: A Case Report

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ABSTRACT

Background: Psychosis is a recognized complication of epilepsy, but psychosis-dominant presentations of frontal lobe epilepsy (FLE) are infrequently reported and FLE-specific prevalence is poorly quantified. A systematic review of psychiatric manifestations of FLE identified only 17 published patients whose primary presentation was psychosis among 66 reported FLE cases with psychiatric manifestations. This case illustrates the diagnostic uncertainty that arises when schizophrenia-like psychosis coexists with paroxysmal motor symptoms but routine electroencephalography (EEG) is normal. ***Case:*** A 19-year-old woman presented with recurrent auditory, visual, and olfactory hallucinations, bizarre grandiose delusions, behavioral disorganization, and fluctuating functional impairment. She also reported brief sudden body jerks, particularly at rest or sleep onset. Routine interictal scalp EEG showed no epileptiform abnormalities. Brain CT/MRI and prolonged video-EEG were not performed. The motor and episodic behavioral phenomena raised clinical suspicion of focal epilepsy of possible frontal origin, but electroclinical localization could not be confirmed. Treatment with risperidone and phenytoin was followed by clinical improvement; because both were administered, treatment response was considered supportive but not diagnostic. ***Discussion:*** FLE may have diverse behavioral and motor manifestations, and scalp EEG may be normal or misleading. However, olfactory phenomena are not specific to the frontal lobe and are more often associated with medial temporal networks, while sleep-onset jerks may be nonspecific. Therefore, the present case is best described as psychosis with clinically suspected focal epilepsy of possible frontal origin rather than confirmed FLE. ***Conclusion:*** In adolescents and young adults with atypical or fluctuating psychosis accompanied by paroxysmal neurological symptoms, focal epilepsy should remain in the differential diagnosis even when routine EEG is normal. Confirmation and localization require correlation of stereotyped events with prolonged video-EEG and, when feasible, epilepsy-protocol brain MRI.

Keywords: Frontal Lobe Epilepsy; Epilepsy-Related Psychosis; Schizophrenia-Like Psychosis; Adolescent Psychiatry

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INTRODUCTION

Psychotic disorders are important neuropsychiatric complications of epilepsy and can closely resemble primary psychotic disorders such as schizophrenia. Meta-analytic and multicenter data indicate that psychosis is substantially more common in people with epilepsy than in the general population, with reported estimates varying according to the population studied and the definition of ictal, postictal, and interictal psychosis[1,2]. This overlap is particularly challenging in adolescents and young adults, in whom a first or recurrent psychotic episode may initially be evaluated within a primary psychiatric framework.

The epidemiology of psychosis specifically in frontal lobe epilepsy (FLE) is much less certain than the overall epidemiology of psychosis in epilepsy. In a cohort of 710 adults with epilepsy, interictal psychosis was identified in 20.65%; among patients with interictal psychosis, 50% had temporal lobe epilepsy (TLE) and 42.47% had FLE[3]. These percentages describe the distribution of epilepsy types among patients with psychosis and should not be interpreted as the prevalence of psychosis within all patients with FLE. A systematic review of psychiatric manifestations of FLE found only 35 published reports describing 66 individual cases over 25 years; psychosis was the primary manifestation in 17 cases[4]. Earlier comparative work likewise emphasized that psychosis in FLE has been studied far less extensively than psychosis in TLE[5]. Thus, schizophrenia-like psychosis associated with FLE is better characterized as an uncommon and underreported presentation with poorly defined lobe-specific prevalence rather than by a precise prevalence estimate.

Epilepsy-related psychosis may occur in temporal relation to seizures as ictal, postictal, or interictal phenomena and can include hallucinations, delusions, behavioral disorganization, affective changes, and other psychiatric symptoms[6,7]. The underlying biology is likely multifactorial, involving dysfunction of fronto-limbic networks, altered excitatory-inhibitory balance, epileptiform network activity, neurotransmitter disturbances, and structural or functional brain abnormalities[8,9]. Because these manifestations can overlap with schizophrenia-spectrum disorders, clinical interpretation requires attention to symptom chronology, stereotypy, neurological accompaniments, and objective epilepsy investigations.

Seizure semiology can provide localizing clues, but no single symptom reliably establishes a frontal, temporal, or parietal onset. Frontal seizures may be brief, abrupt, clustered, and sleep-related with prominent tonic or hyperkinetic motor behavior, while temporal seizures more often feature cognitive, autonomic, experiential, or oroalimentary/manual automatisms; parietal seizures commonly begin with somatosensory, vestibular, or body-perception phenomena[10,11,12]. Olfactory symptoms are particularly important in the present case because they occur most often with medial temporal seizures but can also arise from orbitofrontal or insular regions and therefore do not localize the seizure focus by themselves[13].

A normal routine interictal EEG also does not exclude epilepsy. Reviews indicate that an initial routine EEG may fail to record interictal epileptiform discharges in approximately 47-50% of patients with epilepsy, and scalp EEG can be especially normal or misleading in FLE because deep or rapidly propagating frontal discharges may be difficult to detect[14,15]. When clinical suspicion persists, repeated or sleep-deprived EEG, prolonged video-EEG monitoring, and epilepsy-protocol MRI can substantially strengthen diagnostic and localization confidence[15].

Accordingly, the diagnosis of epilepsy should integrate a detailed event history and semiology with EEG and neuroimaging rather than infer localization from psychiatric symptoms or medication response

alone[11,12,13]. Structured psychiatric assessment remains equally important because psychosis and epilepsy can coexist, and neither an episodic course nor a normal routine EEG independently distinguishes epilepsy-related psychosis from a primary psychotic disorder[7,16].

We present this case because it demonstrates a clinically important diagnostic boundary: a 19-year-old woman with schizophrenia-like multimodal psychosis also reported paroxysmal motor events, yet routine EEG showed no epileptiform abnormality and no neuroimaging or prolonged video-EEG was available. The case therefore illustrates why FLE may be considered in the differential diagnosis while also showing why the more defensible formulation is clinically suspected focal epilepsy of possible frontal origin, not electroclinically confirmed FLE.

CASE

A 19-year-old female patient was admitted to the emergency room of a psychiatric hospital in Central Java, Indonesia, for her uncontrollable screaming, verbal aggression, and bizarre behaviors at home. The patient's condition had been deteriorating for the past two weeks, as indicated by the patient and her family, following a stressful psychosocial event concerning delayed graduation documentation from school.

History of Present Illness

Two weeks before admission, the patient became increasingly withdrawn and isolated, spent most of her time at home, refused food, and showed poor personal hygiene. Her relatives observed her talking to herself, smiling inappropriately, bathing for prolonged periods, and making apparently purposeless hand movements. One week before admission, she reported a voice commenting on her behavior and at times giving commands. She also described vague, colorful shadow-like visual experiences with supernatural interpretations.

Three days prior to admission, she reported recurrent foul-smelling olfactory experiences resembling rotten food despite no apparent external source. Two days prior to admission, she became acutely agitated and screamed loudly. She expressed bizarre grandiose and identity-related delusions, including the belief that she was God and had special powers. She additionally reported sudden brief body jerks as she was falling asleep. The available history did not document a consistently stereotyped sequence linking the psychotic symptoms, olfactory experiences, and motor jerks within the same discrete seizure event.

Past Psychiatric and Medical History

The patient's psychiatric symptoms were first noted in 2021, during her first year in vocational high school, with symptoms of social withdrawal, inappropriate laughter, and insomnia. She had a history of recurrent psychiatric episodes since 2022, with improvement of symptoms with compliance to treatment and worsening with non-compliance to treatment. She was able to complete her education and graduated, with no signs of cognitive deterioration or worsening of her symptoms.

Her medical history revealed a motorcycle accident in 2019 with frontal head trauma that required suturing but no hospitalization. She had recurrent frontal headaches since then. She had no history of drug use. Her family history revealed a sibling with febrile seizures in childhood.

Mental Status and Physical Examination

On examination, the patient was alert and cooperative. There was normal psychomotor activity. Her mood was dysphoric with constricted affect. Thought processes were organized but non-realistic, with the presence of bizarre delusions and poor reality testing. She had auditory hallucinations (commenting and commanding), visual hallucinations, and

olfactory hallucinations. Insight was poor. There were no neurological deficits.

Investigations

Routine laboratory investigations showed leukocytosis (22.43 x 10^3/µL), which decreased with antibiotic therapy. Liver and kidney function tests were within normal limits. Routine interictal scalp EEG showed a normal recording without epileptiform discharges. No epileptiform wave or electrographic seizure was demonstrated. Brain CT or MRI was not performed during this evaluation, and the patient did not undergo prolonged video-EEG monitoring.

Assessment and Differential Diagnosis

Several differential diagnoses were considered, including schizophrenia spectrum disorder, brief psychotic disorder, organic psychotic disorder due to another medical condition, and epilepsy-related psychosis.

A schizophrenia-spectrum disorder remained an important differential diagnosis because the patient had recurrent hallucinations, bizarre delusions, and behavioral disorganization over several years. Features that prompted evaluation for a secondary or neurological contributor included multimodal hallucinations, the presence of olfactory phenomena, and reported sudden motor events. However, an episodic course and preserved functioning between episodes do not by themselves exclude schizophrenia. Brief psychotic disorder was less likely because the symptoms had recurred over several years. A psychotic disorder due to another medical condition could not be definitively established or excluded because structural neuroimaging was not available.

Focal epilepsy was clinically considered because of the reported sudden brief motor jerks and episodic behavioral phenomena. A possible frontal origin was considered because frontal seizures can be brief, motor-predominant, behaviorally unusual, and sleep-related. Nevertheless, the localization remained unconfirmed: the routine interictal EEG contained no epileptiform discharges, no typical event was captured on video-EEG, and no structural brain imaging was obtained. The olfactory experiences were not considered frontal-specific because olfactory auras are more commonly associated with medial temporal structures but can also involve orbitofrontal or insular regions[13]. The sleep-onset jerks were also nonspecific and could not, in isolation, establish epileptic seizures. Therefore, the diagnostic formulation used in this report is psychosis with clinically suspected focal epilepsy of possible frontal origin rather than confirmed FLE.

Treatment and Clinical Decision-Making

The management of the patient's condition initially aimed at stabilizing symptoms by administering a second-generation antipsychotic. The patient was given risperidone 2 mg twice a day to manage psychotic symptoms and agitation. Although some improvement was noted, some symptoms like episodic behavioral difficulties and atypical sensory hallucinations persisted.

Because the treating team considered focal epilepsy a clinically relevant differential diagnosis despite the normal routine EEG, phenytoin 100 mg three times daily was initiated as antiseizure treatment. This decision was based on the clinical suspicion generated by the reported motor events and episodic behavioral symptoms rather than on electrographic confirmation.

During treatment with risperidone and phenytoin, agitation, hallucinations, behavioral disturbance, insight, and functioning improved. Because the antipsychotic and antiseizure medications were administered concurrently, this clinical response cannot determine which treatment produced the improvement and should not be used as proof of an epileptic etiology or frontal localization.

DISCUSSSION

This case describes schizophrenia-like psychosis in a 19-year-old woman in whom focal epilepsy of possible frontal origin was clinically suspected but not electroclinically confirmed. The diagnostic concern arose from the combination of multimodal hallucinations, fluctuating behavioral symptoms, and reported sudden motor phenomena around sleep onset. Importantly, the case does not demonstrate definite FLE: routine EEG showed no epileptiform discharges, no habitual event was captured on video-EEG, and brain imaging was not performed. The principal clinical value of the case is therefore the diagnostic reasoning and the need to maintain, but also appropriately qualify, epilepsy in the differential diagnosis of atypical psychosis.

Psychotic Manifestations in Epilepsy and Differential Diagnosis with Schizophrenia

Psychotic disorders occur more frequently in people with epilepsy than in non-epileptic populations[1]. In FLE specifically, however, the literature is sparse. Gold et al. identified 66 published FLE cases with primary psychiatric manifestations over 25 years, with psychosis as the primary presentation in 17 cases[4]. Adachi et al. also noted that FLE-related psychosis had been much less studied than TLE-related psychosis[5]. These data support the clinical novelty of the present report without implying a precise FLE-specific prevalence that current evidence does not provide.

Psychosis associated with epilepsy is commonly categorized by its temporal relationship to seizures as ictal, postictal, or interictal[7,18,19,20]. Hallucinations, delusions, affective change, agitation, and disorganized behavior may occur across these categories, and interictal psychosis may resemble schizophrenia. In the present patient, the available history did not establish a consistent temporal relationship between psychosis and objectively documented seizures; consequently, classifying the psychosis as specifically ictal, postictal, or interictal would exceed the available evidence.

Frontal and temporal networks both contribute to emotional regulation, salience processing, behavior, and perception, which can help explain why focal epilepsies involving these networks may produce psychiatric manifestations[8,9]. At the same time, psychosis can be a comorbidity independent of individual seizures. Compared with primary schizophrenia, some epilepsy-related psychoses have been reported to show fewer negative symptoms and less progressive functional deterioration, but these are group-level tendencies rather than diagnostic criteria for an individual patient[17,21,22].

Clinical Features Relevant to the Differential Diagnosis

1. **Temporal Course and Symptom Fluctuation**

The recurrent and fluctuating course of symptoms with periods of better functioning prompted consideration of an episodic neurological process. Nevertheless, temporal fluctuation is not specific to epilepsy, and the history did not demonstrate a reproducible time-locking of psychosis to documented seizures. Therefore, symptom course was treated as a reason for further neurological evaluation rather than as evidence sufficient to classify the psychosis as postictal or interictal[18,19,20].

2. **Multimodal and Olfactory Hallucinations**

Auditory, visual, and olfactory hallucinations broaden the differential diagnosis. Olfactory symptoms can occur during focal seizures, but the ILAE semiology literature indicates that they are reported most often with medial temporal structures and may also arise from medial orbitofrontal or insular regions; they have no reliable lateralizing value[13]. Thus, the patient's recurrent foul-smell experiences were a neurological clue but did not specifically establish frontal lobe onset.

3. Motor Events Around Sleep Onset

Frontal seizures often have prominent motor manifestations and may occur during sleep[14]. This made the reported sudden body jerks at sleep onset relevant to the differential diagnosis. However, the events were not captured, their stereotypy and duration were not objectively documented, and sleep-onset jerks can also be physiological or non-epileptic. Accordingly, the motor history increased suspicion for a focal seizure disorder but was insufficient to diagnose or localize.

Frontal, Temporal, and Parietal Lobe Seizure Semiology

The clinical manifestations of focal seizures overlap because seizures can rapidly propagate beyond the lobe of onset. Nevertheless, several semiological tendencies can help guide, but not prove, localization[13,14,23].

In the present case, the sleep-onset motor jerks are compatible with, but not specific for, a frontal motor seizure pattern. Conversely, the olfactory experiences are classically more associated with medial temporal networks but may also involve orbitofrontal or insular cortex[13]. No typical parietal somatosensory, vestibular, or body-schema aura was documented. Because these features point to overlapping networks and no event was captured electrographically, the available semiology supports only a suspected focal epilepsy and does not reliably distinguish frontal from temporal onset

Table 1. Comparative Clinical Features and Scalp EEG Findings

Domain	Frontal lobe	Temporal lobe	Parietal lobe
Typical course	Often abrupt, brief, stereotyped, may cluster and occur during sleep.	Often focal aware symptoms followed by impaired awareness; evolution may be longer than many frontal seizures.	Often begins with focal sensory symptoms; rapid propagation can make later signs resemble frontal or temporal seizures.
Motor/ behavioral signs	Prominent tonic or asymmetric posturing, hyperkinetic behavior, vocalization, or other complex motor activity.	Behavioral arrest and oroalimentary/manual automatisms are common patterns in focal impaired-awareness seizures.	Motor signs can appear after spread, but initial sensory phenomena may be the key localizing clue.
Sensory/ experiential clues	Fear or complex behavioral phenomena may occur; olfactory symptoms can arise from orbitofrontal cortex but are not specific.	Epigastric, experiential (e.g., déjà vu/fear), auditory, and olfactory phenomena may occur; olfactory symptoms are most often linked to medial temporal structures.	Somatosensory tingling/numbness, pain, vestibular symptoms, and body-perception illusions are characteristic clues; somatosensory auras are frequently reported.
Scalp EEG considerations	May be normal or misleading, especially with deep/mesial frontal onset or rapid propagation.	Temporal epileptiform abnormalities may support localization, but a normal routine EEG does not exclude TLE.	Scalp EEG may be nonlocalizing because of rapid spread; multimodal evaluation may be required.

Pathophysiological Insights

Psychosis in epilepsy is increasingly understood as a network-level phenomenon rather than a simple consequence of a single anatomical focus. Proposed mechanisms include fronto-limbic circuit dysfunction, altered excitatory-inhibitory balance, interictal epileptiform network activity, and partially

shared neurobiological or genetic vulnerability between epilepsy and psychotic disorders[8,24]. These models support biological plausibility but cannot establish that the psychosis in an individual patient is caused by epilepsy without adequate electroclinical correlation.

At the neurotransmitter level, glutamatergic hyperexcitability and GABAergic dysfunction have been proposed as contributors to both seizure generation and psychotic symptoms, while structural, functional, and inflammatory mechanisms may further influence psychiatric vulnerability[7,8,9]. These overlapping mechanisms reinforce the need for integrated neurological and psychiatric assessment rather than assuming that psychosis automatically localizes the epileptogenic zone.

EEG Normality and Diagnostic Considerations

The routine interictal EEG in this patient was normal and showed no epileptiform discharges. This finding reduces the amount of objective support available for epilepsy but does not exclude the diagnosis: the first routine EEG fails to record interictal epileptiform discharges in approximately 47-50% of patients with epilepsy[23]. In addition, frontal lobe seizures can be particularly difficult to localize using scalp EEG because frontal sources may be deep and ictal recordings may be obscured by movement artifact or rapid propagation[14].

For this patient, the appropriate next diagnostic steps would have been repeat or sleep-deprived EEG and prolonged video-EEG monitoring to capture a habitual event, together with epilepsy-protocol brain MRI to assess for a structural lesion[23]. These tests were not available in the reported evaluation. Consequently, the manuscript deliberately uses the terms 'clinically suspected focal epilepsy' and 'possible frontal origin' rather than presenting FLE as a confirmed diagnosis. A history of frontal head trauma is clinically relevant but, without imaging or electroclinical concordance, does not establish a frontal epileptogenic focus.

Treatment Implications and Clinical Vigilance

Management of psychosis in a patient with suspected epilepsy requires coordinated neurological and psychiatric care. Antipsychotic treatment may be needed for severe psychotic symptoms, while antiseizure therapy should be individualized according to the probability and type of epilepsy, potential drug interactions, and neurological assessment[10]. Where the epilepsy diagnosis or localization remains uncertain, diagnostic clarification with prolonged EEG and imaging is preferable to inferring causality from symptom response alone.

The patient improved during combined risperidone and phenytoin treatment. This temporal association is clinically encouraging but cannot confirm epilepsy-related psychosis because both medications were used simultaneously and spontaneous fluctuation of psychosis is possible. The therapeutic response therefore supports continued multidisciplinary follow-up but should not be interpreted as a diagnostic test for FLE.

LIMITATION

This report has several important limitations. First, no epileptiform discharges were recorded on routine interictal EEG and no habitual motor or behavioral event was captured on video-EEG. Second, brain CT/MRI was not performed, preventing assessment of a structural lesion or anatomical localization. Third, the reported body jerks occurred around sleep onset and were not sufficiently characterized to distinguish epileptic myoclonus from physiological hypnic jerks or other non-epileptic events. Fourth, the olfactory experiences are not specific to frontal lobe epilepsy. Finally, risperidone and phenytoin were administered in the same treatment period, so clinical improvement cannot be

attributed specifically to antiseizure therapy. These limitations mean that FLE remains a clinical suspicion rather than a confirmed electroclinical diagnosis.

CONCLUSION

This case highlights the need to consider focal epilepsy in the differential diagnosis of atypical or fluctuating psychosis when paroxysmal neurological symptoms are reported, while avoiding over-localization based on nonspecific semiology. A normal routine EEG does not exclude epilepsy, but neither does it provide evidence for FLE. In the present patient, the most defensible formulation is psychosis with clinically suspected focal epilepsy of possible frontal origin. Prolonged video-EEG and epilepsy-protocol MRI would be required to strengthen diagnostic and anatomical certainty.

INFORMED CONSENT

Written informed consent for publication of this anonymized case report was obtained directly from the patient.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest in the publication of this article. The authors affirm that this work was conducted independently and that the results and interpretation presented do not reflect the interests or influence of any related institutions or parties.

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