Affective, Behavioral, and Cognitive Symptoms Associated With Focal Impaired Awareness (Complex Partial) Seizures:

Evaluation and Treatment

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Lessons Learned at the Interface of Medicine and Psychiatry

The Psychiatric Consultation Service at Massachusetts General Hospital sees medical and surgical inpatients with comorbid psychiatric symptoms and conditions. During their twice-weekly rounds, Dr Stern and other members of the Consultation Service discuss diagnosis and management of hospitalized patients with complex medical or surgical problems who also demonstrate psychiatric symptoms or conditions. These discussions have given rise to rounds reports that will prove useful for clinicians practicing at the interface of medicine and psychiatry.

Prim Care Companion CNS Disord 2025;27(1):24f03813

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Have you ever wondered whether the episodic anxiety experienced by your patients indicated more than just a panic attack? Have you been uncertain about how to conduct an ictal review of systems? Have you been unsure of which antiepileptic drug to prescribe to ameliorate their symptoms? If you have, the following case vignette and discussion should prove useful.

CASE VIGNETTE

Mr D, a healthy 30-year-old right-handed man, had recently relocated from out of state and sought to establish care with a primary care provider. He had started to experience what he referred to as "anxiety attacks" once or twice a week. During these episodes, he felt anxious, was diaphoretic, and struggled to maintain his focus or speak; afterward, he noted "brain fog" and felt that he lost chunks of time. He worked remotely as an accountant and was alone in his home while he was working. In addition, his wife expressed concern about his new and unusual nocturnal behaviors (ie, once a week after he fell asleep, he sat up, grunted, and picked at his clothing). She thought he appeared unaware of his surroundings and did not respond when she spoke to him. She estimated that the episodes lasted for 1–3 minutes. He had no memory of these behaviors the following day.

WHAT ARE COMPLEX PARTIAL SEIZURES?

The term *complex partial seizures* was renamed *focal-onset impaired awareness seizures* by the International League Against Epilepsy (ILAE) in 2017 to describe seizures that originate from a solitary brain location.[1] The revised nomenclature was pursued to better describe seizure types, account for advancements in the neurobiological understanding of seizures, and adopt consensus terminology for better communication across the globe. The term *focal* describes seizures that arise from 1 hemisphere, whereas the term *generalized* describes seizure activity that originates simultaneously in both hemispheres. Unlike focal aware seizures, which do not impact consciousness, unaware seizures cause a significant alteration (including confusion and memory deficits during or after the seizure) or loss of consciousness.

Some patients with focal impaired awareness seizures (FIAS) experience an aura, which can include odd sensations (eg, smells, tastes, sounds, visual disturbances, and feelings), emotions (eg, fear and anxiety), perceptions (eg, déjà vu and jamais vu), and psychical experiences (eg, hallucinations and delusions).[2] As these seizures progress, altered consciousness arises. Patients may appear confused and experience automatisms (ie, repetitive automatic

Clinical Points

- The semiology of focal impaired awareness seizures (FIAS) encompasses motor and nonmotor phenomena, including sensory, affective, psychiatric, autonomic, and behavioral components, and their duration varies from 30 seconds to several minutes.
- The diagnosis of FIAS begins with obtaining a comprehensive clinical history and an ictal review of symptoms, which is supplemented by laboratory testing (eg, electroencephalogram) and brain imaging.
- Treatment involves the administration of a therapeutic dose of an antiseizure medication, the selection of which is guided by its accessibility and affordability, metabolic concerns, side effect profiles, drug interactions, and comorbid medical conditions.

behaviors), such as lip smacking or picking at their clothing. During this phase, patients may not respond appropriately to their surroundings. FIAS typically last for several seconds to a few minutes, and they are often followed by postictal confusion. Typically, patients with these seizures do not recall the seizure or the events that occurred just prior to the seizure. This period can last for several minutes to hours before the patient returns to their baseline.

HOW CAN FOCAL IMPAIRED AWARENESS SEIZURES MANIFEST?

FIAS have myriad manifestations. The semiology of FIAS encompasses motor and nonmotor phenomena, including sensory, affective, psychiatric, autonomic, and behavioral components, and their duration varies, typically lasting from 30 seconds to several minutes. In many instances, FIAS can be stereotyped, meaning the events look similar to one another. Clinical signs include the following:

- **Altered awareness and/or decreased arousal.** Individuals experiencing FIAS often appear confused, unresponsive, or unaware of their surroundings, and their perception of time and space may be distorted. In addition, they may be unable to respond to questions or commands.
- **Unusual sensory experiences.** A bevy of sensations, such as tingling of the extremities, nausea, a rising sensation in the stomach, or dizziness, may be experienced.
- **Automatisms and behavioral changes.** A variety of repetitive, involuntary, and purposeless movements (such as lip smacking, chewing, swallowing, or simple hand movements) may appear. Movements may also be more complex or appear semipurposeful (eg, appearing to pick at one's clothes or walk aimlessly).
- **Emotional responses.** People with FIAS may experience intense feelings (eg, fear, joy, anger, or sadness). They may also describe déjà vu (ie, feeling as though they have experienced a situation before) or jamais vu (when familiar things or situations feel novel or unfamiliar).
- **Hallucinations**. Individuals with FIAS may experience several types of hallucinations (including those that are olfactory, auditory, gustatory, tactile, or visual).
- **Autonomic phenomena**. Changes in vital signs may arise (such as alterations in heart rate or blood pressure) as well as changes in skin color (such as pallor or flushing).[3]
- **Speech difficulties.** If a seizure affects the dominant hemisphere for certain functions (such as language in the left temporal lobe for right-handed patients), the symptoms may include speech difficulties or altered language comprehension.

These seizures most commonly originate from the temporal and frontal lobes. Compared to frontal lobe epilepsy, temporal lobe epilepsy has been associated with a longer seizure duration (ie, 2–3 minutes) and with a lower frequency. Somatosensory auras, abdominal auras, and olfactory hallucinations are often correlated with temporal lobe epilepsy.[4] In addition, some studies have suggested that there may be a difference in clinical presentation depending on the person's age.[5] Although FIAS originate from a single hemisphere of the brain, they can spread to the other hemisphere and become bilateral tonic-clonic seizures, which can last for several minutes.

Following the seizure, individuals typically enter a postictal state characterized by fatigue, confusion, and difficulty communicating. During this phase, they may experience a cognitive haze with impaired memory before becoming lucid, with full awareness and normal function. Postictal psychosis (with delusions, hallucinations, and aggressive behaviors) may occur following FIAS and can even develop almost a week after the return to lucidity.[6] Overall, these symptoms can interfere dramatically with a person's quality of life, underscoring the importance of early detection and treatment to reduce morbidity and mortality.

WHAT CAN LOOK LIKE FIAS BUT IS NOT?

Given the large array of possible manifestations, FIAS can look like other psychiatric, neurological, and medical (including cardiovascular) presentations. Due to the changes in affect, behavior, and cognition that flow from FIAS, the condition is often misdiagnosed as a primary (idiopathic) psychiatric condition.[2,7] Viewing this

condition through a psychiatric lens, transient psychotic episodes, dissociative disorders, and panic attacks each have features that resemble FIAS. For example, ictal, peri-ictal, and postictal psychosis may be misidentified initially as primary psychosis.[8] Functional neurological disorder, specifically functional seizures,[9] often presents with altered consciousness, unusual sensory experiences, and abnormal movements, which may present like FIAS. Unlike FIAS, they will have semiological characteristics of a functional seizure (tight eye closure and asynchronous movements) and will not have abnormal electrical brain activity findings on the electroencephalogram (EEG) during an episode. An absent EEG signature with semiological features for epileptic seizures should warrant additional epileptic seizure workup.[10] Another rule-out diagnosis can be factitious disorder, in which a patient may feign seizure symptoms to appear sick.

Transient ischemic attacks, which present with temporary focal neurological deficits that typically resolve within 24 hours, can also mimic FIAS. Migraine auras, characterized by paroxysmal visual, sensory, and speech disturbances, frequently resemble FIAS. Vasovagal syncope and orthostatic hypotension can share symptoms (such as transient loss of consciousness or altered awareness) with FIAS. In addition, FIAS may be mistaken initially for movement disorders that involve tremors, tics, paroxysmal dystonia, and dyskinesias. Sleep disorders, including narcolepsy and rapid eye movement, sleep behavior disorder, substance intoxication or withdrawal, or medication toxicity, may also resemble manifestations of FIAS.[11]

WHY DO FIAS DEVELOP?

The manifestations of FIAS stem from abnormal electrical activity that originates in a focal area of a single brain hemisphere. The underlying etiologies of this aberrant electrical activity may involve genetic and developmental predispositions as well as acquired conditions.

Structural brain abnormalities associated with the development of FIAS include intraventricular hemorrhages, brain tumors, and cortical dysplasia. Notably, seizures are among the most common clinical presentations of brain tumors.[12] Perinatal insults, such as hypoxic-ischemic encephalopathy and periventricular leukomalacia from intraventricular hemorrhage, have also been linked with an increased risk of seizures. Additionally, acute brain insults, such as traumatic brain injuries (TBIs), also raise the risk of developing epilepsy, with the likelihood of epilepsy correlating directly with the severity of the injury.[13]

Cerebrovascular disease has been estimated to account for about 50%–70% of epilepsy cases in adults,[14] while stroke has been the most common cause of late-onset epilepsy. Neurodegenerative disorders and dementia also contribute to the onset of epilepsy in the elderly.[15] In general, the incidence and prevalence of epilepsy increase with older age[16]; FIAS are thought to be responsible for roughly 40%–50% of new-onset seizures in the elderly.[5]

Genetic factors for epilepsy predispose people to developing FIAS and potentially correlate with different phenotypic expressions.[17] Metabolic factors, including certain mitochondrial conditions, such as DNA polymerase subunit gamma–related disorders and mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes (MELAS syndrome), can present with FIAS. Autoimmune conditions, inflammatory processes, and infections (including encephalitis, meningitis, human immunodeficiency virus infection, tuberculosis, and neurocysticercosis) can also increase the risk of developing seizures.

Furthermore, several factors can precipitate underlying seizure disorders. For example, use of certain medications and substances or withdrawal from substances can lower the seizure threshold, as can sleep deprivation and stress.[18,19]

HOW OFTEN DO FIAS COEXIST WITH GENERALIZED TONIC-CLONIC SEIZURES?

If ictal electrographic activity remains confined to 1 brain area, then patients experience FIAS. However, if ictal electrographic activity spreads to other brain regions, then patients experience secondary generalized tonic-clonic seizures (GTCS). This occurs with a variable prevalence; however, at least 5%–25% of FIAS can secondarily generalize, depending on their region of onset.[20] The prevalence is difficult to quantify in part because patients may be amnestic to the focal symptoms that arise prior to their generalizing.[21,22] Even though FIAS can secondarily generalize to GTCS, it is also possible that FIAS and GTCS can occur independently in the same individual. Patients can have discrete types of seizures due to pathophysiology of underlying conditions, which can create different foci of seizure activity.

HOW CAN FIAS BE DIAGNOSED?

The diagnosis of FIAS begins with obtaining a comprehensive clinical history and an ictal review of symptoms. For patients who report classic symptoms, a presumptive diagnosis can be made without supportive testing. For example, if a patient reports having stereotyped episodes of déjà vu in concert with a rising epigastric sensation followed by confusion, the patient can be diagnosed with confidence as having typical FIAS

that arises from the mesial temporal lobe, as abdominal aura are associated with temporal lobe seizures with a probability of 74%.[4,23] Nevertheless, a thorough ictal review of systems should always be performed. This includes asking about sensory auras, the presence of motor automatisms, episodes of confusion or a loss of time, and provocative triggers (eg, stroboscopic lights, anaerobic exercise, sleep deprivation, and recent illness).[20] Ascertaining the frequency and duration of symptoms is key. The timing of symptoms is another important factor; symptoms that awaken a patient from physiological (EEG confirmed) sleep are less likely to represent nonictal events.[24] As patients may be amnestic to the events, it is helpful to obtain collateral information to clarify both ictal and postictal behavior. Eyewitnesses can also comment on the frequency of behavioral arrest or alteration in speech.[25] It also behooves the examiner to inquire about seizure risk factors (eg, Is there a history of birth trauma, developmental delay, complex febrile seizures, meningoencephalitis, or TBIs? Is there a strong family history of epilepsy? Does the patient have a personal history of a genetic or clinical syndrome that is often associated with epilepsy?). If any of these risk factors are present, it may make the presenting clinical history more consistent with FIAS.[26]

However, in cases in which uncertainty persists, obtaining a brain magnetic resonance imaging (MRI) scan and an EEG is appropriate. An MRI brain scan can assess for focal epileptogenic lesions, such as mesial temporal sclerosis or a focal cortical dysplasia. For highest yield, the study should be done with a seizure protocol that includes thin slices through the temporal lobes.[25] EEG monitoring is done during both wakefulness and sleep to screen for focal spikes, which can indicate an underlying area of cortical irritability and an increased seizure tendency. Typically, patients will begin with a 20- to 60-minute routine EEG in the office. If needed, a longer sampling of sleep can be obtained with an overnight ambulatory EEG to increase diagnostic yield.[27] If there is a focal abnormality on the MRI or EEG in a region that localizes to a plausible area of symptom onset, then this supports a diagnosis of FIAS. If the patient experiences typical clinical symptoms while undergoing EEG monitoring, and an associated ictal electrographic change is seen on the EEG, then this confirms FIAS. However, having a normal MRI scan or EEG does not exclude a diagnosis of FIAS.[25,28] Additional biomarkers such as ictal single-photon emission computed tomography or interictal positron emission tomography, or in some cases depth electrode recordings, may be used for diagnostic clarification.

HOW CAN FIAS BE TREATED?

Once the diagnosis has been made, the patient should receive a therapeutic dose of an antiseizure medication (ASM). Currently, a multitude of ASMs are available to treat FIAS. Selection is guided by factors that include accessibility and affordability, metabolic concerns, side effect profiles, drug interactions, and comorbid medical conditions. Seizure burden is also a key deciding factor. If a patient is experiencing frequent FIAS with secondary convulsions, then the physician will likely select an ASM that can be titrated rapidly and safely. If the events are infrequent, then a slower titration schedule is acceptable.[29] For patients who continue to experience seizures while on 1 ASM, polytherapy can be considered; however, this requires careful consideration of potential side effects and drug interactions.

A 2016 expert opinion survey concluded that levetiracetam, lamotrigine, and oxcarbazepine were considered the ASMs of choice for the initial treatment of focal seizures, including FIAS.[30] In practice, levetiracetam is often prescribed as a first-line agent.[31] This is due to its availability in intravenous and oral formulations and its ease of safe titration, which make it an attractive option in both inpatient and emergency department settings. Levetiracetam has a relative paucity of pharmacologic interactions and an overall favorable side effect profile. Its most notable adverse effect is increased irritability or depressed mood, which is seen in a minority of patients. Since the medication is renally excreted, care should be taken to avoid toxicity in patients with impaired renal function.[29] However, this makes it the ASM of choice in patients with hepatic dysfunction or following organ transplantation.[30] Both lamotrigine and levetiracetam are considered the ASMs of choice in the elderly with FIAS as well as in women of child-bearing potential.[30] This is due to the relatively favorable teratogenicity profile of both agents. Given its mood-stabilizing properties, lamotrigine is favored in patients with psychiatric disorders, and especially in those who have not tolerated levetiracetam due to its effects on mood. However, due to the risk of rash, such as occurs in Steven-Johnson syndrome, lamotrigine titration must occur over several weeks to months. Serum levels are required to guide dose adjustment. Thus, it is a less attractive option for patients with a high burden of disruptive seizures.[20]

Other options include use of agents with sodium channel properties, such as oxcarbazepine. Oxcarbazepine also carries a risk of rash and has some mood-stabilizing properties, but it can be titrated safely, much more rapidly than lamotrigine. A possible side effect of this medication is hyponatremia, so patients should undergo routine serum chemistries and assessment of drug levels.[29]

Fortunately, the 3 ASMs mentioned above (levetiracetam, lamotrigine, and oxcarbazepine) are also formulated in extended-release formulations, which may assist with compliance or minimize side effects. Other agents that treat FIAS include brivaracetam, cenobamate,

clobazam, eslicarbazepine, felbamate, gabapentin, lacosamide, pregabalin, and zonisamide.[29] Older ASMs such as carbamazepine, phenytoin, and phenobarbital treat FIAS, but they have fallen out of favor due to their potential for long-term adverse effects and numerous medication interactions.[30] For patients who fail to achieve adequate seizure control despite therapeutic trials of multiple ASMs, a referral should be made to an epilepsy center for consideration of possible epilepsy surgery.[26]

For patients who tend to experience a cluster of multiple FIAS in the same day, or consistently experience secondary generalization to bilateral tonic-clonic seizure, it is useful to have a rescue medication on hand. This is typically a benzodiazepine, as this class of medication provides robust antiseizure properties with a relatively rapid effect onset. If a patient can swallow pills safely, then oral lorazepam or clonazepam are reasonable options. If not, then other newer agents on the market include intranasal diazepam or midazolam. These nasal sprays are designed to ease administration by either the patient or a bystander. Care should be taken to monitor for respiratory status after the administration of any benzodiazepine, particularly if the patient is benzodiazepine naive.[32]

Patients with epilepsy are at a higher risk than the general population of developing psychiatric disorders. This can include depression, anxiety, and psychosis, as well as attention-deficit/hyperactivity disorder or other cognitive disorders. Patients with affective, behavioral, and cognitive symptoms associated with FIAS also benefit from collaboration between neurology and psychiatry, particularly those with persistent neuropsychiatric symptoms.

In addition to good ASM adherence and treatment of comorbid psychiatric symptoms, patients with FIAS should adopt lifestyle habits to avoid lowering their seizure threshold. This includes avoiding excessive sleep deprivation or becoming intoxicated with recreational substances, such as alcohol.[25]

WHO SHOULD INITIATE TREATMENT FOR FIAS?

The decision to start ASMs is based on specific patient factors. While typically initiated by neurologists, and more specifically epileptologists, primary care clinicians treat a wide variety of diagnoses and can start medications in a patient with straightforward epilepsy. Accurate diagnosis involves obtaining a detailed medical history, conducting a physical examination, and performing diagnostic tests (eg, EEG, MRI, and computed tomography). If the diagnosis remains unclear or if the seizures are refractory to treatment (ie, uncontrolled after 1 or 2 medication trials), referral to a neurologist may be needed. Guidelines established by the National Association of Epilepsy Centers recommend that primary care providers initiate medications, and if the patient continues to experience seizures after 3 months, referral to an epilepsy specialist is indicated.[33] If the diagnosis is uncertain, the presentation is atypical, or the seizures are the consequence of another neurological diagnosis, this should also be an indication for a referral.[33] Treatment includes use of ASMs, but it should also include a discussion of lifestyle changes (including maintaining a regular sleep schedule, managing stress, and avoiding triggers).

WHAT HAPPENED TO MR D?

Mr D underwent a 72-hour EEG while at home, which showed epileptic activity originating from his temporal lobe during both daytime and nighttime episodes. He was started on levetiracetam, and he tolerated it well (without notable side effects). He also focused on improving his sleep hygiene and stress management. He reported resolution of his seizures after starting the medication.

CONCLUSION

Complex partial seizures, renamed FIAS by the ILAE in 2017, are seizures that originate from a solitary brain location and cause a significant alteration (including confusion and memory deficits during or after the seizure) or loss of consciousness. Some patients with FIAS experience an aura with odd sensations, emotions, perceptions, and psychical experiences. As these seizures progress, altered consciousness arises. Patients may appear confused and experience automatisms (ie, repetitive automatic behaviors), such as lip smacking or picking at their clothing. FIAS typically last between several seconds and a few minutes, and they are often followed by postictal confusion; patients with these seizures typically do not recall the seizure or the events that occurred just prior to the seizure.

FIAS most commonly originate from the temporal and frontal lobes. Compared to frontal lobe epilepsy, temporal lobe epilepsy tends to have a longer seizure duration (ie, 2–3 minutes) and occurs less often. Although FIAS originate from a single brain hemisphere, they can spread to the other hemisphere and become bilateral tonic-clonic seizures, which can last several minutes. The underlying etiologies of FIAS are plentiful and include genetic and developmental disorders, structural brain abnormalities, perinatal insults, TBIs, cerebrovascular disease, neurodegenerative disorders and dementia, metabolic disorders, autoimmune conditions, inflammatory processes, infections, and toxicity and withdrawal from medications.

The diagnosis of FIAS begins with obtaining a comprehensive clinical history (replete with an ictal

review of symptoms), which is supplemented by laboratory testing (eg, EEG) and brain imaging. Treatment involves the administration of a therapeutic dose of an ASM; selection of the agent is guided by its accessibility and affordability, metabolic concerns, side effect profiles, drug interactions, and comorbid medical conditions.[34,35]

Article Information

Published Online: January 3, 2025. https://doi.org/10.4088/PCC.24f03813

Submitted: July 10, 2024; accepted September 30, 2024.

To Cite: Adams C, Basovic L, Cantos A, et al. Affective, behavioral, and cognitive symptoms associated with focal impaired awareness (complex partial) seizures: evaluation and treatment. *Prim Care Companion CNS Disord*. 2025;27(1):24f03813

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Relevant Financial Relationships: None.

Funding/Support: None.

Author Contributions: Drs Adams, Basovic, and Cantos contributed equally as first authors.

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