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Review Article

The Occipital Crossroads: Post-Stroke Epilepsy in the Posterior Circulation: A Semiology We Misread, a Risk Score That Cannot Score It, and a Driving Assessment Nobody Owns

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Abstract

The SeLECT score awards points for involvement of the middle cerebral artery territory. A patient with an extensive cortical occipital infarct in the posterior cerebral artery territory therefore scores zero on that item, by construction, regardless of how much cortex is involved. This is not a fault in the score. SeLECT performs as designed in the population it was derived from, which was dominated by anterior circulation disease. It is a boundary condition, and it has been carried into bedside use without being stated. The occipital lobe compounds this in three ways. Its seizures are expressed as visual phenomena that overlap almost entirely with migraine aura, transient monocular visual loss and release hallucinations, so they are systematically attributed elsewhere — most often to migraine, in patients too old to be developing it. Its infarcts produce homonymous field defects that mask new ictal visual symptoms, because the patient already has a visual problem and reports the new one as more of the old one. And a patient with both a hemianopic defect and post-stroke epilepsy faces two separate statutory barriers to driving, assessed by different clinicians against different standards, with neither assessment obliged to know about the other. We review post-stroke epilepsy with specific attention to posterior circulation disease, give a structured framework for telling occipital seizures from their mimics, examine where the existing risk instruments do and do not apply, and propose an eight-item assessment set. Four predictions follow, one of which two of us disagree about and have said so in the text. We are not claiming occipital infarcts are more epileptogenic than others. We are claiming that we currently have no adequate basis for saying whether they are, and that this is not how the scores are being used.

Keywords: *Post-Stroke Epilepsy; Occipital Lobe Seizures; Homonymous Hemianopia; Select Score; Posterior Circulation Stroke; Fitness to Drive*

1. Introduction

A woman of 68 is admitted with a right occipital infarct and a left homonymous hemianopia. Four months later she mentions brief episodes of coloured circles moving across her vision, a minute or two at a time, followed by headache. She is told she has migraine. Nine months after that she has a bilateral tonic–clonic seizure.

In retrospect the coloured circles were focal aware seizures of occipital origin, and she had been having them, untreated, for the better part of a year.

Every step of that misdiagnosis was reasonable at the time. The episodes were brief and self-limiting. They were followed by headache. There was no motor involvement, no impairment of awareness and no witness. She already had a visual deficit, which made new visual symptoms feel like an extension of a known problem rather than a new one. And an interictal EEG, had anyone performed one, would probably have been normal.

The composite is not unusual, and readers who run a stroke clinic will recognise it.

Post-stroke epilepsy has a substantial and improving literature. Our argument here is narrower: that the entity has been characterised largely in anterior circulation disease, and that the occipital lobe raises diagnostic, predictive and medicolegal problems the general literature does not deal with. We take those in turn, and the third of them — driving — is the one we would most like a reader to act on, because it requires no new evidence at all.

2. Definitions, And Why They Decide the Treatment

Precision about definitions is not pedantry here. The definition determines both the reported incidence and whether the patient gets a drug.

Acute symptomatic (early) seizures occur within seven days and are provoked by the acute lesion. They do not by themselves constitute epilepsy, and in the absence of further provocation their recurrence risk is comparatively low.[1]

Unprovoked (late) seizures occur beyond seven days. Under the 2014 ILAE operational definition, one unprovoked seizure in the setting of a structural lesion carrying more than 60% ten-year recurrence risk is enough to diagnose epilepsy.[2] A single late seizure after stroke meets that threshold.

Which means the patient in Section 1 had epilepsy from her first episode of coloured circles. Not from the tonic–clonic seizure nine months later.

The population figures are consistent. In the SeLECT derivation cohort, late-seizure risk was about 4% at one year and 8% at five.[3] National cohort data give a cumulative epilepsy incidence of roughly 3% at two years and 5% at ten after ischaemic stroke, rising to about 9% at two years and 12–13% at ten after intracerebral haemorrhage.[4] Published figures across the wider literature run from 2% to 20%, and that spread is driven by differences in definition, ascertainment and follow-up rather than by real differences between populations.

The strongest single predictor of late epilepsy is an acute symptomatic seizure, and its type matters a great deal. Acute symptomatic status epilepticus was associated with a ten-year epilepsy risk in survivors of 81% or more, against roughly 40% after brief acute symptomatic seizures and about 13% in those with none. Ten-year mortality followed the same gradient.[5] That finding is what drove the recalibration into SeLECT 2.0, which separates brief seizures from status and extends the scale accordingly.[5]

3. Why Late Seizures Happen, and Why it Matters That Cortex Survives

Early and late seizures are different processes.

Early seizures are the acute consequence of ischaemic depolarisation: glutamate release, failure of ATP-dependent ion pumping, extracellular potassium accumulation, calcium influx, peri-infarct spreading depolarisation. These are properties of tissue in metabolic crisis, and they resolve when the crisis does.

Late seizures reflect epileptogenesis: a delayed remodelling of tissue that survived. Reactive astrogliosis with impaired potassium and glutamate buffering. Blood–brain barrier breakdown with albumin extravasation and TGF-β signalling. Aberrant axonal sprouting forming recurrent excitatory circuits. Selective interneuron loss. Chronic neuroinflammation through IL-1β and HMGB1. Haemosiderin after haemorrhagic transformation. Cortical superficial siderosis has emerged as a particularly robust imaging correlate and improves existing risk scores when added to them.[6]

Two features of that account bear directly on the occipital lobe.

Every mechanism listed requires surviving cortex. Complete tissue destruction does not generate epilepsy; the peri-infarct rim does. Occipital infarcts are frequently cortical and frequently incomplete.

And blood–brain barrier disruption with haemosiderin deposition is, if anything, more relevant in posterior circulation disease, given how readily posterior cerebral artery territory infarcts undergo haemorrhagic transformation.

There is no mechanistic reason to expect occipital cortex to be spared. There is also very little direct evidence either way, because the cohorts were never powered to answer the question. That gap is the subject of the next section.

4. A Score That Cannot Score This Territory

SeLECT is the best-validated instrument for predicting late seizures after ischaemic stroke, with a concordance statistic around 0.77 across validation cohorts and good calibration.[3] Its components are stroke severity by NIHSS, large-artery atherosclerotic aetiology, early seizures, cortical involvement, and territory of the middle cerebral artery. The scale runs 0 to 9, and five-year late-seizure risk rises from about 1.3% at zero points to about 63% at nine.[3]

The instrument is sound. Our concern is with how it is used.

Two patients. Patient A has a cortical MCA territory infarct. Patient B has an extensive cortical occipital infarct in the PCA territory, with haemorrhagic transformation and a dense hemianopia. Assume identical severity, identical aetiology, no early seizures in either. Patient A scores on the territory item. Patient B cannot, whatever their imaging shows. The two receive different risk estimates on the basis of a variable that entered the model because it was empirically predictive in a cohort dominated by anterior circulation stroke — not because posterior circulation cortex was demonstrated to be less epileptogenic.

This is an ordinary and well-understood property of prognostic modelling. A score is valid within the case mix it came from, and its behaviour at the edges of that case mix is a question for external validation, not an assumption. The difficulty is that SeLECT is now used at the bedside and in trial eligibility criteria, and the territorial restriction is rarely mentioned when it is.

There is a signal running the other way that deserves weight. In analyses from the SeLECT consortium, acute symptomatic seizures were associated with posterior cerebral artery territory infarcts as well as with severe strokes, cortical involvement and large-artery atherosclerosis.[5] If acute symptomatic seizures are the strongest single predictor of subsequent epilepsy, and PCA territory infarction is associated with acute symptomatic seizures, then the posterior circulation is unlikely to be the benign territory that a naive reading of the score implies.

We want to be careful. We are **not** claiming occipital infarcts are more epileptogenic than MCA infarcts. We are claiming that applying SeLECT to a posterior circulation stroke means applying an instrument outside its validation population, that the direction of the resulting bias is unknown, and that nobody currently says so at the point of use.

Table 1. Applicability of post-stroke epilepsy risk instruments to posterior circulation and occipital stroke.

Instrument	Derived from	Territorial component	Occipital applicability	Principal caveat
SeLECT[3]	Ischaemic stroke, European cohorts	Explicit MCA item	Applicable; item cannot score	Territorial bias of unknown direction
SeLECT 2.0[5]	Ischaemic stroke, multicentre	Retained	As above, better weighting of seizure type	Discrimination improved; territorial limit unchanged
SeLECT-EEG[7]	Ischaemic stroke with early EEG	Retained	Adds electrographic data, potentially valuable posteriorly	Needs early EEG, unavailable in most units
CAVE[8]	Intracerebral haemorrhage	None	Applicable to occipital ICH	Not applicable to ischaemic stroke
IsCHEMiA[9]	Ischaemic stroke, imaging-based	Imaging-derived, not territorial	Potentially territory-neutral	Needs validation in posterior circulation subgroups

5. Telling An Occipital Seizure from What it Looks Like

Occipital lobe epilepsies account for perhaps 5–10% of epilepsies and are widely thought to be underdiagnosed.[10] The reason is not mysterious. Transient visual phenomena have one of the most crowded differentials in neurology, and the discriminating features are qualitative and depend on a history that patients are rarely asked for in enough detail.

The most useful work here is still Panayiotopoulos's, which established that visual seizures and migraine aura can be reliably separated when the elements are taken together, and are often indistinguishable when any single element is taken alone.[11,12] That is the point most often lost in practice, where one feature is used to settle the question. Colour does not settle it. Neither does headache: postictal headache indistinguishable from migraine occurred in around two-thirds of his patients with occipital seizures, including after brief visual seizures with no convulsion.[12]

Table 2 should be used whole. Any single row will mislead.

Table 2. Discriminating transient visual phenomena in the stroke survivor.

Feature	Occipital seizure	Migraine aura	Monocular visual loss	Release hallucination	Recrudescence
Onset to peak	Seconds	5–20 min	Seconds	Variable	Seconds–minutes
Duration	1–3 min	5–60 min	Seconds–minutes	Minutes–hours	Minutes–hours
Form	Coloured, circular, multiplying	Zigzag, scintillating, angular	Negative; greying or curtain	Complex, formed; faces, figures	Prior deficit
Field	Binocular hemifield; peripheral to central	Binocular hemifield; centrifugal march	**Monocular**	Within blind field	Original territory
Positive/negative	Positive; may end in amaurosis	Positive then negative	Negative only	Positive	Negative

Table 2. Continued...

Feature	Occipital seizure	Migraine aura	Monocular visual loss	Release hallucination	Recrudescence
Stereotypy	High; clustered	Variable	Variable	Elaborate, variable	Stereotyped
Frequency	Daily or weekly clusters	Weeks–months apart	Sporadic	Often daily	Precipitant-related
Insight	May be impaired	Retained	Retained	Retained; known unreal	Retained
Progression	To eye deviation, temporal features, bilateral tonic–clonic	To sensory or aphasic aura	None	None	None
Headache	Postictal, migraine-like	Core to phenotype	Absent	Absent	Absent
Key discriminator	Speed + stereotypy + clustering	Slow centrifugal march	**Monocularity**	Occurs *within* a field defect	Reproduces prior deficit

Some practical consequences.

The history that discriminates is a history of speed of development, shape, colour, direction of movement, duration, frequency and progression. Asked “did you see flashing lights?”, a patient will say yes to almost everything in the table. Asked “how long did it take from the first thing you noticed to the worst it got — seconds, or minutes?”, the answer carries real diagnostic content.

Monocularity is the highest-yield single question in a stroke clinic, because it separates ocular and retinal causes from cortical ones. It is also the question patients answer least reliably, since a homonymous hemianopic defect is very commonly reported as a problem with one eye. Covering each eye in turn during an episode is the definitive test, and patients should be taught to do it explicitly, in those words, before they leave the ward.

Release hallucinations are the specific trap of this population. A patient with a dense hemianopia describing formed images in the blind field is far more likely to have Charles Bonnet phenomena than occipital epilepsy. The discriminators are the elaboration of the imagery, the preserved insight and the absence of stereotypy and clustering. Getting this wrong commits the patient to antiseizure medication, a driving prohibition and a great deal of unnecessary fear.

And new-onset migraine with aura after 60 should be treated with scepticism. Not impossible; but in someone with a recent occipital infarct it is a diagnosis of exclusion, and the exclusion needs an EEG with adequate posterior sampling.

6. Investigation

EEG. The yield of routine interictal EEG in occipital epilepsy is modest. In Panayiotopoulos’s series a substantial proportion of patients with unequivocal visual seizures and structural lesions had normal or non-specific recordings.[12] Two things follow. A normal EEG does not exclude the diagnosis and must never be used to overturn a convincing semiological history, a point that has to be made repeatedly to non-specialist colleagues and is worth putting in the clinic letter. And standard 10–20 montages sample the posterior head sparsely; where occipital onset is suspected, additional inferior posterior temporal and occipital electrodes, a sleep recording, and prolonged or ambulatory monitoring all improve yield materially. Early continuous EEG after stroke adds independent prognostic information, and instruments incorporating it exist, though availability is the limiting factor almost everywhere.[7]

6. Investigation

MRI, including susceptibility-weighted sequences. Not to diagnose epilepsy, which is clinical, but to find cortical superficial siderosis and haemosiderin, which carry independent prognostic weight.[6]

Perimetry and OCT. These are not conventionally epilepsy investigations. They should be. Formal perimetry establishes the baseline field against which any new or fluctuating deficit can be judged, which is otherwise guesswork. Macular OCT gives an objective, effort-independent structural correlate of the retrogeniculate lesion through retrograde trans-synaptic degeneration, and separately provides a baseline against which later medication-related retinal change can be assessed.[13,14] In a patient who may be on medication for decades and whose vision is already compromised, that baseline costs almost nothing and cannot be recovered later.

Postictal states. Todd's visual deficits after occipital seizures can be clinically indistinguishable from recurrent stroke, and this recurs as an emergency department dilemma. Perfusion imaging showing ictal or postictal hyperperfusion in a non-vascular distribution helps where available. The absence of a new diffusion lesion is the more widely available discriminator.

7. Management

Primary prophylaxis is not indicated. There is no evidence that antiseizure medication started after stroke in the absence of seizures prevents epilepsy, and reasonable evidence of harm through adverse effects, interactions and possible interference with recovery. Antiepileptogenesis trials in high-risk stroke populations are running; until they report, prophylaxis sits outside standard care.

For acute symptomatic seizures, short-term treatment is reasonable. The decision to continue beyond the acute period should be made explicitly and revisited, not inherited. This is a common failure: medication begun on the stroke unit is often still being swallowed three years later because nobody ever decided it should be. The exception is acute symptomatic status epilepticus, where the ten-year epilepsy risk in survivors approaches or exceeds 80% and long-term treatment is defensible on that basis alone.[5]

A single late seizure is epilepsy in this population and warrants treatment.[2]

On drug selection, the relevant considerations in an older, polypharmacy-exposed, post-stroke patient are enzyme induction and its consequences for anticoagulants, antiplatelets and statins; sodium homeostasis; bone health; cognition and mood; and renal and hepatic clearance. Agents with low interaction potential and reasonable tolerability in older adults are generally preferred, and enzyme inducers are generally avoided where secondary prevention depends on drugs subject to induction.

Two agents need specific comment in this context. Vigabatrin should be avoided in anyone with a pre-existing hemianopia. It causes permanent concentric constriction, and superimposing that on a hemianopic defect risks a composite deficit that is functionally catastrophic.[15,16] Topiramate carries a rare but real risk of acute angle-closure glaucoma with secondary myopia, which matters more than usual when visual reserve is already reduced.

Visual rehabilitation should not wait for seizure control. Visual field loss follows roughly a third of strokes, most often as complete or partial homonymous hemianopia,[17,18] and spontaneous recovery is largely confined to the first three to six months. Our companion review argues that retinal ganglion cell loss narrows that window further still.

8. The Driving Problem

This is the part of the review we would most like read by someone in a position to change a clinic letter template.

A patient who has had an occipital stroke and then develops epilepsy faces two independent statutory barriers to driving.

The visual barrier is a field standard. In the United Kingdom, Group 1 licensing requires a binocular horizontal field of at least 120 degrees with at least 50 degrees either side of centre, and no significant defect within 20 degrees of fixation above or below the horizontal meridian; assessment is by binocular Esterman perimetry with reliability requirements attached.[19] Homonymous and quadrantic defects approaching fixation are not normally compatible with a licence. Comparable standards operate in most jurisdictions, and the underlying concern has empirical support: drivers with hemianopia or quadrantanopia have been reported to have substantially higher at-fault collision rates per mile driven than drivers with full fields.[20]

The seizure barrier is temporal. Group 1 licensing after epilepsy generally requires a defined seizure-free interval — twelve months in the United Kingdom, with a shorter interval potentially applicable after a single unprovoked seizure in some circumstances — and considerably more for Group 2 entitlement.[21]

These two assessments are performed by different clinicians, against different standards, on different timescales, and reach the licensing authority by different routes. Neither is required to account for the other, and the failures run in both directions.

A patient may complete the seizure-free interval, be told by the neurologist that they may resume driving, and carry a field defect that was never quantified because nobody arranged perimetry.

Or a patient may fail the field standard, surrender the licence, and consequently never be assessed for the seizure requirement at all — so the epilepsy is never flagged as driving-relevant, and reappears unaddressed if the field later becomes acceptable, whether through partial recovery or a successful application under exceptional-case provisions.[22]

There is a third interaction that is essentially uncharacterised, and it is the one that worries us. If focal aware occipital seizures produce brief ictal visual disturbance — coloured phenomena, or transient ictal amaurosis — in a driver who has only one functioning hemifield to begin with, then an event that would be a curiosity in someone with full fields may amount to complete transient loss of usable vision at the wheel. We are not aware of any evidence addressing this. We are not aware of any licensing framework that considers the two conditions jointly rather than in sequence.

Three things follow, and none of them waits on new evidence.

1. Every patient with an occipital or posterior circulation stroke should have formal perimetry documented before anybody gives any advice about driving.
2. Advice after post-stroke epilepsy should state explicitly that completing the seizure-free interval does not establish fitness to drive where a field defect exists, and that passing the field standard does not establish it where seizures have occurred.
3. Correspondence between ophthalmology, neurology and stroke services should carry both assessments in the same letter. At present it carries one.

9. POSE-8

Table 3 sets out an eight-item assessment set for the survivor of occipital or posterior circulation stroke. Its only virtue is being short enough to use.

#	Item	Timing	Purpose
1	Territory, cortical involvement, haemorrhagic transformation, cortical superficial siderosis	Admission	Anchors epileptogenic risk; siderosis independently predictive
2	Any acute symptomatic seizure, with type stated (brief vs status)	First 7 days	Strongest single predictor; type materially alters risk
3	Formal perimetry with reliability indices	Before discharge or within 6 weeks	Baseline field; prerequisite for all driving advice

Table 3 continued...

4	Macular OCT with GCL segmentation	Within 6 weeks, repeated at 6–12 months	Structural correlate; baseline for later drug monitoring
5	Risk score recorded with territorial caveat	Before discharge	SeLECT or SeLECT 2.0, with a note that the MCA item cannot score
6	Written semiology counselling for patient and family	Before discharge	What an occipital seizure looks like; instruction to cover each eye during an episode
7	Joint driving statement	Each relevant review	One document recording both field status and seizure interval
8	Named 12-month review	Discharge	The interval when late seizures cluster and most services have already discharged the patient

10. Predictions, and a Disagreement

Prediction 1 — Territorial miscalibration. In a cohort of ischaemic stroke survivors with adequate posterior circulation representation, SeLECT and SeLECT 2.0 will show systematic miscalibration in that subgroup, with observed late-seizure incidence exceeding predicted incidence at low score values. *Refuted by:* calibration slope and intercept in the posterior circulation subgroup not differing meaningfully from the anterior circulation subgroup. This should be tested by reanalysis of existing cohorts with recorded territory, not by new recruitment; usable precision requires a posterior circulation subgroup of perhaps 400–500 patients with adequate follow-up, which will only be reached by pooling. Of everything in this review, this is the question we would most like answered.

Prediction 2 — Diagnostic latency. Time from first unprovoked seizure to diagnosis of epilepsy will be significantly longer in occipital-onset than in non-occipital post-stroke epilepsy, with a substantial proportion of occipital cases receiving an intervening alternative diagnosis, most often migraine with aura. *Refuted by:* no difference between groups. Testable retrospectively from existing records, and by some distance the cheapest of the four.

Prediction 3 — Written counselling shortens the delay. Providing a written structured description of occipital seizure semiology at discharge, with instruction in monocular occlusion testing, will reduce time to diagnosis compared with standard discharge information. *Refuted by:* no difference in time to diagnosis. Cluster randomisation at unit level avoids contamination; roughly 12–16 units with combined annual occipital stroke volume sufficient to accrue 300 participants.

Prediction 4 — Compound visual risk. Among drivers with hemianopia who develop occipital post-stroke epilepsy, the functional visual impact of focal aware seizures, measured as duration of usable-field loss during an event, will exceed that in seizure-matched patients with intact fields. *Refuted by:* no difference. This is the hardest to operationalise ethically and would need simulator rather than on-road assessment. We state it because it has the clearest public-safety implication and is presently unaddressed by anyone.

Where we disagree. VK considers Prediction 1 close to certain, on the grounds that occipital cortex is cortex and the territory item is a proxy for lesion size and cortical burden that happens to have been fitted in an MCA-dominant sample; on that reading, miscalibration in the posterior circulation is almost arithmetically guaranteed. AD is less confident, and thinks the territory item may be capturing something real about MCA cortex — extent of association cortex involved, or the particular vulnerability of perisylvian and temporal structures — in which case posterior circulation strokes might be correctly assigned lower risk and the score would calibrate acceptably. We could not reconcile these positions and have not tried to. The disagreement is itself an argument for doing the reanalysis.

11. Limitations

Narrative review, with the usual consequences: no systematic search, no pre-specified eligibility criteria, no formal risk-of-bias appraisal, and a selection of evidence that reflects our reading.

Our central claim about SeLECT is a structural observation about how the score was derived and how it behaves at the boundary of its derivation population. It is not a demonstration that the score performs poorly in posterior circulation stroke, and we have tried hard not to let it read as one. The direction of any bias is genuinely unknown, which is the point of the disagreement in Section 10.

Table 2 is synthesised largely from work in populations that were not principally post-stroke. Its discriminative performance in older stroke survivors with pre-existing field defects has not been formally evaluated, and we would expect it to be worse in that group than in the series it derives from.

The driving standards in Section 8 are those of the United Kingdom and are used illustratively. Thresholds, testing methods and reporting obligations differ substantially between jurisdictions, and readers must apply their own.

POSE-8 has not been piloted. Item 4 assumes access to OCT that many stroke services do not have.

Our sample size figures are indicative, not completed power calculations.

And the authorship spans stroke medicine, ophthalmology, general medicine and epileptology, which is deliberate and also guarantees that a specialist in any one of them will find our handling of theirs thinner than they would like.

12. Conclusion

The occipital lobe sits at an awkward junction. Its infarcts are common. Its seizures are expressed in a modality shared with three or four conditions that are commoner still. The best-validated tool for predicting its epileptogenicity contains an item it cannot score. And its survivors face two statutory barriers to driving that no single clinician is required to assess together.

None of this reflects incompetence. The stroke physician, the ophthalmologist and the epileptologist are each doing their part properly. The failures are at the joins, and joins are nobody’s specialty.

We have proposed three things needing no new evidence: perimetry before driving advice, risk scores recorded with their territorial limitation stated, and a written description of occipital seizure semiology handed to the patient at discharge. One thing needing pooled reanalysis rather than recruitment: the calibration of SeLECT in posterior circulation stroke. And four ways of showing us wrong, one of which we cannot agree about among ourselves.

The patient in Section 1 lost a year. Nobody made an error of knowledge. The coloured circles she described belonged simultaneously to three specialties and therefore to none.

Declarations

Author Contributions

CRediT taxonomy. **AD:** Conceptualisation; methodology; writing — original draft; writing — review and editing; supervision; project administration. **SK:** Conceptualisation; investigation (visual field, perimetric and driving-standard content); writing — original draft (Sections 5 and 8); writing — review and editing. **GG:** Investigation; data curation (literature identification and screening); writing — review and editing; visualisation (tables). **VK:** Conceptualisation; investigation (epileptological content, risk scores, pharmacotherapy); writing — original draft (Sections 2, 3 and 7); writing — review and editing. All authors read and approved the final version and agree to be accountable for all aspects of the work. The disagreement recorded in Section 10 is stated with the consent of both parties.

Ethics Statement

This is a review article. It does not report primary research involving human participants, human material, human data or animals. No ethical approval and no patient consent were required. The clinical vignette in Section 1 is a composite constructed for illustration and does not describe any identifiable individual. The studies proposed in Section 10 would each require prospective ethical approval in the jurisdiction where undertaken.

Consent for Publication

Not applicable.

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Conflict of Interest Statement

No conflict of interest to declare.

Data Availability Statement

No new primary datasets were generated or analysed. All evidence discussed is available in the cited literature. POSE-8 (Table 3) and the semiological framework (Table 2) are released without restriction to support independent adoption and evaluation.

Use of Artificial Intelligence

There was NO use of AI in preparing any part of this article.

Abbreviations

ASM, antiseizure medication; ATP, adenosine triphosphate; CAVE, cortical involvement–age–volume–early seizure score; DVLA, Driver and Vehicle Licensing Agency; EEG, electroencephalography; GCL, ganglion cell layer; HMGB1, high-mobility group box 1; ICH, intracerebral haemorrhage; IL-1β, interleukin-1 beta; ILAE, International League Against Epilepsy; MCA, middle cerebral artery; MRI, magnetic resonance imaging; NIHSS, National Institutes of Health Stroke Scale; OCT, optical coherence tomography; PCA, posterior cerebral artery; POSE-8, Posterior circulation and Occipital Stroke Epilepsy assessment set; PSE, post-stroke epilepsy; SeLECT, severity–large-artery atherosclerosis–early seizure–cortical involvement–territory score; TGF-β, transforming growth factor beta.

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