

REVIEW ARTICLE

Clinical semiology of epileptic seizures: A scoping review of pre-ictal, ictal, and post-ictal phenomenology

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Abstract

Introduction: Clinical semiology of epileptic seizures encompasses the spectrum of manifestations occurring before, during, and after seizure events, reflecting the dynamic involvement of functional brain networks. Careful recognition of these clinical phenomena is fundamental for seizure classification, localization of the epileptogenic zone, differential diagnosis, prognostication, and individualized epilepsy management. Although individual aspects of seizure semiology have been extensively described, a comprehensive synthesis spanning the pre-ictal, ictal, and post-ictal phases remains limited.

Objectives: This review aims to map and synthesize the available evidence on the clinical semiology of epileptic seizures across the pre-ictal, ictal, and post-ictal phases, highlighting their epidemiological characteristics, neurobiological basis, diagnostic significance, and clinical applications.

Methods: This scoping review was conducted following the Joanna Briggs Institute methodology and reported according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews. A comprehensive search of multiple databases was performed without date restrictions. Eligible studies were screened, and evidence was charted descriptively according to the pre-ictal, ictal, and post-ictal phases of epileptic seizures.

Results: A total of 1,120 records were identified, of which 60 studies met the inclusion criteria after screening and full-text review and were included in this review. The included studies demonstrated that pre-ictal manifestations may facilitate seizure prediction and provide important localizing information. Ictal semiology remains the cornerstone of seizure classification, reflecting the anatomical origin and propagation of seizure activity. Post-ictal phenomena contribute valuable diagnostic, lateralizing, and prognostic information. Collectively, the evidence highlights the complementary role of seizure semiology alongside electroencephalography, neuroimaging, and other contemporary diagnostic modalities in comprehensive epilepsy evaluation.

Conclusion: Clinical semiology remains an important component of epilepsy assessment despite advances in modern neurodiagnostic techniques. Mapping the available evidence across the pre-ictal, ictal, and post-ictal phases underscores the continued importance of meticulous clinical observation for accurate diagnosis, seizure localization, patient counseling, and treatment planning, while identifying areas requiring further research to improve precision epilepsy care.

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1. Introduction

Epileptic seizures are among the most common neurological disorders, affecting more than 1% of the global population and contributing substantially to morbidity, quality-of-life impairment, injury, and sudden unexpected death in epilepsy.¹ A seizure is defined as a transient occurrence of signs and/or symptoms resulting from abnormal excessive or synchronous neuronal activity in the brain, whereas epilepsy refers to a chronic brain disorder characterized by an enduring predisposition to generate recurrent unprovoked seizures.² Because seizures constitute the principal clinical manifestation of epilepsy, careful characterization of seizure semiology remains fundamental to accurate diagnosis, syndrome classification, and localization of seizure onset, treatment selection, and surgical evaluation.

Clinical semiology refers to the observable signs and symptoms that characterize different phases of a seizure. Seizure semiology reflects activation or disruption of specific cortical and subcortical networks and may involve sensory, motor, autonomic, cognitive, emotional, or awareness-related manifestations, either in isolation or in combination. Careful recognition of these clinical features provides important clues regarding seizure type, localization, and propagation pathways. In addition to its benefits in medical evaluation, seizure semiology plays an important role in the presurgical workup and can be used effectively to differentiate between epileptic and non-epileptic events.³⁻⁶

The clinical evolution of an epileptic seizure can be broadly divided into three clinically relevant phases: the pre-ictal, ictal, and post-ictal phases. The pre-ictal state encompasses the period preceding seizure onset during which prodromal symptoms or auras may occur; the ictal state represents the clinical expression of the seizure itself; and the post-ictal state comprises the recovery period following seizure termination.⁷ Careful recognition of the characteristic manifestations occurring across these phases not only facilitates accurate seizure classification and localization but also may improve seizure prediction, patient safety, prognostication, and individualized therapeutic decision-making.^{8,9}

Although numerous publications have described individual seizure types or specific aspects of seizure semiology, a comprehensive synthesis of the evolving

clinical manifestations across the entire seizure continuum, from the pre-ictal through the ictal to the post-ictal phases, remains limited.^{10,11} This scoping review addresses this gap by integrating contemporary evidence on the clinical phenomenology of all three seizure phases within a single clinically oriented framework.

In addition to summarizing current knowledge, the review highlights the diagnostic and localizing value of seizure semiology, its role in differentiating epileptic from non-epileptic events, its application in presurgical evaluation, and its integration with modern diagnostic modalities, including video-electroencephalography (EEG) monitoring and neuroimaging. The review is intended for clinicians involved in epilepsy care, including general neurologists, pediatric neurologists, epilepsy specialists, trainees, and allied healthcare professionals, and focuses on clinically relevant manifestations applicable to both adult and pediatric populations where appropriate.

This scoping review aims to systematically map and synthesize the available evidence on the clinical semiology of epileptic seizures across the pre-ictal, ictal, and post-ictal phases. Specifically, the review addresses the following question: what evidence is available regarding the clinical manifestations, epidemiological characteristics, neurobiological basis, diagnostic significance, and clinical applications of seizure semiology across the pre-ictal, ictal, and post-ictal phases in individuals with epilepsy?

2. Methods

2.1. Study design and protocol

This study was conducted as a scoping review to systematically map and synthesize the available evidence on the clinical semiology of epileptic seizures across the pre-ictal, ictal, and post-ictal phases. The review was performed in accordance with the Joanna Briggs Institute (JBI) methodology for scoping reviews and subsequently refined by Peters *et al.*¹² The reporting of this review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses Extension for Scoping Reviews guideline.¹³ To enhance methodological rigor, the protocol was independently reviewed by two epilepsy specialists who were not members of the research team before implementation. The final protocol was registered on the Open Science Framework to ensure transparency and reproducibility of the review process before study initiation.

2.2. Eligibility criteria

Studies were considered eligible if they met one or more of the following criteria:

- (i) Described the clinical manifestations or semiology of epileptic seizures occurring during the pre-ictal, ictal, or post-ictal phases.
- (ii) Investigated the epidemiology, neurobiological basis, classification, localization, lateralization, or clinical significance of seizure semiology.
- (iii) Evaluated the diagnostic, prognostic, or therapeutic relevance of seizure semiology in epilepsy care.
- (iv) Included adult or pediatric populations with epilepsy.
- (v) Were original research articles, observational studies, clinical trials, systematic reviews, narrative reviews, consensus statements, or international clinical guidelines.

Studies that did not meet the eligibility criteria were excluded. No restrictions were placed on publication year to allow comprehensive mapping of the available evidence. However, only articles published in English or translated into English were included.

2.3. Information sources and search strategy

A comprehensive literature search was performed across multiple electronic databases, including PubMed, MEDLINE, PubMed Central, Embase, Scopus, Web of Science, and Google Scholar (no publication date restrictions were applied). Additional studies were identified through manual screening of the reference lists of eligible articles, citation tracking of key publications, and consultation with experts in epilepsy where appropriate. Both Medical Subject Headings and regular keywords were used to maximize retrieval of relevant literature.

The search strategy combined terms related to epilepsy and seizure semiology using Boolean operators. The search terms included combinations of the following terms: “epilepsy,” “epileptic seizure,” “seizure semiology,” “clinical semiology,” “pre-ictal,” “prodrome,” “aura,” “ictal,” “ictal phenomenology,” “post-ictal,” “postictal state,” “Todd’s paralysis,” “seizure localization,” “epilepsy classification,” and “video-EEG.” Search strategies were adapted to the indexing system of each database.

2.4. Study selection process

The study selection process was conducted in three stages. First, duplicates were manually removed from the retrieved studies to ensure accuracy. Second, two independent reviewers screened titles and abstracts for relevance. Finally, the same independent reviewers performed a full-text review of eligible articles. Studies meeting eligibility

criteria were included in the final synthesis. Because this review aimed to comprehensively map the breadth of available evidence, review articles, consensus statements, and clinical guidelines were included to provide contextual information and support thematic synthesis but were not considered primary evidence when describing epidemiological or clinical findings.

2.5. Data charting and synthesis

Data from eligible studies were extracted using a standardized data-charting form developed by the review team. Information extracted included the first author, publication year, country of origin, study design, study population, sample size, age group (adult or pediatric), seizure type, phase of seizure investigated (pre-ictal, ictal, or post-ictal), principal semiological findings, neuroanatomical localization where applicable, and diagnostic or clinical significance.

Extracted data were organized into three major thematic domains corresponding to the clinical phases of epileptic seizures:

- (i) Pre-ictal phenomenology, including prodromal symptoms, aura characteristics, epidemiology, and clinical significance.
- (ii) Ictal phenomenology, including seizure classification, motor and non-motor manifestations, localization and lateralization features, and differentiation from psychogenic non-epileptic seizures.
- (iii) Post-ictal phenomenology, including neurological, cognitive, psychiatric, autonomic, and systemic manifestations together with their diagnostic and prognostic implications.

Discrepancies were resolved by consensus discussion or by a third reviewer. Findings extracted from the included studies were synthesized using a narrative approach, with information summarized and presented through text, tables, and figures consistent with scoping review methodology.

3. Results

3.1. Literature search results

A comprehensive search of PubMed, MEDLINE, PubMed Central, Embase, Scopus, Web of Science, and Google Scholar identified 1,120 records. Following the removal of duplicate records, 641 articles remained for title and abstract screening. After screening, 503 articles were excluded because they did not meet the predefined eligibility criteria. Full-text assessment was subsequently performed on 138 articles, of which 60 studies fulfilled the inclusion criteria and were included in the final scoping

review. Figure 1 presents the study selection process in the Preferred Reporting Items for Systematic Reviews and Meta-Analyses Extension for Scoping Reviews flow diagram.

3.2. Characteristics of included studies

A total of 60 studies were included in this scoping review. The included literature consisted of original observational studies, prospective and retrospective clinical studies, systematic reviews and meta-analyses, narrative reviews, consensus statements, and international clinical practice guidelines. Collectively, these studies investigated the clinical manifestations, epidemiology, neurobiological basis, diagnostic significance, localization value, and clinical applications of seizure semiology in both adult and

pediatric populations. The majority of publications were journal articles, with relatively few conference proceedings and book chapters. Most studies originated from specialized epilepsy centers and tertiary neurological institutions and focused primarily on focal epilepsy, temporal lobe epilepsy, generalized epilepsy, or populations with mixed epilepsy types. While many investigations evaluated individual components of seizure semiology, relatively few comprehensively described clinical manifestations across all three phases of epileptic seizures.

3.3. Pre-ictal phenomenology

3.3.1. Background

The term pre-ictal state refers to the period preceding the clinical onset of a seizure during which physiological,

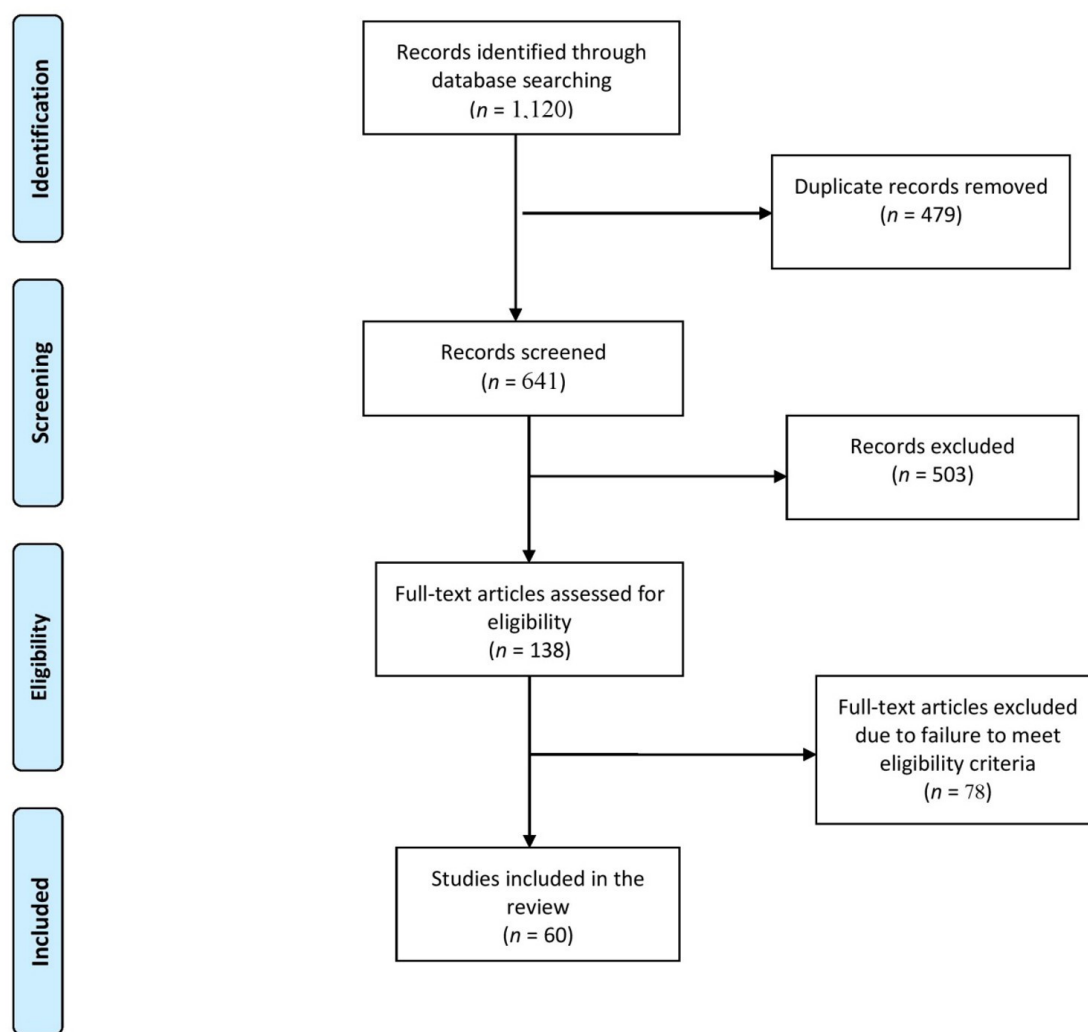


Figure 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses Extension for Scoping Reviews flow diagram illustrating the study selection process

electrophysiological, or behavioral changes may occur.¹⁴ The period is characterized by abnormal subjective sensations or altered behavior seconds, minutes, hours, or even days before the onset of a seizure, which help predict impending seizure events. Pre-ictal symptoms are promising non-invasive clinical biomarkers for seizure prediction, and they reflect a pre-seizure state in which underlying neuronal instability begins to manifest clinically before the actual seizure discharge.¹⁵

Although pre-ictal symptoms are not universally experienced and lack the specificity required for reliable clinical prediction, they hold increasing relevance in both patient self-management and research on seizure forecasting. Recognizing these symptoms may help affected individuals take precautionary measures, reduce injury risk, and better understand seizure patterns.¹⁶

Two clinically recognized manifestations may arise during this period: prodromal symptoms and auras. Although both may precede an overt seizure, they differ fundamentally in their timing and pathophysiological significance. Table 1 presents the comparison between the pre-ictal state, prodromes, and aura.

3.3.2. Classification

(a) Aura

The term aura is derived from the Greek word meaning “breeze” and has long been used to describe the earliest subjective manifestation of an epileptic seizure.^{17,18} In

a proposal for a glossary of epilepsy terminology by the Commission of the International League Against Epilepsy (ILAE), an aura is defined as “that portion of the seizure which occurs before consciousness is lost and for which memory is retained afterwards.”¹⁹ Therefore, aura symptoms are an integral part of epileptic seizures, representing a subjective sensory, emotional, autonomic, or cognitive experience that precedes an observable seizure.

Unlike the prodrome, which is a warning sign that can occur hours or days before a seizure, an aura is not merely a warning sign; it reflects the actual onset of focal ictal activity in the brain in its early phase before it propagates to the other brain regions. Because it represents the earliest expression of focal cortical dysfunction, consciousness is often preserved during the aura, and therefore patients can reliably describe and localize these phenomena, making auras important clinical tools in localizing the site of seizure onset in the cortical region within the cortex of a given hemisphere.

However, auras cannot reliably be used for lateralizing the seizure onset site, that is, determining which cortical hemisphere is involved (left or right). Although some aura types, such as aphasic aura, ictal speech, and orgasmic aura, may provide lateralizing clues, localization is generally more robust than lateralization.^{3,18} Auras classically occur several seconds to a few minutes before seizure onset.²⁰ Conventionally, auras are considered indicative only of focal rather than generalized seizures; however, this notion

Table 1. Comparison of the pre-ictal state, prodrome, and aura

Feature	Pre-ictal state	Prodrome	Aura
Definition	Overall period preceding seizure onset	Clinical symptoms preceding seizure but not part of it	Earliest subjective manifestation of the seizure
Relationship to seizure	Encompasses prodrome and/or aura	Not part of seizure	Part of the seizure (initial ictal phase)
Typical timing	Seconds to days before seizure	Minutes to days before seizure	Seconds to minutes before observable seizure
Pathophysiology	Progressive network instability before seizure	Pre-seizure physiological changes	Focal epileptic discharge
Clinical manifestations	May include prodromal symptoms and aura	Mood, behavioral, cognitive, autonomic, sleep, or somatic symptoms	Sensory, psychic, autonomic, visual, auditory, olfactory, gustatory, or somatosensory symptoms
Localizing value	Limited	Minimal	High; helps localize seizure onset
Clinical significance	Represents the transition toward seizure onset	Useful for seizure prediction and patient safety	Useful for seizure classification and presurgical localization

has been disputed and challenged by many studies.²¹

(b) Prodrome

The term prodrome refers to clinical symptoms that precede seizure onset but are not part of the seizure itself. It is defined as “a subjective or objective clinical alteration, such as an ill-localized sensation or agitation, that heralds the onset of an epileptic seizure but does not form part of it.”¹⁹ Prodromes are typically distinguished from auras by their long duration before seizure onset, but there is no consensus on the pre-seizure threshold, defined as the minimum duration of prodrome.

According to some scholars, the minimum duration is between 30 minutes and 1 hour, implying that symptoms of shorter duration would not be considered prodromes. However, some clinicians extend the duration of the prodrome up to two days.^{15,22} Similarly, there is considerable debate among scholars on what constitutes the typical difference between prodrome and auras. The majority considers time as the yardstick, but some consider the clinical characteristics of the symptoms. Some scholars have argued that the distinct semiology of the prodrome, rather than the time interval to seizure onset, should be the temporal criterion for distinguishing between prodrome and aura; however, the timeframe remains the most commonly used criterion by both patients and clinicians.²³

3.3.3. Epidemiology

(a) Epidemiology of prodromal symptoms

The reported prevalence of prodromal symptoms varies considerably because of differences in study populations and diagnostic definitions, with estimates generally ranging from 10% to 40% and an average prevalence of approximately 20%.^{1,15,23,24} Prodromal symptoms are more frequently reported in patients with frequent seizures, focal impaired-awareness seizures, and generalized tonic-clonic seizures than in those with absence or myoclonic seizures.^{15,23} Higher seizure frequency is an important risk factor for experiencing prodromes, and one multicenter study reported prodromal symptoms in approximately 32.9% of patients with generalized tonic-clonic seizures and 32.6% of those with focal impaired-awareness seizures, compared with only 7.6% of patients with myoclonic seizures and none with absence seizures.^{1,23} Prodromal symptoms most commonly develop between 30 minutes and 24 hours before seizure onset, with many studies reporting typical warning periods of 1–3 hours; however, their onset may range from a few minutes to several days, including insidious prodromes occurring 24–48 hours before seizures.¹⁵

(b) Epidemiology of auras in epilepsy

The reported prevalence of auras ranges from approximately 20% to over 80%, depending on the epilepsy subtype and assessment methodology.^{25–27} Although traditionally regarded as a hallmark of focal epilepsy, particularly temporal lobe epilepsy, accumulating evidence demonstrates that auras are also common in generalized epilepsy, with one cohort reporting aura symptoms in 64.3% of patients, including cephalic sensations, limb stiffening, visual and auditory phenomena, déjà vu, gustatory hallucinations, and nonspecific somatosensory or vegetative symptoms.^{26,27}

Demographic associations remain inconsistent; while one study reported that women were more likely to experience auras, this difference may reflect variations in age distribution rather than a true sex effect, and most large studies have not demonstrated robust associations between aura occurrence and demographic characteristics.^{25,26} However, younger age at seizure onset has been associated with an increased likelihood of headache aura in patients with generalized epilepsy.²⁸ Auras typically occur several seconds to several minutes before seizure onset, providing important information for seizure classification and localization; nevertheless, although they generally maintain a consistent temporal relationship with the seizures they precede, not all auras progress to clinically evident seizures.²⁰

3.3.4. Clinical semiology

(a) Prodromal symptoms

Prodromal symptoms represent early manifestations preceding seizure onset and encompass autonomic, psychiatric, cognitive, sleep-related, and somatic disturbances. Autonomic symptoms reflect altered autonomic nervous system regulation and include sweating, palpitations, gastrointestinal discomfort, diarrhea, vomiting, piloerection, flushing, pallor, and abnormal temperature sensations such as chills or heat (dysthermic sensation). Although piloerection and skin color changes are more commonly observed during seizures, they may also occur pre-ictally due to autonomic and vascular alterations.^{1,29}

Psychiatric symptoms comprise behavioral, emotional, and mood changes, including social withdrawal, hyperactivity, irritability, anxiety, depression, and marked emotional fluctuations ranging from sadness to euphoria, which may be subtle but are often recognized by caregivers.^{1,30} Cognitive symptoms include impaired concentration, attention deficits, confusion, and dysmnestic experiences such as déjà vu and jamais vu, particularly in temporal lobe epilepsy, and may serve as important early warning signs of seizure onset.^{1,31}

Sleep-related symptoms, particularly in individuals with nocturnal epilepsy, include insomnia, difficulty initiating or maintaining sleep, hypersomnia, excessive daytime sleepiness, and increased seizure susceptibility following poor sleep, underscoring the relationship between circadian rhythms and epileptogenesis.^{1,32} Somatic symptoms include headache, migraine, dizziness, vertigo, myalgias, generalized bodily discomfort, and fatigue, with many patients reporting unusual tiredness or physical exhaustion preceding seizure events, particularly after prolonged physical or mental exertion.^{1,33}

Table 2 summarizes the prodromal indicators.

Table 2. Summary of prodromal indicators

Category	Common symptoms
Autonomic	Palpitations, sweating, flushing/pallor, piloerection, and diarrhea
Psychiatric	Irritability, anxiety, euphoria, and mood swings
Cognitive	Confusion, lack of focus, and déjà vu
Somatic	Headache, fatigue, and dysthermic sensations
Sleep-related	Insomnia, hypersomnia, and excessive daytime sleepiness

(b) Aura symptoms

Aura symptoms represent focal seizure manifestations arising from activation of specific cortical and subcortical networks. They include somatosensory, visual, auditory, olfactory/gustatory, psychic, autonomic, and nonspecific auras.

Somatosensory auras, originating from the primary or secondary somatosensory cortex (typically the parietal lobe), present with unilateral tingling, numbness, “pins and needles,” electric shock-like, or crawling sensations affecting a limb, the face, or one side of the body, and may demonstrate a Jacksonian march along the somatotopic cortex.^{3,25,34,35}

Visual auras result from epileptic activity in the occipital or posterior temporo-occipital regions and range from simple visual hallucinations, including flashing lights, colored spots, or geometric shapes, to complex hallucinations involving detailed images of people, animals, or objects, with simple phenomena typically arising from the primary visual cortex and complex manifestations

arising from the temporo-occipital visual association cortex.^{35,36}

Auditory auras, arising from the superior temporal gyrus (Heschl’s gyrus), manifest as simple auditory hallucinations such as buzzing, ringing, humming, clicking, or hissing, or complex hallucinations including voices, music, or environmental sounds, with the latter being more characteristic of temporal lobe epilepsy involving the language-dominant hemisphere.^{35,37,38}

The olfactory and gustatory auras—classic features of mesial temporal lobe epilepsy involving the amygdala, hippocampus, and orbitofrontal cortex—are characterized by unpleasant smells such as burning rubber or rotten eggs and abnormal taste sensations, particularly bitter tastes. Although relatively uncommon, olfactory auras are highly localizing for temporal lobe epilepsy.^{35,39} Psychic auras, typically associated with activation of limbic and temporal lobe structures, include dysmnestic phenomena such as déjà vu and jamais vu, as well as affective symptoms including intense fear, panic, anxiety, impending doom, depersonalization, and derealization, reflecting dysfunction of the hippocampus and amygdala.^{40,41}

Autonomic auras, among the most frequently reported aura types, arise from seizure activity involving the insula, limbic system, and hypothalamic pathways, producing cardiovascular symptoms (e.g., palpitations, tachycardia, bradycardia), respiratory symptoms (dyspnea), gastrointestinal symptoms (including the characteristic epigastric rising sensation of mesial temporal lobe epilepsy), cutaneous manifestations (e.g., sweating, flushing, pallor, piloerection), sexual or genital sensations including the rare orgasmic aura associated with right parietal lobe seizures, and urinary urgency or bladder fullness. These manifestations are clinically important because of their association with cardiorespiratory dysfunction and potential implications for the risk of sudden unexpected death in epilepsy.^{3,29,42,43}

Finally, nonspecific auras comprise vague, difficult-to-characterize sensations, including indescribable strange feelings, cephalic sensations, a sense that something is about to happen, or internal discomfort and unease, which, despite their nonspecific nature, remain clinically important because they are commonly reported by patients with epilepsy.^{17,35}

Table 3 summarizes the aura symptoms and the localization.

In summary, the pre-ictal period encompasses a spectrum of clinical phenomena that differ in both timing and biological significance. Prodromal symptoms reflect pre-seizure alterations that may occur minutes

to days before seizure onset and can facilitate seizure forecasting and patient self-management. In contrast, an aura represents the earliest clinical expression of focal ictal activity and therefore forms part of the seizure itself while providing important information regarding seizure localization and classification. Distinguishing these entities is essential for accurate clinical assessment and appropriate interpretation of patient-reported symptoms.⁴⁴

Table 3. Summary of aura symptoms and localization

Aura category	Common symptoms	Likely brain region
Epigastric rising	Butterfly sensation in stomach	Mesial temporal lobe
Somatosensory	Tingling/numbness	Parietal lobe
Visual	Flashing lights/lines	Occipital lobe
Olfactory	Burning smell	Uncus/mesial temporal
Psychic	Fear/déjà vu	Amygdala/hippocampus

3.4. Ictal phenomenology

3.4.1. Background

The ictal phase is the period of the actual seizure and refers to the period during which a seizure is actively occurring, representing the core clinical manifestation of epilepsy. The ictal phase begins with the onset of abnormal electrical discharges in the brain and ends when normal cortical activity is restored or when the seizure transitions into the post-ictal state. It includes all objective signs and symptoms occurring from the onset to the termination of the seizure. The symptoms observed depend on the brain region where the seizure originates and how it spreads, the seizure type, and the degree of consciousness impairment.⁴⁵

In focal seizures, ictal symptoms may be localized, such as unilateral motor jerking, sensory distortions, or autonomic changes, with or without impairment of awareness. In contrast, generalized seizures involve widespread bilateral networks from onset, resulting in loss of consciousness and bilateral motor manifestations, as observed in tonic-clonic seizures. The duration of the ictal phase is usually brief, ranging from a few seconds to several minutes, but prolonged ictal activity may lead to significant metabolic stress, neuronal injury, and systemic complications.^{46,47} Understanding the ictal phase also informs acute management strategies, guides antiepileptic treatment selection, and plays a critical role in counseling patients and caregivers regarding seizure recognition and

safety.

3.4.2. Semiologic classification

Seizures are generally classified according to the most recent ILAE classification of epileptic seizures into four categories: focal seizures, generalized seizures, unknown (whether focal or generalized) seizures, and unclassified seizures. Focal seizures are described in terms of observable phenomena (e.g., motor phenomena) and non-observable features (e.g., cognitive or sensory experiences). They also include semiological descriptors such as automatisms, autonomic changes, and epileptic spasms. Some focal seizures may subsequently evolve into focal seizures with bilateral tonic-clonic features.

Generalized seizures include those with observable manifestations, such as generalized tonic-clonic activity, tonic, clonic, atonic, myoclonic, myoclonic-tonic-clonic, and myoclonic-atonic phenomena, as well as generalized absence phenomena and epileptic spasms. Seizures of unknown onset are seizures that occur either in sleep or in a condition that cannot be described.

Additionally, when the clinician is certain that the information given is a seizure event but cannot classify it due to insufficient information, it is regarded as an unclassified seizure.⁴⁸ Therefore, the term unknown onset describes the available level of information rather than a characteristic of the seizure itself.

3.4.3. Semiologic terms

Semiologic terms describe the observable or experiential features of seizures and include the following.⁴⁸⁻⁵⁰

- (i) Automatisms, referring to involuntary automatic behaviors.
- (ii) Emotional manifestations, such as the appearance or expression of emotions.
- (iii) Tonic activity, characterized by sustained muscle contraction with extension or flexion postures.
- (iv) Autonomic features include sweating or flushing.
- (v) Myoclonic activity, defined as brief, arrhythmic muscle jerks.
- (vi) Clonic activity, consisting of rhythmic jerking movements.
- (vii) Cognitive symptoms, including language or thinking disturbances and phenomena such as déjà vu.
- (viii) Eyelid myoclonia, which involves repetitive eyelid jerking.
- (ix) Atonic activity, marked by sudden loss of muscle tone.
- (x) Sensory symptoms, such as abnormal sensations including tingling or numbness.
- (xi) Behavioral arrest, referring to a pause or cessation of

ongoing activity.

- (xii) Hyperkinetic activity, characterized by vigorous movements such as thrashing or pedaling.
- (xiii) Spasms, which involve sudden, brief contractions of the limbs or trunk.

3.4.4. Common descriptors

Common descriptors are recommended descriptive terms rather than seizure types and are used to characterize seizure features. These include the following:⁴⁸⁻⁵⁰

- (i) Motor descriptors, such as paralysis, paresis, dystonia, dysarthria, incoordination, and acalculia.
- (ii) Automatism descriptors, including aggression, eye blinking, head nodding, pedaling, pelvic thrusting, vocalization or speech, and walking.
- (iii) Cognitive descriptors, such as aphasia, attention impairment, memory impairment, déjà vu, hallucinations, illusions, forced thinking, and acalculia.
- (iv) Emotional or affective descriptors, including agitation, anger, anxiety, fear, crying, laughing, and pleasure.
- (v) Autonomic descriptors, such as bradycardia, tachycardia, palpitations, flushing, genital erection, and asystole.
- (vi) Sensory descriptors, including auditory, visual, olfactory, gustatory, and somatosensory experiences.
- (vii) Laterality descriptors, specifying left-sided, right-sided, or bilateral involvement.

3.4.5. Clinical semiology

Clinical semiology refers to the observable clinical manifestations and temporal evolution of a seizure, including changes in motor activity, awareness, sensation, autonomic function, cognition, emotion, and behavior. A detailed evaluation of semiological features is essential not only for establishing a diagnosis of epilepsy but also for differentiating epileptic seizures from non-epileptic paroxysmal events, classifying seizure types, and determining the lateralization and localization of the epileptogenic zone. Clinical semiology therefore provides an important link between the patient's observed ictal manifestations and the underlying neuroanatomical and network mechanisms of seizure generation and propagation. In accordance with the updated ILAE framework, epileptic seizures are categorized based on their clinical onset into focal, generalized, or unknown-onset, with unclassified seizures reserved for events with insufficient diagnostic data. The specific semiological features associated with each of these major classes, along with their clinical localization value and differential diagnostic relevance, are outlined below.

(a) Focal seizures

According to the 2025 ILAE classification, focal seizures originate within networks confined to one cerebral hemisphere and are categorized according to the level of consciousness as focal preserved consciousness seizures (FPCS) and focal impaired consciousness seizures. Some focal seizures subsequently evolve into focal-to-bilateral tonic-clonic seizures, replacing the former term "secondary generalized tonic-clonic seizures."^{48,51-53}

(b) Focal preserved consciousness seizure

An FPCS is a type of focal seizure that originates within a network confined to one cerebral hemisphere and, throughout its course, does not impair the person's level of consciousness (i.e., the individual remains both aware of themselves and their surroundings and can respond appropriately to external stimuli); patients can typically recall the event afterward. Clinical manifestations depend on the cortical region involved and may include motor manifestations (e.g., clonic, tonic, myoclonic, dystonic, or hyperkinetic movements), sensory symptoms (e.g., visual, auditory, somatosensory, olfactory, or gustatory phenomena), autonomic manifestations (e.g., epigastric rising sensation, sweating, flushing, or palpitations), cognitive disturbances (e.g., aphasia, forced thinking, or memory impairment), emotional symptoms (e.g., fear, anxiety, or déjà vu), or behavioral arrest. Because the earliest manifestations closely reflect the seizure onset zone, FPCS provide important information for seizure localization.

(c) Focal impaired consciousness seizure

A focal impaired consciousness seizure is a focal seizure in which the abnormal electrical activity begins in one hemisphere but, at some point during the event, results in impaired consciousness, defined operationally as reduced awareness and/or responsiveness to the environment. Patients commonly exhibit behavioral arrest, staring, impaired responsiveness, orolimentary or manual automatisms, and post-ictal confusion or amnesia. Temporal lobe epilepsy frequently presents with epigastric sensations followed by impaired consciousness and automatisms, whereas frontal lobe epilepsy more commonly manifests with brief hyperkinetic movements, asymmetric tonic posturing, or bizarre motor behaviors.

(d) Focal-to-bilateral tonic-clonic seizure

A focal-to-bilateral tonic-clonic seizure is a focal seizure that initially arises in a localized region of one cerebral hemisphere and then propagates to involve both hemispheres, with the semiology evolving into a bilateral tonic-clonic phase characterized by widespread

stiffening (tonic) followed by rhythmic jerking (clonic) of the muscles and impairment of consciousness during the bilateral phase; this type reflects seizure spread beyond the focal network and replaces the older term “secondary generalized tonic–clonic seizures.” Recognition of the initial focal manifestations remains clinically important because they frequently identify the epileptogenic region and guide presurgical evaluation.

3.4.6. Generalized seizures

Generalized seizures engage bilateral neuronal networks from onset and are broadly classified into generalized non-motor (absence) and generalized motor seizures.^{48,51–54}

(a) Generalized non-motor (absence) seizures

These seizures are characterized by abrupt impairment of consciousness with behavioral arrest and staring lasting only a few seconds, followed by rapid recovery without a significant post-ictal state. The principal subtypes include typical absence, atypical absence, myoclonic absence, and eyelid myoclonia with or without absence seizures.

Typical absence seizures have abrupt onset and termination, whereas atypical absences are more prolonged, have less abrupt onset and termination, with often more pronounced changes in muscle tone or motor behavior, and are commonly associated with developmental epileptic encephalopathies. Myoclonic absence seizures have abrupt onset and offset, but can last longer than typical absences and are accompanied by bilateral, rhythmic myoclonic jerks, typically involving the upper limbs, sometimes the head or other muscles. Eyelid myoclonia with/without absence refers to generalized epileptic events characterized by brief, repetitive myoclonic jerks of the eyelids frequently accompanied by upward eye deviation, which may occur with or without concurrent absence seizures.

(b) Generalized motor seizures

Generalized motor seizures include generalized tonic–clonic, tonic, clonic, myoclonic, myoclonic–tonic–clonic, myoclonic–atonic, atonic, epileptic spasms, and generalized negative myoclonic seizures. Generalized tonic–clonic seizures typically present with sudden loss of consciousness followed by tonic stiffening and rhythmic bilateral clonic jerking, accompanied by autonomic manifestations and a prolonged post-ictal period.

Myoclonic seizures consist of sudden, brief bilateral shock-like muscle jerks; tonic seizures are characterized by sustained muscle contraction, clonic seizures by rhythmic bilateral jerking, and atonic seizures by abrupt loss of muscle tone resulting in falls (drop attacks). Myoclonic–atonic and myoclonic–tonic–clonic seizures combine these motor features. Generalized epileptic spasms are

marked by sudden, brief flexion or extension movements that typically involve the neck, trunk, and extremities simultaneously, often occurring in clusters and commonly seen in early childhood syndromes. Generalized negative myoclonic seizures are characterized by brief interruptions or loss of muscle activity (negative myoclonus) across bilateral muscle groups, such as sudden lapses in posture or dropping objects due to a transient absence of muscle tone, reflecting a transient bilateral inhibitory event in motor circuits.

3.4.7. Unknown-onset seizures

Unknown-onset seizures are diagnosed when available clinical or electroencephalographic information is insufficient to determine whether seizure onset is focal or generalized. Depending on the observed semiology, these seizures may occur with preserved consciousness, impaired consciousness, or bilateral tonic–clonic activity. Classification should be revised whenever additional clinical information, EEG findings, or video-EEG monitoring become available.^{48,51–53}

3.4.8. Unclassified seizures

An unclassified seizure refers to an event that the clinician is confident represents an epileptic seizure, but for which there is insufficient information available to categorize it into any of the defined seizure classes. This can occur when clinical details, witness accounts, or EEG data are too limited or ambiguous to determine the seizure’s onset type (focal or generalized), semiology, or state of consciousness, preventing reliable classification. As more information becomes available later, for example, through observation of subsequent events, EEG recordings, or additional history, an unclassified seizure may be reclassified into an appropriate seizure class.^{48,51–53}

3.4.9. Clinical significance of ictal semiology for seizure localization

Ictal semiology provides valuable information regarding the localization and propagation of epileptic activity. Early unilateral tonic or clonic movements suggest involvement of the contralateral primary motor cortex, whereas dystonic posturing of one upper limb often indicates seizure onset within the ipsilateral temporal lobe with propagation to the basal ganglia.⁵⁵ Automatism, particularly oroalimentary and manual automatisms, are characteristic of temporal lobe epilepsy, whereas hyperkinetic movements are more suggestive of frontal lobe epilepsy.⁵⁶

Epigastric sensations, autonomic symptoms, and psychic phenomena frequently localize to mesial temporal structures, whereas visual, auditory, somatosensory, and olfactory symptoms indicate involvement of the

occipital, temporal, parietal, and mesial temporal cortices, respectively.⁵⁷ Although no individual semiological sign is perfectly localizing, careful interpretation of the sequence and evolution of ictal manifestations substantially improves localization of the epileptogenic zone, particularly when integrated with EEG and neuroimaging findings.⁵⁸

3.4.10. Epileptic seizures and psychogenic non-epileptic seizures

Differentiating epileptic seizures from psychogenic non-epileptic seizures remains an important clinical challenge because both conditions may present with apparent convulsive-like episodes.⁵⁹ Certain ictal features favor psychogenic non-epileptic seizures, including prolonged seizure duration, asynchronous or fluctuating limb movements, side-to-side head movements, pelvic thrusting, tightly closed eyes with resistance to opening, preserved awareness despite bilateral motor activity, ictal crying, and rapid recovery without a typical post-ictal period.^{59,60}

In contrast, epileptic seizures more commonly demonstrate stereotyped semiology, tonic-clonic evolution, open eyes during generalized tonic-clonic seizures, tongue biting (particularly lateral tongue biting), urinary incontinence, and a characteristic post-ictal phase with confusion or somnolence.^{59,60} Nevertheless, no individual clinical sign is sufficiently sensitive or specific to establish the diagnosis, and prolonged video-EEG monitoring remains the diagnostic gold standard when uncertainty exists.⁶¹

3.5. Post-ictal phenomenology

3.5.1. Background

The post-ictal phase is the recovery period that follows the end of a seizure and lasts until the patient returns to their usual baseline state.⁶² The duration and severity of symptoms vary widely based on the seizure type and characteristics. It may take hours to days, as in patients with generalized and focal seizures, or only a few seconds or minutes, as in individuals with absence seizures.⁶² The post-ictal period is often accompanied by slowing or suppression on EEG and may reflect reversible functional disturbances rather than permanent structural injury.

Severity ranges from brief disorientation lasting minutes to prolonged deficits persisting for hours or, rarely, days, and is usually determined by seizure type, seizure duration, and underlying brain pathology; for many people, the immediate post-ictal symptoms are the most disabling part of the seizure-related burden.⁶³ Because post-ictal phenomena substantially affect morbidity and safety, and can mimic other medical emergencies such as

stroke, clinicians should screen for and document post-ictal symptoms, counsel patients and caregivers about typical timelines, and consider targeted interventions like seizure control, safety planning, and psychiatric input when psychosis or prolonged mood disturbance occurs.⁶⁴

3.5.2. Epidemiology

Post-ictal manifestations are experienced by most individuals with epilepsy, although symptom frequency varies according to seizure type and patient characteristics. Post-ictal headache occurs in approximately one-third of patients, whereas post-ictal migraine and psychosis occur in approximately 16% and 4%, respectively. Generalized tonic-clonic seizures, status epilepticus, prolonged seizures, temporal lobe epilepsy, structural brain lesions, and pre-existing cognitive impairment are associated with more severe or prolonged post-ictal manifestations. Most symptoms resolve within minutes to several hours; however, focal neurological deficits, mood disturbances, and post-ictal psychosis may persist for days.^{63,65,66}

3.5.3. Clinical semiology

The post-ictal state represents a transient period of altered neurological function following the termination of an epileptic seizure. Characterized by temporary cerebral dysfunction and complex neurophysiological changes, post-ictal semiology encompasses a diverse spectrum of manifestations that vary according to seizure type, duration, and anatomical networks involved. Beyond their immediate diagnostic value in identifying recent seizure activity, post-ictal features often provide important lateralizing and localizing clues regarding the epileptogenic zone while assisting in the differentiation of post-ictal phenomena from ongoing non-convulsive status epilepticus or acute structural brain injury. The principal neurocognitive, neurological, somatic, and psychiatric manifestations of the post-ictal state are described in the following subsections and summarized in [Table 4](#).

(a) Neurocognitive manifestations

Neurocognitive symptoms are among the most common post-ictal manifestations and include confusion, disorientation, amnesia, and aphasia. Post-ictal confusion and disorientation range from mild cognitive slowing to profound impairment of consciousness, during which patients may be unable to recognize familiar people or surroundings, respond appropriately to questions, or make coherent decisions. These symptoms are particularly common after generalized tonic-clonic seizures and in individuals with underlying structural brain lesions.^{67,68}

Transient post-ictal amnesia commonly affects memory of the seizure and the immediate peri-ictal period

owing to temporary dysfunction of the hippocampal and related memory networks. It should be distinguished from transient epileptic amnesia, a distinct epilepsy syndrome typically associated with temporal lobe epilepsy.⁶⁹ Post-ictal aphasia is characterized by transient impairment of speech production and/or comprehension following seizures involving the dominant temporal or frontal language networks and may provide useful lateralizing information during epilepsy evaluation.⁷⁰

(b) Neurological deficits

Transient focal neurological deficits are well-recognized post-ictal manifestations. The most characteristic example is Todd's paralysis (Todd's paresis), a temporary focal weakness or paralysis affecting one limb or one side of the body that usually resolves within minutes to 48 hours. However, it may last much longer. Because the deficit typically occurs contralateral to the epileptogenic focus, it provides valuable localizing information and should be distinguished from acute neurological emergencies such as acute ischemic stroke.⁷¹

(c) Somatic manifestations

Somatic symptoms are common during post-ictal recovery and include fatigue, excessive somnolence, headache, muscle soreness, nausea, and vomiting. Fatigue and sleepiness, particularly after generalized tonic-clonic seizures, reflect transient neuronal metabolic exhaustion and often result in prolonged sleep lasting several minutes to hours.⁶⁸ Post-ictal headaches are frequently throbbing or migraine-like and are thought to result from neurovascular dysregulation and metabolic stress associated with intense neuronal firing.⁷² Generalized muscle aches arise from sustained tonic and clonic muscle contractions during convulsive seizures, whereas nausea and vomiting are attributed to transient autonomic dysregulation. These somatic manifestations are generally self-limiting and resolve with supportive care.^{51,54,55,59}

(d) Psychiatric manifestations

Psychiatric symptoms range from transient emotional disturbances to more severe neuropsychiatric complications. Patients may experience irritability, agitation, anxiety, emotional lability, crying spells, or depressed mood during the immediate post-ictal period.⁷³ Post-ictal psychosis, although uncommon, is a clinically significant complication that typically develops after a lucid interval of several hours to a few days following a seizure cluster. It is characterized by hallucinations, delusions, paranoia, aggression, or marked behavioral disturbances and usually requires prompt neurological assessment and psychiatric management after exclusion of ongoing seizures or other underlying medical causes.⁵¹

Table 4 summarizes the key post-ictal symptoms.

Table 4. Key post-ictal symptoms

Symptom	Typical features	Clinical notes
Disorientation and confusion	Inability to think clearly or recognize environment	Commonest immediate symptom
Fatigue and somnolence	Deep sleep or extreme tiredness	Can last hours
Headache	Severe and throbbing	May resemble migraines
Amnesia	Loss of memory of ictal period and surroundings	Can be ictal seizure event or post-ictal feature
Aphasia	Speech disturbance	Linked to language cortex seizure focus
Todd's paralysis	Transient focal weakness or paralysis	Useful for lateralizing seizure focus
Muscle aches	Soreness post-contraction	Typical after convulsions

4. Discussion

This scoping review maps the breadth of current evidence on the clinical semiology of epileptic seizures across the pre-ictal, ictal, and post-ictal phases, emphasizing that seizure manifestations represent a dynamic continuum rather than isolated clinical events. The findings demonstrate that careful characterization of seizure semiology remains fundamental to epilepsy diagnosis, seizure classification, localization of the epileptogenic zone, and individualized patient management. Recognition of prodromal symptoms may facilitate seizure anticipation and patient safety, whereas detailed analysis of ictal manifestations provides valuable localizing and lateralizing information that complements electroclinical and neuroimaging findings.

Furthermore, post-ictal manifestations, although frequently under-recognized in routine clinical practice, offer important insights into seizure propagation, recovery, and potential seizure-related complications, underscoring the importance of evaluating the entire seizure sequence during clinical assessment.^{50,74}

The review also highlights several important knowledge gaps. Although considerable progress has been made in standardizing seizure terminology and improving electroclinical characterization, evidence describing

the complete evolution of seizure semiology across all three phases remains poor, with most studies focusing predominantly on ictal phenomena.

Nevertheless, several limitations remain, including heterogeneous study designs, narrative synthesis, publication bias, absence of formal quality appraisal of the included studies, and inability to quantify associations. Future research should prioritize prospective multicenter studies employing standardized semiological terminology, synchronized video-EEG monitoring, wearable seizure-detection technologies, and multimodal neurophysiological and neuroimaging approaches to better define temporal patterns of seizure evolution and improve seizure prediction, localization, and personalized epilepsy care. Such efforts may enhance diagnostic accuracy, optimize presurgical evaluation, facilitate earlier therapeutic intervention, and ultimately improve clinical outcomes and quality of life for individuals living with epilepsy. In addition, further studies are needed to better characterize semiological patterns in genetic and developmental epilepsies and to determine how these observations can support precision diagnosis and individualized treatment. Continued integration of clinical expertise with emerging technologies will further enhance diagnostic accuracy, improve patient outcomes, and advance personalized epilepsy care.^{75,76}

5. Conclusion

Comprehensive assessment of pre-ictal, ictal, and post-ictal phenomenology remains fundamental to the diagnosis and management of epilepsy. Careful evaluation of pre-ictal symptoms may facilitate seizure prediction and timely implementation of preventive measures, while ictal semiology provides essential information for seizure classification, localization of the epileptogenic zone, and presurgical evaluation. Post-ictal manifestations offer complementary diagnostic and prognostic insights, helping to distinguish epileptic seizures from seizure mimics, identify seizure-related complications, and guide patient counseling and follow-up. Although advances in diagnostic technologies, including prolonged video-EEG monitoring, high-resolution neuroimaging, functional neurophysiological techniques, and emerging wearable seizure-detection devices, have substantially improved epilepsy evaluation, meticulous bedside observation and detailed history-taking remain indispensable. Clinical semiology should therefore be viewed as complementary to, rather than replaced by, these modern diagnostic tools.

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The authors declare they have no competing interests.

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