Epilepsy syndrome of adolescent and adulthood

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Outline

01 Encephlopathic Epilepsy

02 Idiopathic Generalized Epilepsy

03 Idiopathic Focal Epilepsy

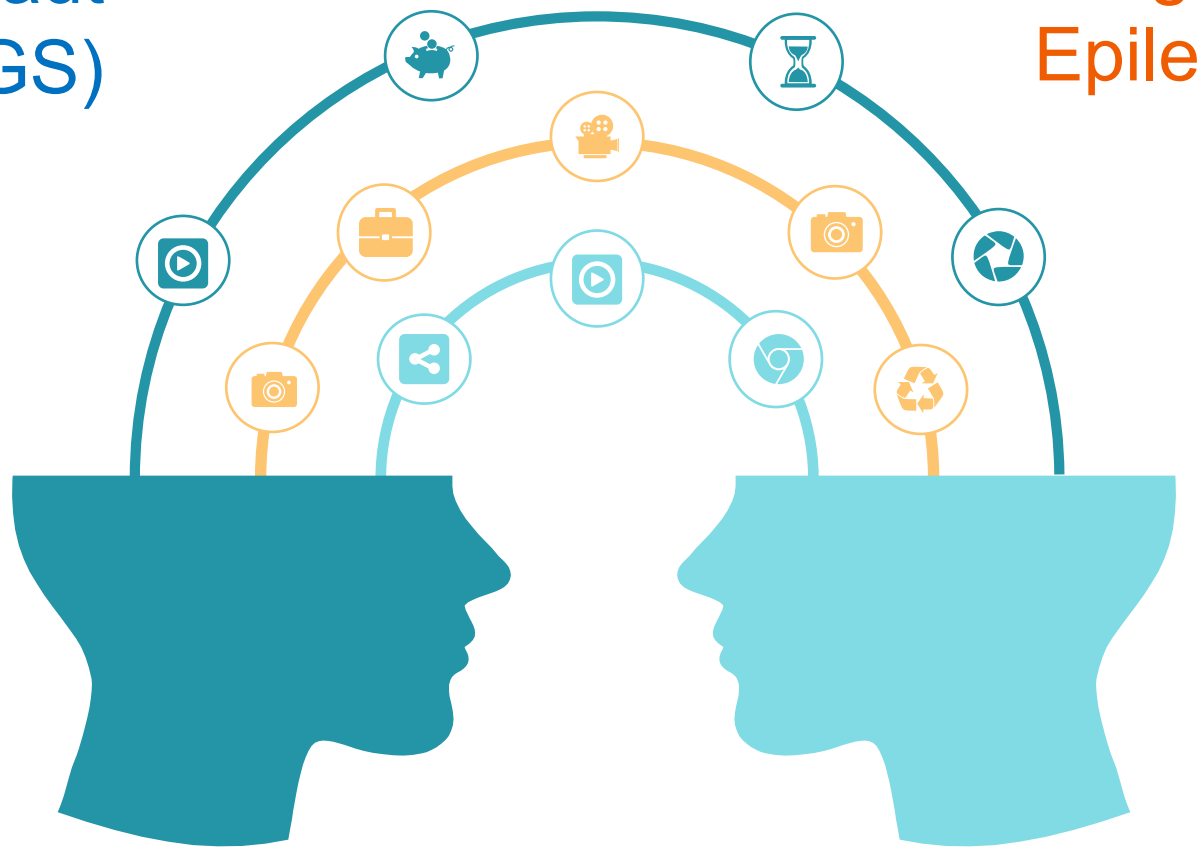
04 Symptomatic Focal Epilepsy

05 **Epilepsy syndrome in special condition**

Encephlopathic Epilepsy

Lennox-Gastaut Syndrome (LGS)

Progressive Myoclonic Epilepsy (PME)



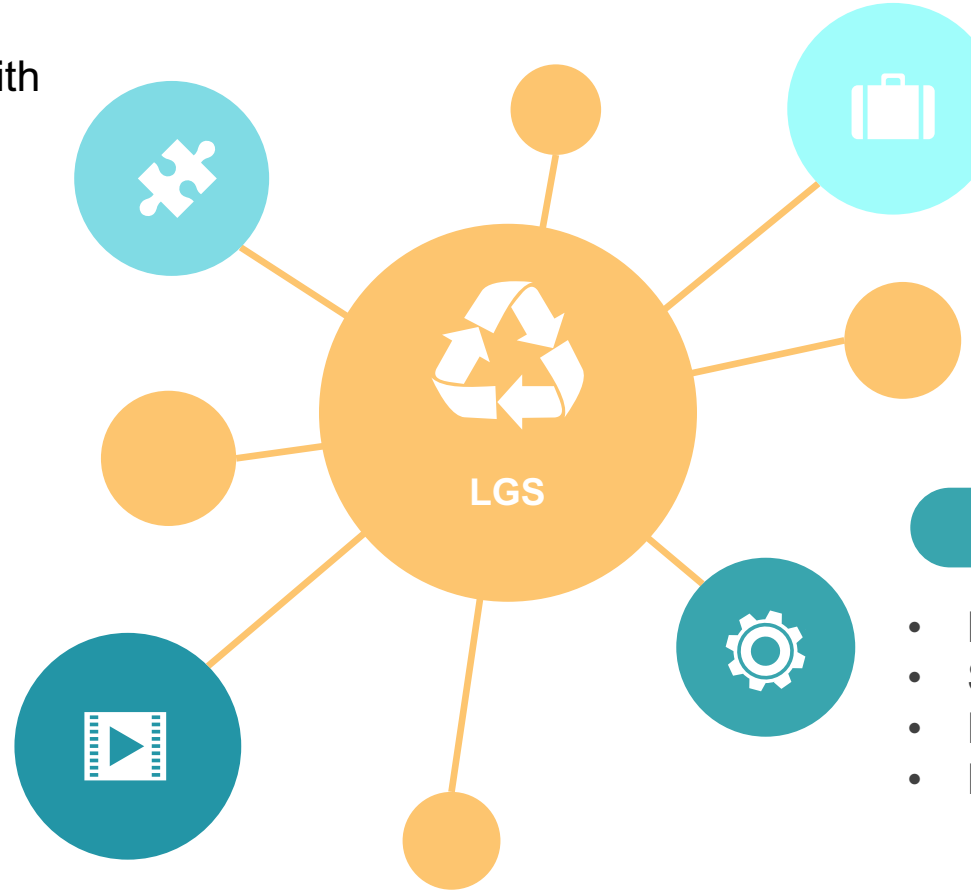
Lennox Gastout Syndrome (LGS)

Cause

- Lack of oxygen during birth
- Severe brain injuries linked with pregnancy or birth
- Infections(encephalitis, meningitis, or rubella)
- Cortical malformation
- Tuberous sclerosis
- Genetics

Diagnosis

- Multiple seizure types
- EEG : slow spike-wave pattern between seizures
- Cognitive impairment Developmental delays



Symptoms

- Atonic seizures "drop attacks
- Tonic seizures
- Absence seizure
- Focal seizure
- Tonic status

Treatment

- Medication
- Surgery
- Diet
- Neurostimulation

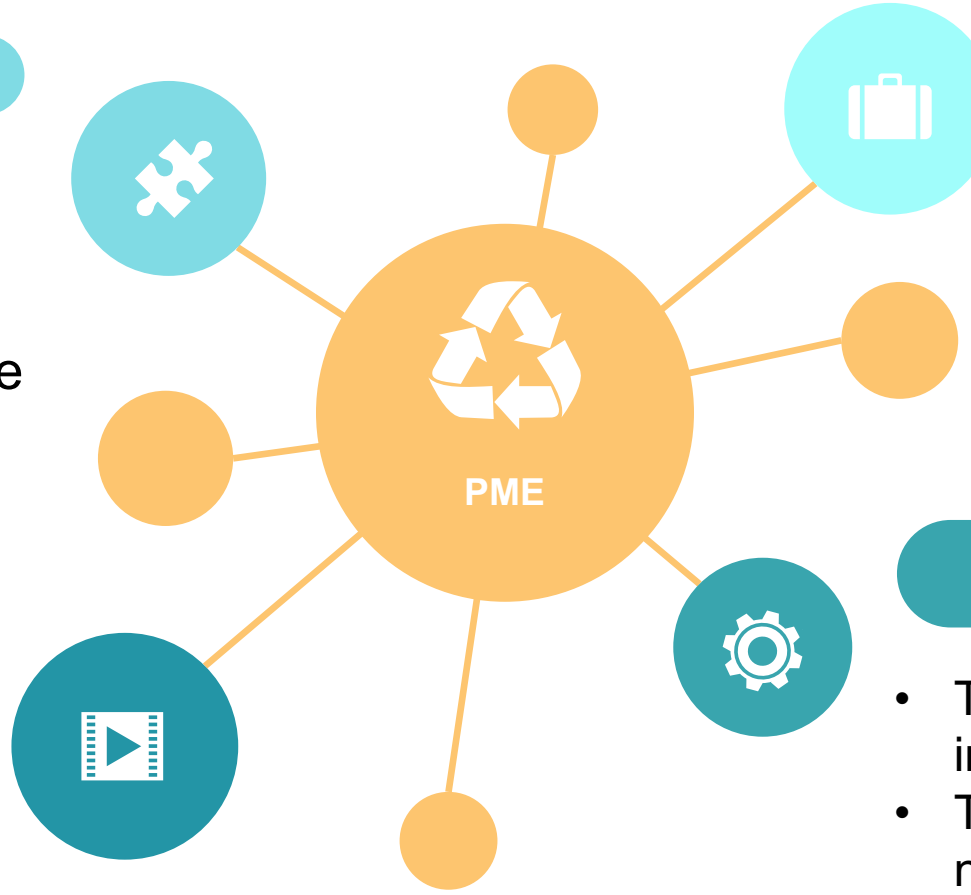
Progressive Myoclonic Epilepsy (PME)

PME

Disorders that are genetic cause and “progressive.” decline in motor skills, balance and cognitive function and seizure

Cause

Most forms are inherited in AR pattern affect both sexes equally



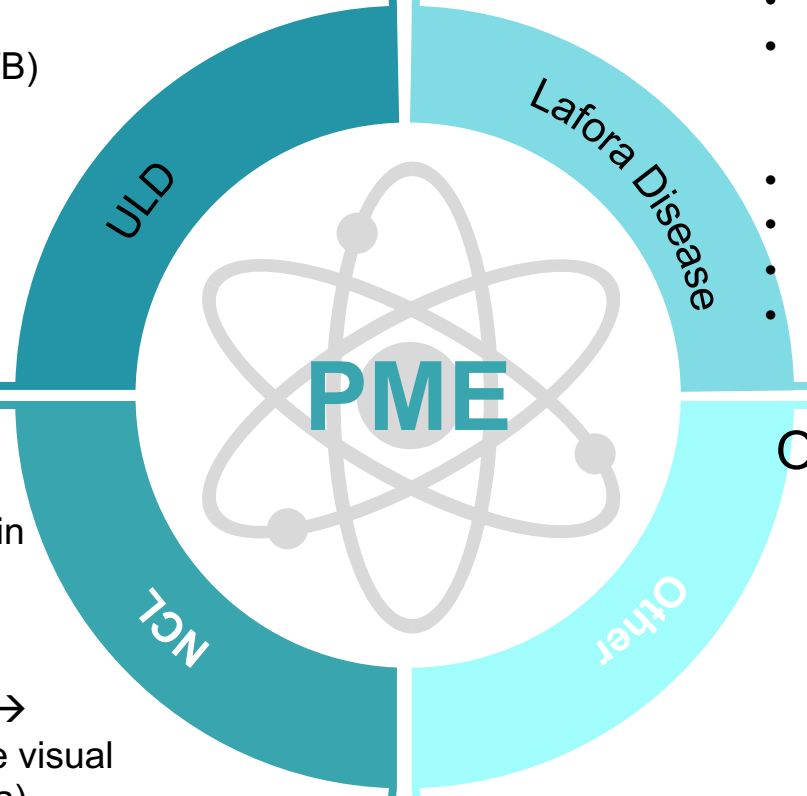
Symptoms

- Mix of myoclonic and tonic-clonic seizures
- Unsteadiness
- Muscle rigidity
- Balance problems
- Mental decline

Course

- The age of onset : vary from infancy to adulthood
- The most common forms are first noted in early adolescence to late childhood

TYPES AND SYMPTOMS



Unverricht-Lundborg Disease

- The most common form of PME
- Mutation in the cystatin B gene (CSTB)
- Myoclonic jerks : age 6–16 (Arms & legs)
- triggered due to a variety of common external stimuli
- Seizures begin at an average age of 10.8 years

Lafora Disease

- AR disorder,
- Mutations in laforin glycogen phosphatase gene (EPM2A) or malin E3 ubiquitin ligase gene(NHLRC1)
- "Lafora bodies" within the cytoplasm
- More specifically in adolescents (12-15 years)
- Seizures start→ rapid worsening of cognition
- (thinking), visual loss, and coordination

Neuronal Ceroid Lipofuscinoses

- Abnormal accumulation of ceroid and lipofuscin
- within neurons
- Adult subtype (Kufs disease) : AR
- Begin at age around 30
- The first symptoms are myoclonus and GTCs→ cognitive and emotional decline, motor decline visual loss leading to blindness (damage to the retina)

Other Less Common Forms Of PME

- Sialidosis
- MERRF
- Type 3 Neuronopathic Gaucher Disease
- Dentatorubral-Pallidoluysian Atrophy
- Myoclonus-renal Failure Syndrome
- Progressive Myoclonus Epilepsy-Ataxia Syndrome

DIAGNOSIS

Diagnosing the different types of PME can be difficult.

The early way to tell the difference is an EEG with background slowing

Symptoms : Myoclonic jerks, cognitive decline and motor slowing, GTC, or visual/occipital seizures

Genetic and enzyme testing
Skin biopsy

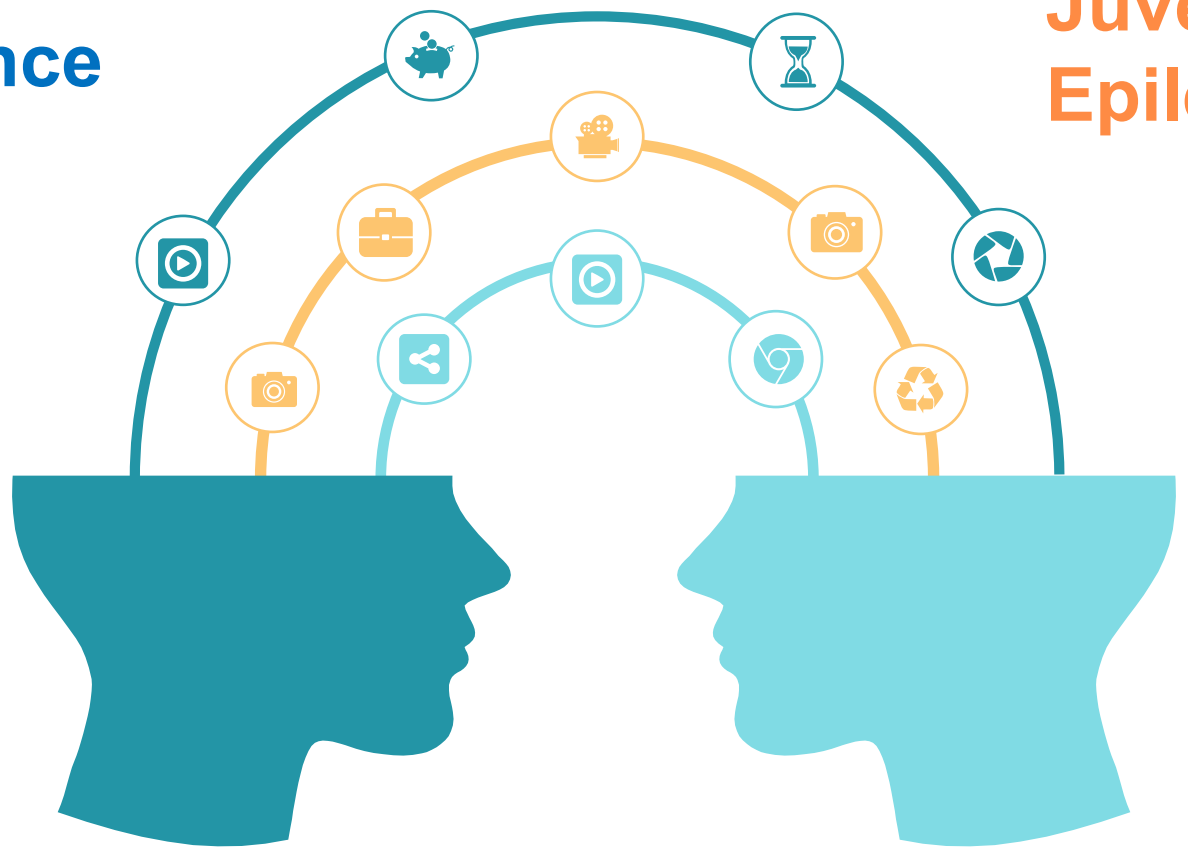
Treatment

No Current Cure : Supportive & symptomatic treatment

- Require many AEDs, comprehensive rehabilitation treatment, and treatment of mood symptoms. Social and psychological support
- Avoid Na Channel block AEDs and GABAergic
- In mitochondrial forms , VPA should be avoided.

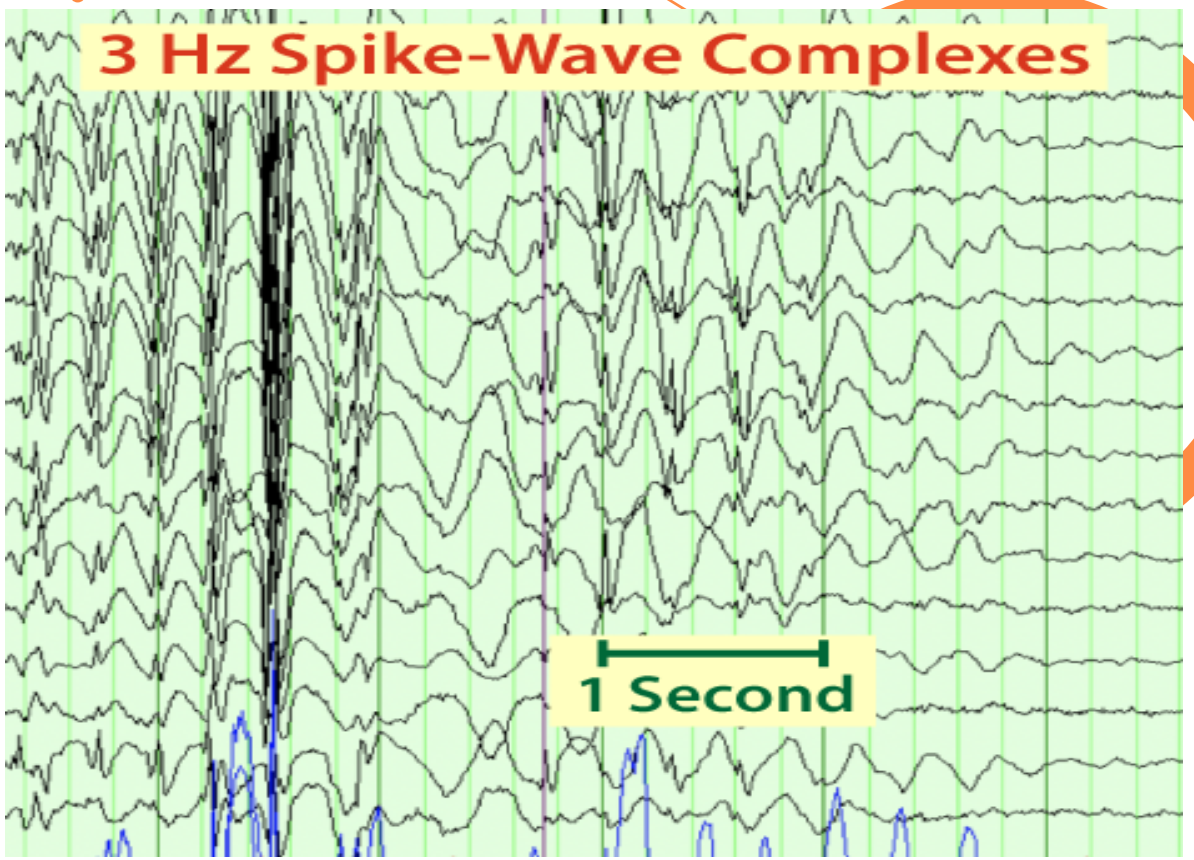
Idiopathic Generalized Epilepsy

Juvenile Absence Epilepsy (JAE)



Juvenile Myoclonic Epilepsy(JME)

JAE



Seizure

Typical absences, tonic-clonic,
less common myoclonic

Course

Can be life long
Good response to AED

EEG

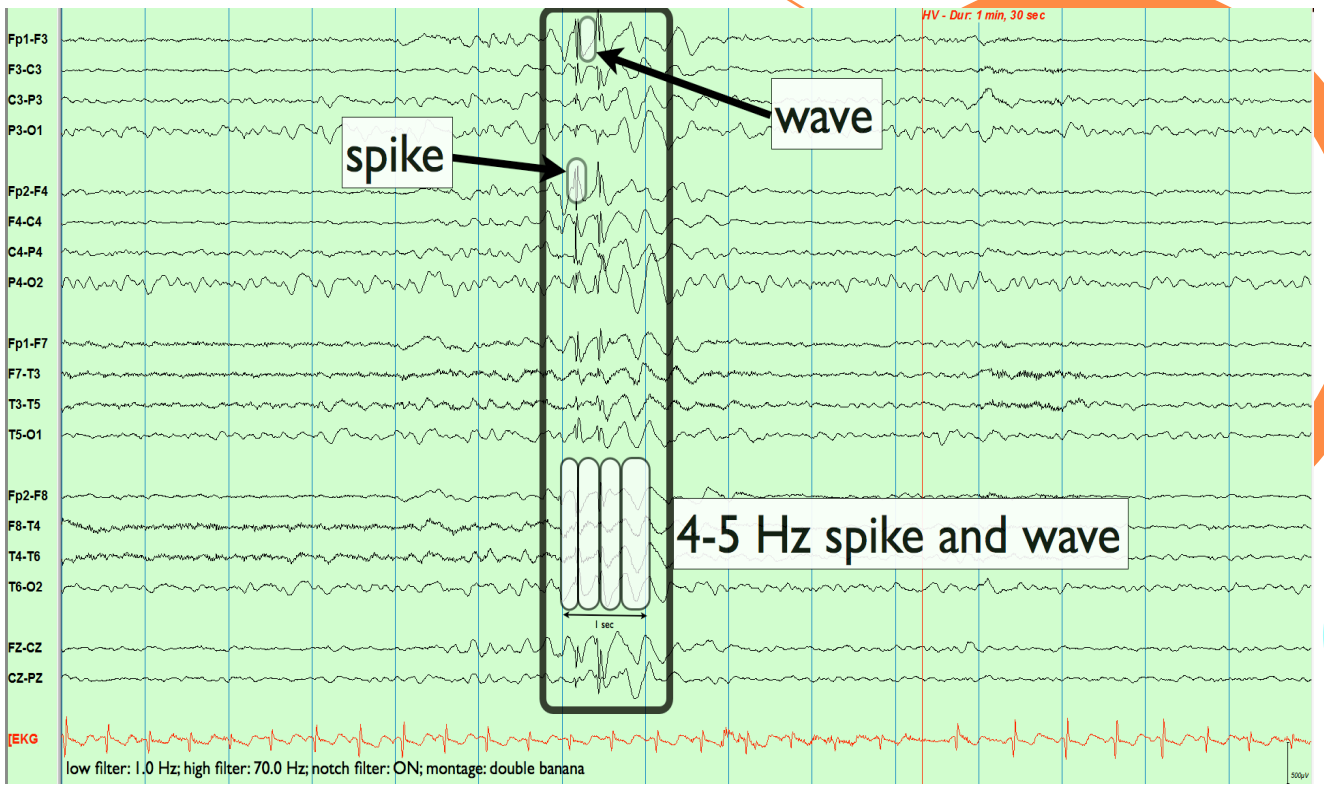
3-4 Hz Generalized spike&wave
Normal BG
PS triggered in few cases

Treatment

VPA : first line
LTG, clobazam: Alt

JME

EEG



Seizure

Myoclonus, tonic clonic, atypical absence, reflex myoclonus

Course

Life long

Well response to AED

EEG

4-6 Hz (poly)spike&wave time lock to myoclonic

PS triggered

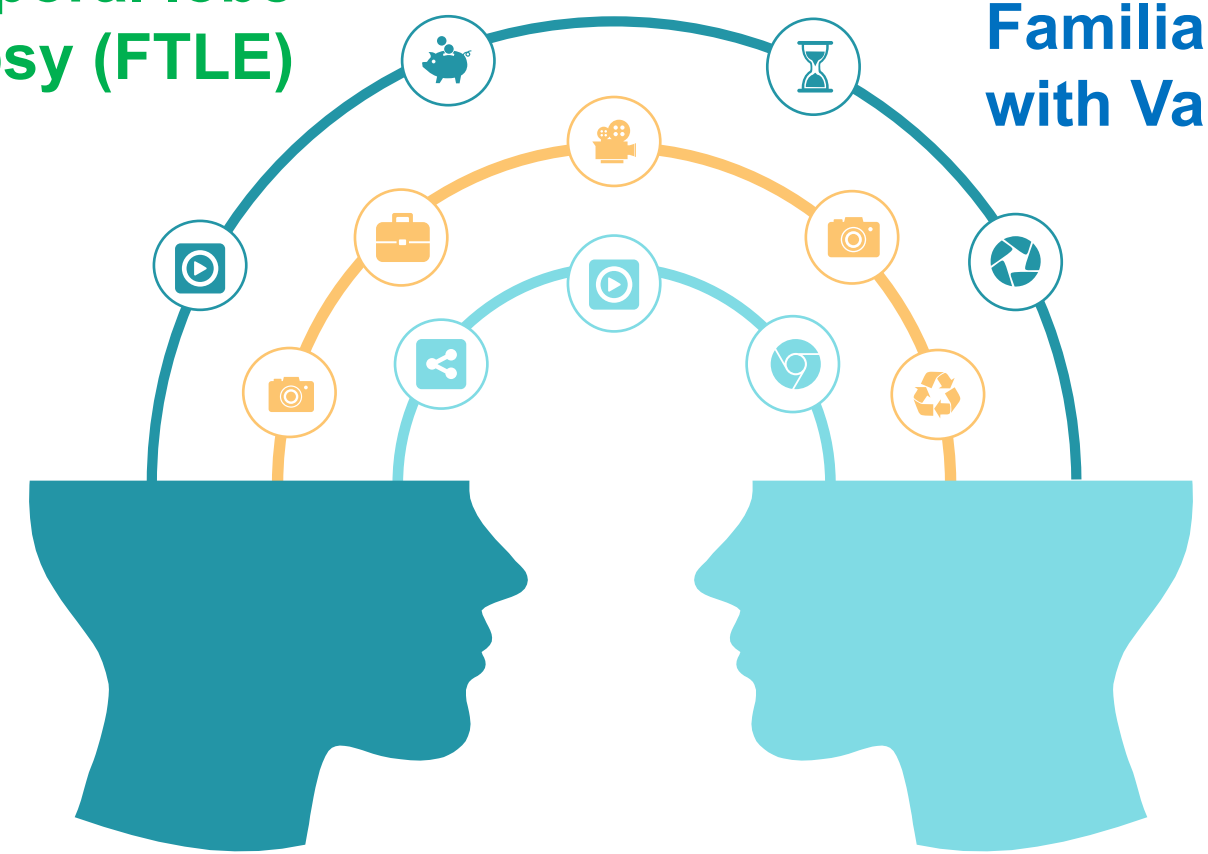
Treatment

VPA : first line

LVT /LMG : alternative

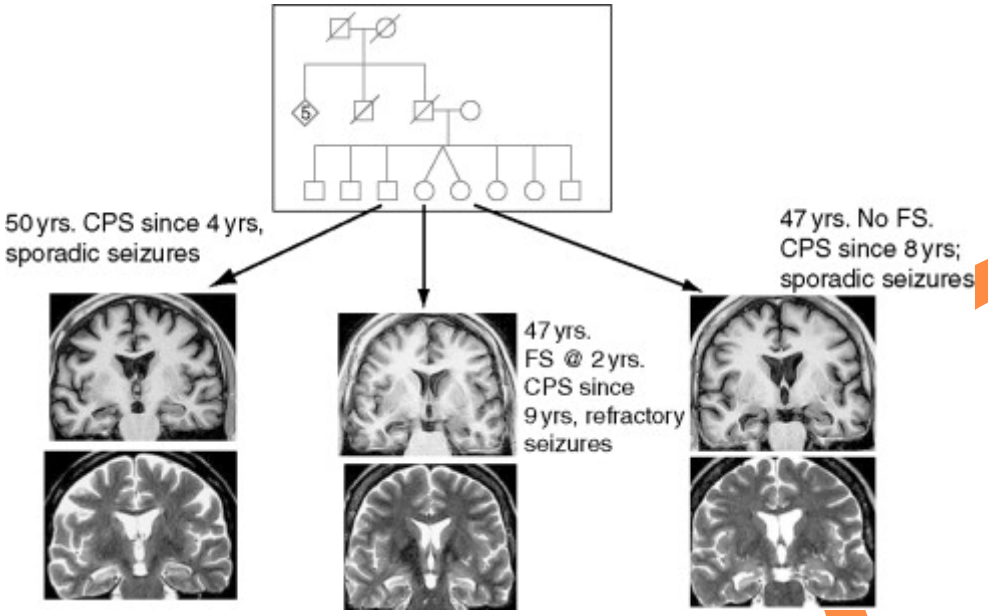
Idiopathic Focal Epilepsy

Familial Temporal lobe Epilepsy (FTLE)



Familial Focal Epilepsy with Variable Foci(FFEVF)

Familial Temporal lobe Epilepsy



FTLE

10q24 LGI1 : LFTLE

4q13 LGI1: MFTLE

Seizure

Clinical temporal lobe seizure

De ja vu aura / auditory aura

Course

Onset : Variable

Easily control with AED

EEG

IED : Anterior temporal/Posterior Temporal SW

Ictal : typical lateral or mesial temporal seizure

Treatment

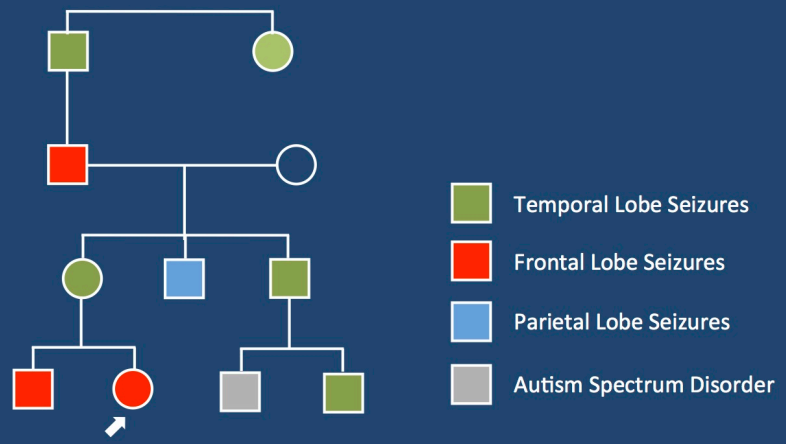
AED

Surgery if refractory

NICE guideline: Epilepsy :updated: 12 May 2021

Familial Focal Epilepsy with Variable Foci

DEPDC5 mutations and Familial Focal Epilepsy with Variable Foci (Dibbens et al., 2013)



FFEVE

22q12 DEPDC5 gene

Seizure

Frontal love/ Temporal lobe, rarely occipital or parietal

Course

Sz less frequent
Rare cluster and aura
Frequent daytime Sz and 2nd GTC

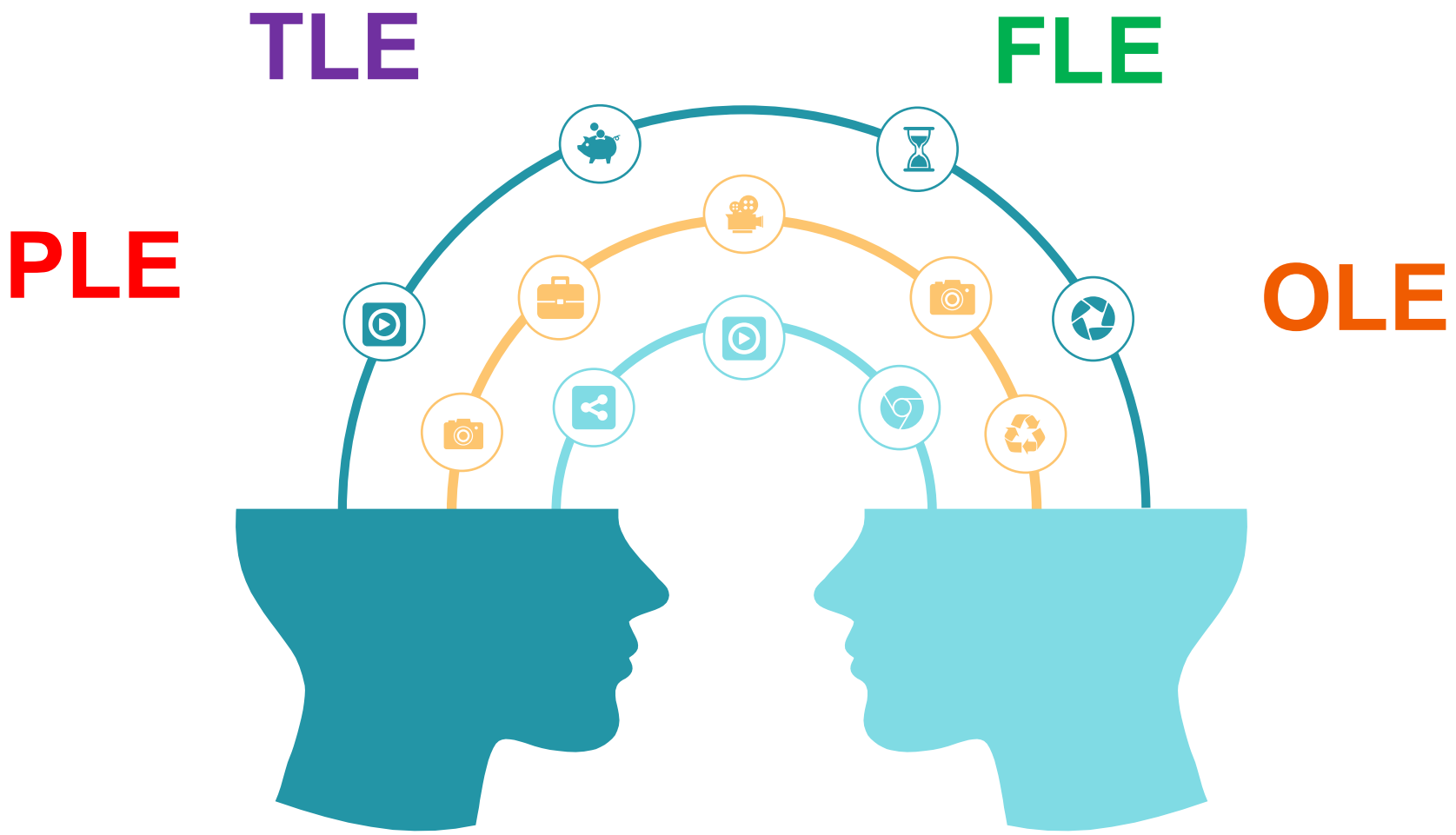
EEG

Variable depends on type of Sz

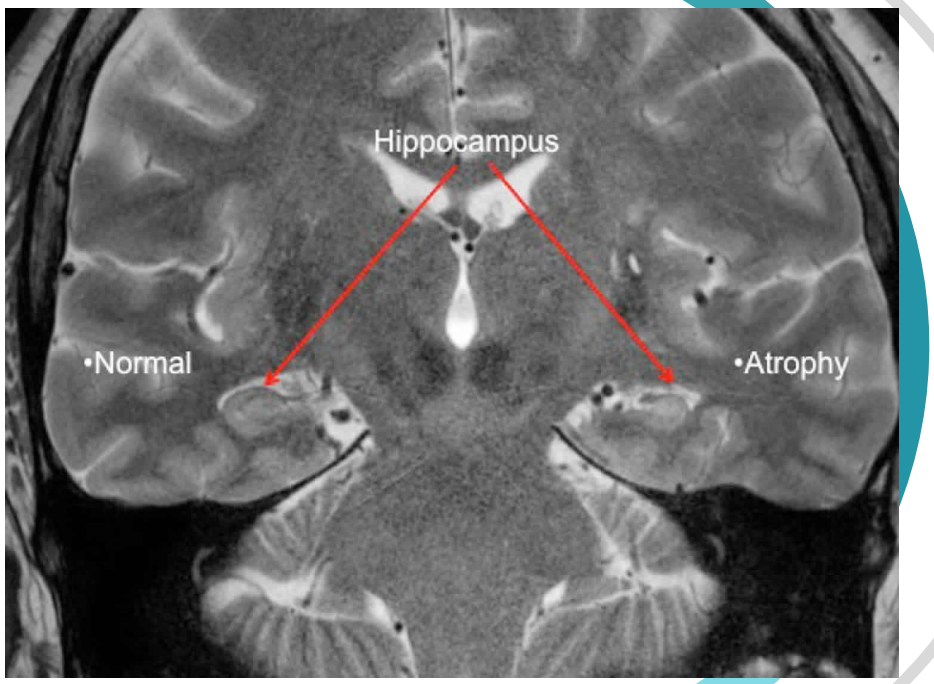
Treatment

Same asfocal epilepsy

Symptomatic Focal Epilepsy



Medial Temporal lobe Epilepsy



Clinical

Hx of FS
Seizures longer (typically > 2 min), with a slower evolution and more gradual onset/offset
Auras : visceral, cephalic, gustatory, affective, perceptual or autonomic auras
Motor arrest with loss of awareness
Post-ictal confusion and dysphasia common
Autonomic changes
Vocalisation also common

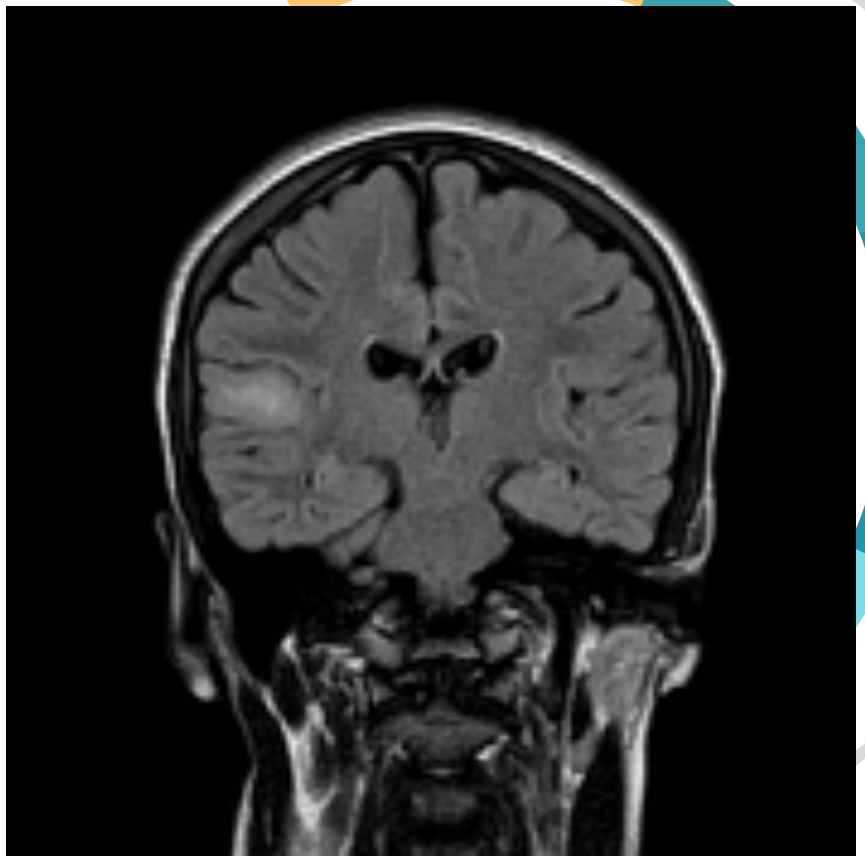
EEG

Anterior or mid-temporal spikes/sharp waves
Non-epileptiform regional slowing
Ictal : Rhythmic temporal alpha or theta activity within 30 seconds of onset (in ~80% of MTLE seizures)

Imaging

HS
Structural lesion

Lateral Temporal lobe Epilepsy



Clinical

Typically no history of FS
Auras : Hallucinations or illusions→ lateral rather than mesial temporal
The motionless stare and the automatisms are similar to medial temporal lobe

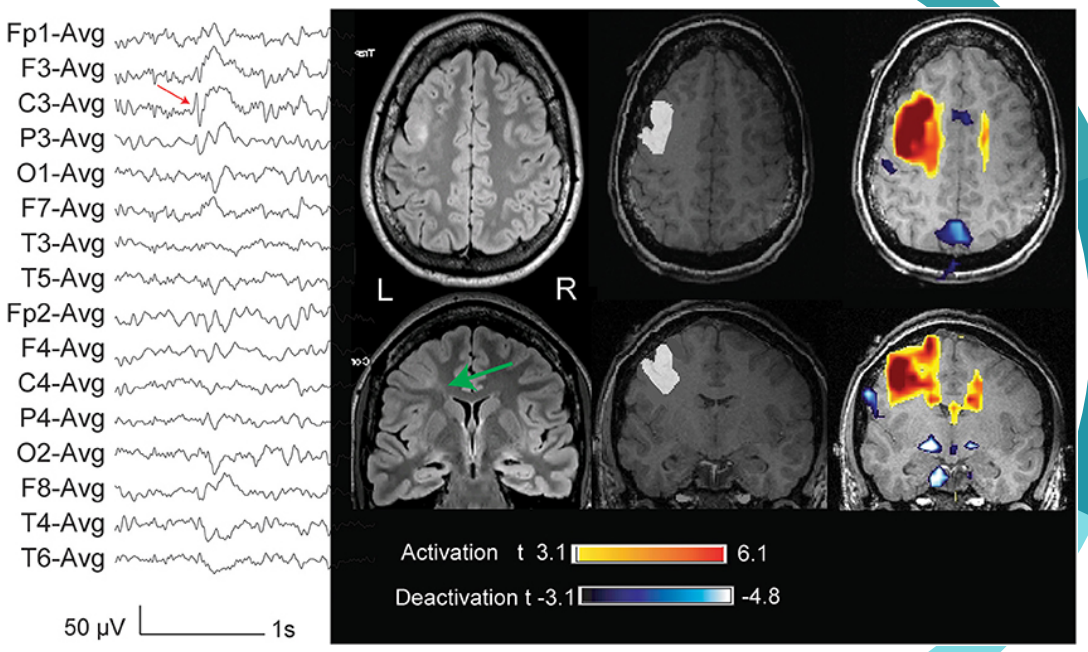
EEG

FT Spikes
Posterior SPK
Polyspikes : neocortical generators

Imaging

Structural changes , tumor, cavernous angioma

Frontal lobe Epilepsy



Clinical

<u>Classical</u> : hemiclonic Jacksonian motor seizures
Complex motor seizures : frontopolar, anterior cingulate, opercular-insular and orbitofrontal

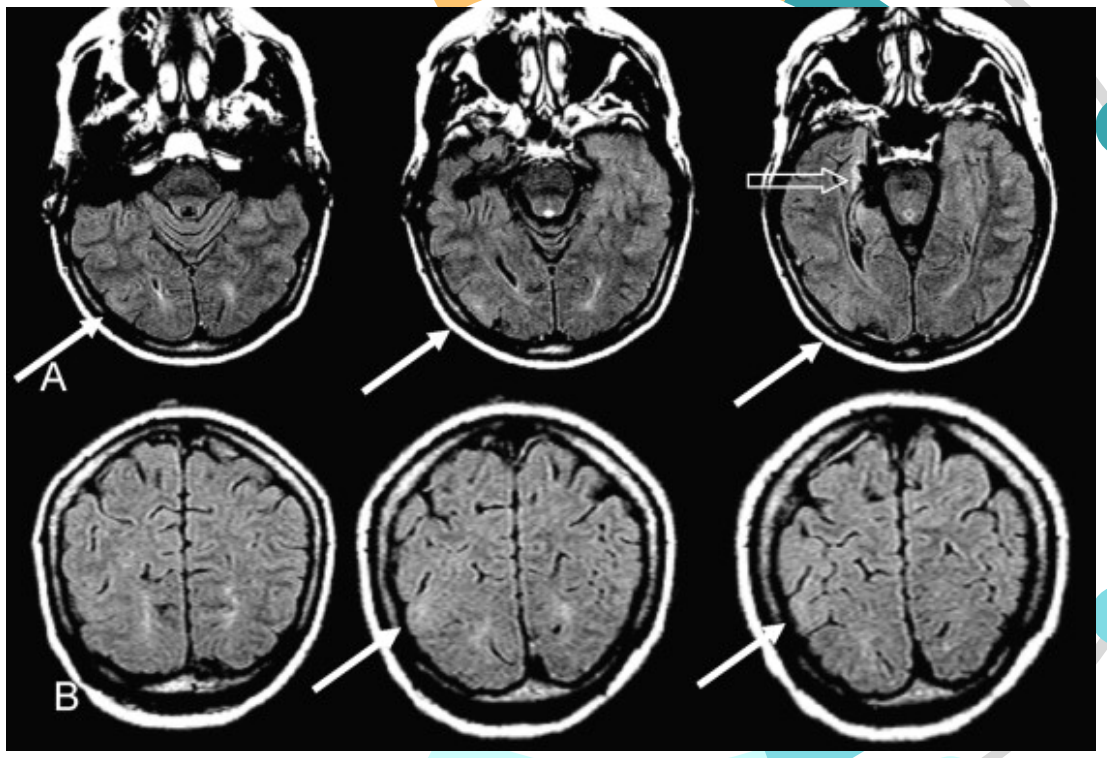
EEG

40% : No IED
Ictal EEG may be attenuated and undetectable

Imaging : localized value

CT : 20%
MRI :30-40%

Parieto-occipital lobe Epilepsy



Clinical

Somatosensory symptomatology
Visual symptomatology
Other seizure phenomena

EEG

Often electrically silent
Lateralising rather than localizing
Changes in the posterior background activity may be helpful in OLE

Imaging

MRI: tumors, trauma, malformations, ischemia, mitochondrial disease

Epilepsy syndrome in special condition

Stroke related epilepsy

Dementia related epilepsy

Autoimmune Epilepsy

Transient Epileptic Amnesia

Stroke related epilepsy

Contents Title

You can simply impress your audience and add a unique zing and appeal to your Presentations.

Stroke is the cause of about 10% of all epilepsy and 55% of newly diagnosed seizures

Early (within 7 days of onset of stroke) Late (beyond 7 days of onset of stroke)

Good prognosis
Being well controlled by AED
Up to 25% of cases become DRE

A. M. Feyissa.European Journal of Neurology 2019

Stroke related epilepsy

Table 1 Incidence rate of post-stroke seizures

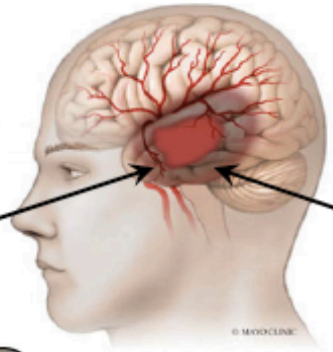
	Incidence rate (95% CI)
Subtype[a]	
Ischaemic[b]	0.06 (0.04–0.08)
TACI	0.14 (0.04–0.36)
PACI	0.05 (0.03–0.09)
POCI	0.03 (0.02–0.05)
LACI	0.02 (0.01–0.04)
Hemorrhagic[c]	0.10 (0.08–0.13)
ICH	0.09 (0.07–0.11)
SAH	0.11 (0.08–0.16)
Extent [86]	
Cortical	0.15 (0.10–0.21)
Subcortical	0.08 (0.04–0.13)
Lesion size [21]	
10 mm[d]	0.16
Time of onset [86]	
≤7 days (ESs)	0.04 (0.03–0.05)
>7 days (LSs)	0.05 (0.04–0.08)

Table 2 Risk factors for stroke-related epilepsy

Risk factor	Prevalence (%)	*P*-value
Age (years) [19]		
<65	10.7	<0.001
65–74	6.9	
75–84	3.9	
>85	1.6	
Gender [19]		
Male	6.6	0.74
Female	6.3	
Time of onset		
ESs [63]	33*	20.7–49.9*
LSs [5]	71.5*	59.7–81.9*
Stroke subtypes [19]		
TACI	9.3	<0.001
PACI	7.3	
POCI	3.1	
PICH	8.1	
SAH	11.1	
LACI	4.4	
Stroke severity (Barthel Index) (points) [88]		
0–4 (0–20)	40.5	<0.0001
5–9 (25–45)	14.3	
10–14 (50–70)	14.3	
15–10 (75–95)	11.9	
20 (100)	19.5	

A. M. Feyissa.European Journal of Neurology 2019

Stroke related epilepsy



- Hypoperfusion/reperfusion
  - Hypoxia
    - Glutamate-induced excitotoxicity
    - Reduced GABAergic inhibition
  - Ischemic encephalopathy
- Blood–brain barrier disruption
  - Activation of astrocytes
    - TGFβ, TNFα, IL-6 (neuroinflammation)
    - Protease activated receptor-1
  - K^+ inc
  - Glutamate-induced excitotoxicity
  - Thrombin-induced depolarization
- Ion channel dysfunction
  - Intracellular Na^+ and Ca^{++} inc
  - Extracellular K^+ inc

  } Depolarization
- Altered gene expression
  - Upregulation of inflammation and cell death
  - Alteration of neuronal plasticity
- Hemosiderin deposition

- Chronic inflammation
- Gliotic scarring
  - Leukoaraiosis
  - Angiogenesis
  - Neurogenesis
  - Selective neuronal loss
- Altered synaptic plasticity
- Deafferentation
- Collateral sprouting
- Selective neuronal loss

} Hyperexcitablity + neuronal synchrony

- Siderosis
- Comorbidities with stroke
  - Hyperglycemia (DM)
  - Dementia
  - Hypertension
  - Depression and use of antidepressants
  - Alcoholism

Early seizures

Late seizures

Stroke-related epilepsy

A. M. Feyissa.European Journal of Neurology 2019

Stroke related epilepsy

Table 3 Prediction models for stroke-related epilepsy (STRE)

Prediction model	Stroke type	Cohort size (*n*)	Follow-up (years)	Risk factors scored (values)	STRE predictions
SeLECT score (maximum score 9) [28]	Ischaemic	1200	5	Severity of stroke (0–2) Large-artery atherosclerosis (0–1) ESs (0–3) Cortical involvement (0–2) Territory of MCA involvement (0–1)	Total score ≥ 6 Sensitivity 18.2% Specificity 96.7% PPV 27.2% NPV 94.6%
Post-Stroke Epilepsy Risk Scale (PoSERS) (maximum score 8) [89]	Ischaemic and hemorrhagic	264	1	Supratentorial stroke (0–1) Cortical ICH (0–1) Cortical or subcortical ischaemic stroke (0–1) Ischaemia + ongoing neurological deficit (0–1) Stroke-related neurological deficit, mRS>3 (0–1) Vascular encephalopathy (0–1) ESs (0–1) LSs (0–1)	Total score ≥7 Sensitivity 70% Specificity 99.6% PPV 87.5% NPV 98.8%
CAVE score (maximum score 4) [90]	ICH	993	2.7	Cortical involvement (0–1) Age <65 years (0–1) Volume >10 mm (0–1) ESs (0–1)	Score ≥2 Sensitivity 81% Specificity 89% PPV 18% NPV 97%

Table 1 Seven items of the Post-Stroke Epilepsy Risk Scale

Item	Weight
Supratentorial stroke	2
ICH involving cortical areas	2
Ischaemia involving cortical or cortical-subcortical areas	1
Ischaemia + ongoing neurological deficit	1
Stroke caused neurological deficit with mRS > 3	
Seizure occurred up to 14 days after stroke	1
Seizure occurred 15 days or later after stroke	2

Table 2 CAVE score (for LS from ICH)

CAVE	Risk of LS
C: cortical involvement (1 point)	0 point: 0.6%
A: age <65 years (1 point)	1 point: 3.6%
V: volume >10 mL (1 point)	2 points: 9.8%
E: early seizure (1 point)	3 points: 34.8%
	4 points: 46.2%

Cut-off	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
>5	100	95.7	47.6	100
>6	70	98.4	63.6	98.8
>7	70	99.6	87.5	98.8
>8	20	100	100	97.0

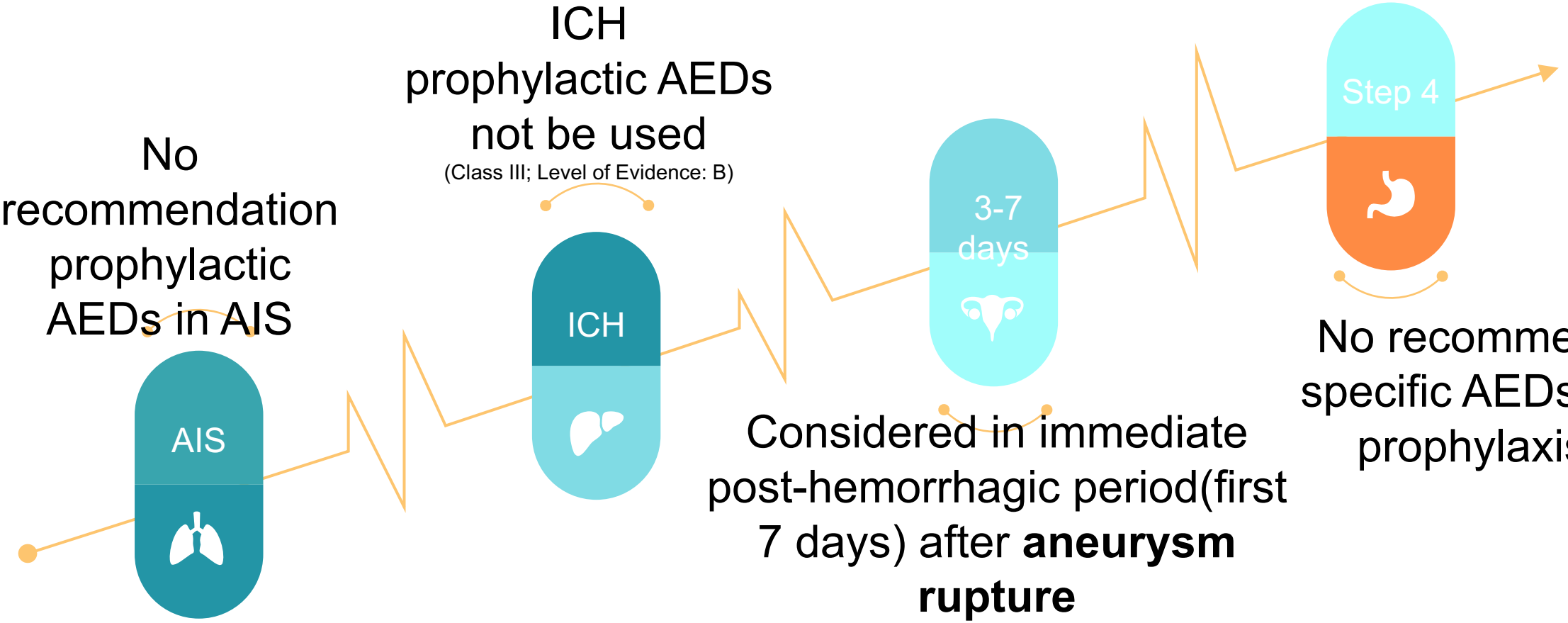
A. M. Feyissa. European Journal of Neurology 2019

Xu MY. Stroke and Vascular Neurology 2019

Role of AED prophylaxis in Stroke related epilepsy

No recommendation prophylactic AEDs in AIS

ICH prophylactic AEDs not be used
(Class III; Level of Evidence: B)



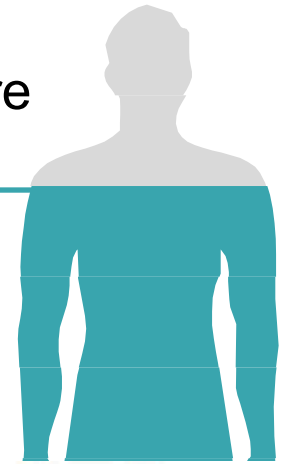
Considered in immediate post-hemorrhagic period(first 7 days) after **aneurysm rupture**
(Class IIb, Level of Evidence B)

No recommend specific AEDs for prophylaxis

The AHA / ASA guidelines,2015

Infographic Style

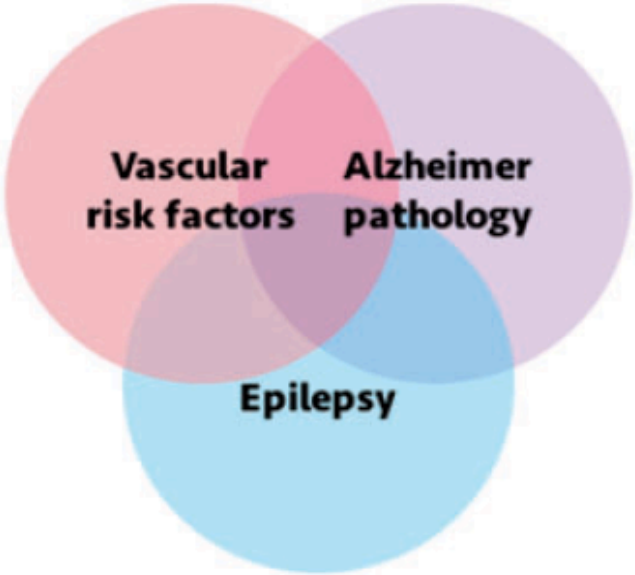
Accumulation of Aβ peptide in animal models of AD leading to recurrent seizure activity



The physiological response to seizures →hippocampal dysfunction →the memory impairment

Obesity
Dyslipidaemia
Hyperuricaemia
Insulin resistance
Carotid intima thickening
Small vessel disease

Stroke
Coronary artery disease
Heart failure
Diabetes
Hypertension



Amyloid plaques

Tau tangles

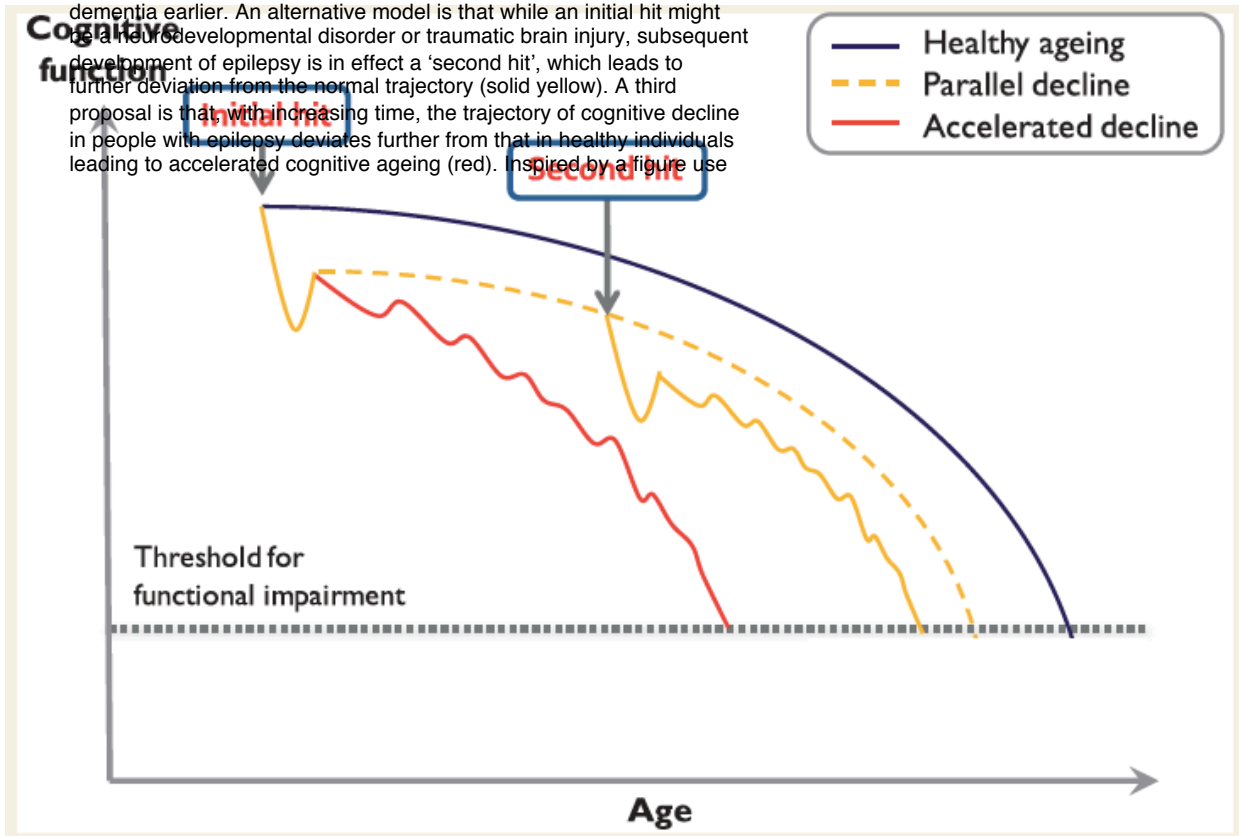
Vascular disease

Recurrent seizures → neuronal restructuring → abnormal neuronal activity → interfere with learning and memory→
Cognitive impairment

Arjune Sen.BRAIN 2018

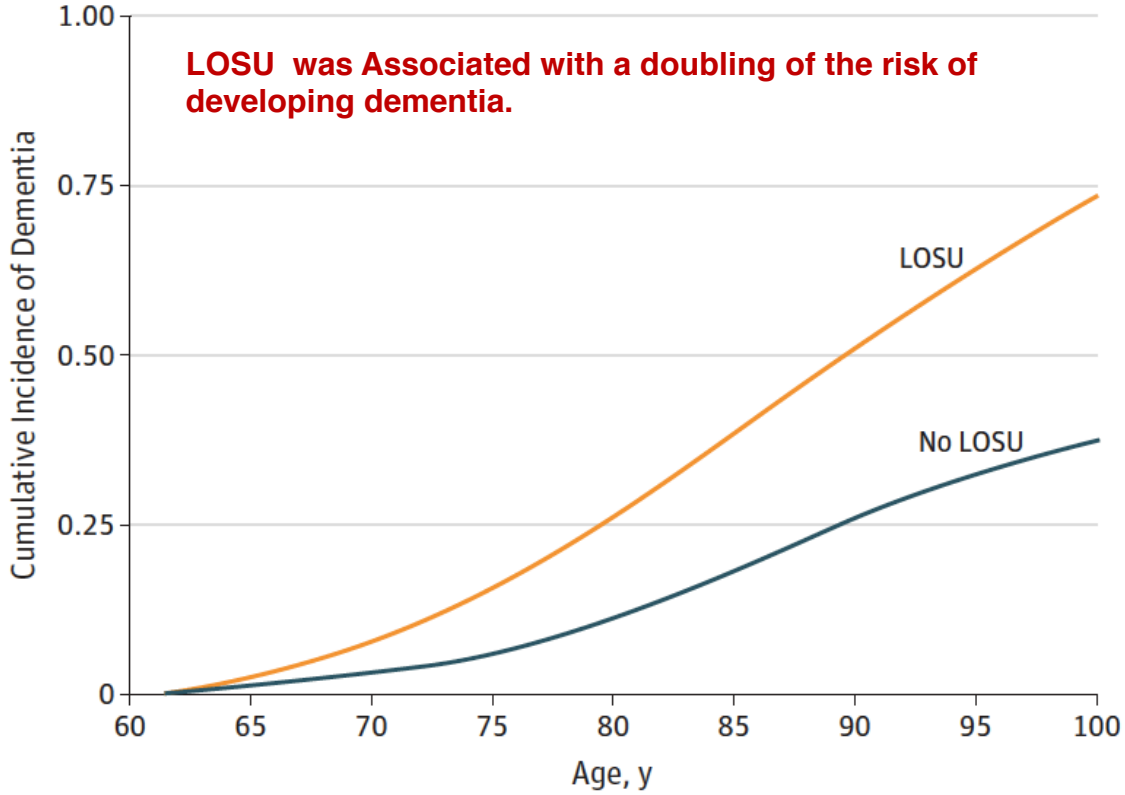
Dementia related epilepsy

The schematic illustrates how cognitive function in people with epilepsy might decline compared to healthy ageing (blue). One model proposes that an initial brain insult ('initial hit') leads to cognitive decline in epilepsy patients simply running parallel to but below the normal trajectory (dashed yellow). These individuals start from lower cognitive performance and also reach the threshold for functional impairment or dementia earlier. An alternative model is that while an initial hit might be a neurodevelopmental disorder or traumatic brain injury, subsequent development of epilepsy is in effect a 'second hit', which leads to further deviation from the normal trajectory (solid yellow). A third proposal is that, with increasing time, the trajectory of cognitive decline in people with epilepsy deviates further from that in healthy individuals leading to accelerated cognitive ageing (red). Inspired by a figure use



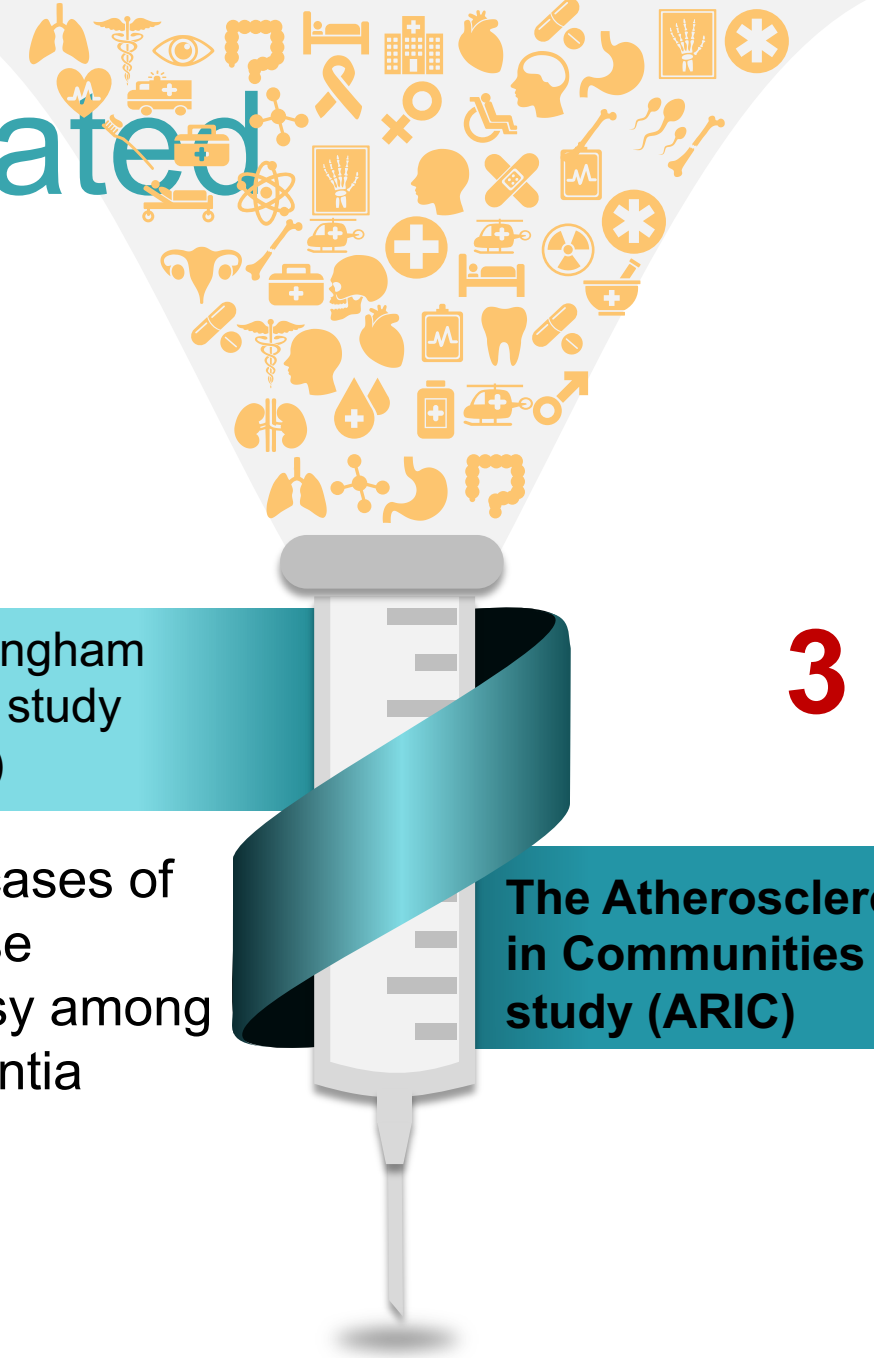
Arjune Sen.BRAIN 2018

LOSU was Associated with a doubling of the risk of developing dementia.



Ophir Keret. JAMA Neurol. 2020

Dementia related Epilepsy



01 Framingham Heart study (FHS)

2 Fold ↑ risk for dementia in cases of epilepsyand a similar increase in risk for subsequent epilepsy among people with diagnosed dementia

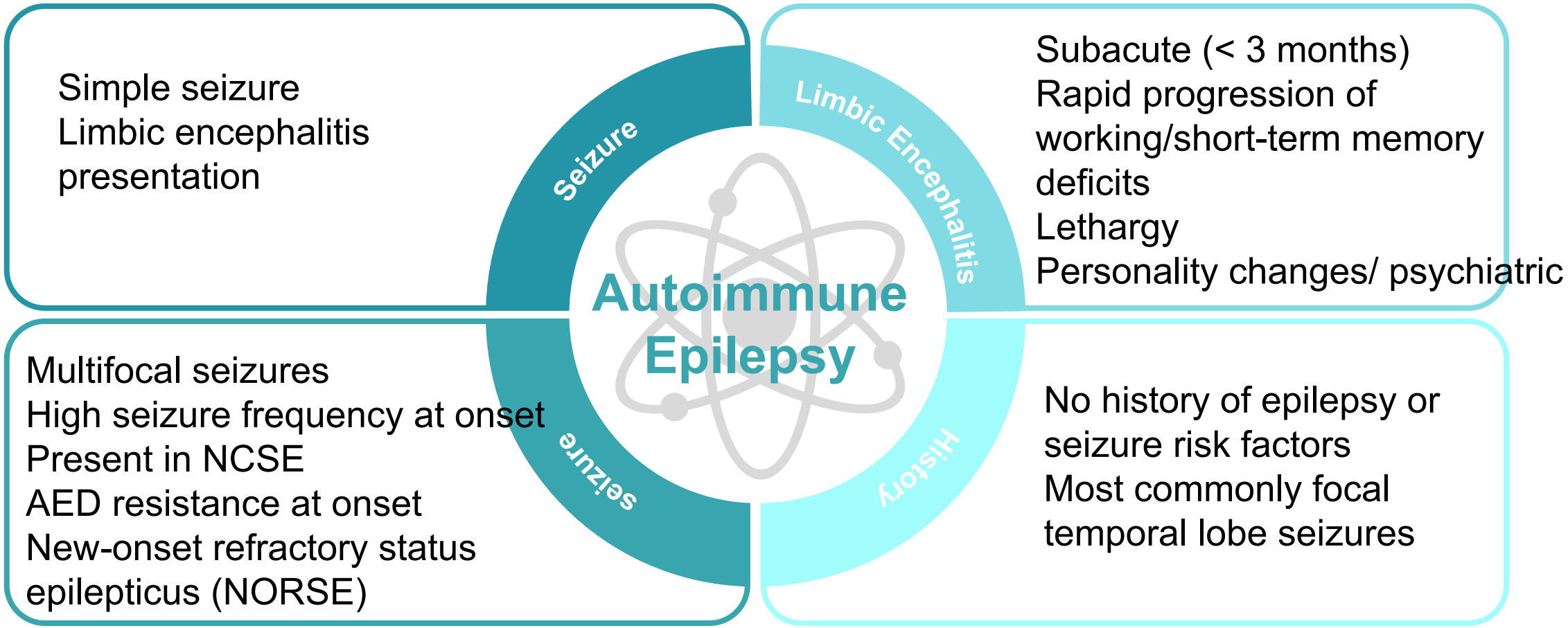
Bidirectional

The Atherosclerosis Risk in Communities study (ARIC) 02

3 Fold ↑ risk for dementia among new onset epilepsy

W. Allen Hauser.Neurology 2020

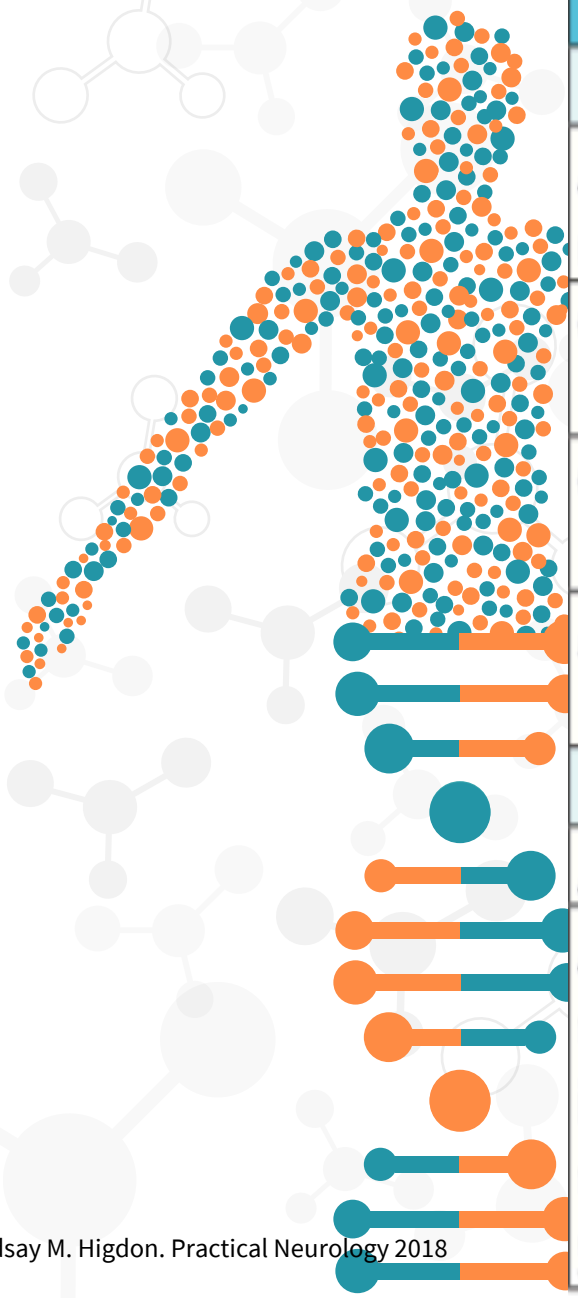
Autoimmune Epilepsy



Lindsay M. Higdon. Practical Neurology 2018

Antigen	Associated Features	Associated Tumors	Isolated Seizures?	Relapses?
SURFACE ANTIGENS				
Common				
LGI1	Autonomic dysfunction, behavior and cognitive changes, FBDS, hyponatremia, insomnia, and limbic encephalitis	Varied (eg, thymoma, endocrine) (10%)	Yes	Yes
NMDAR	Ataxia, autonomic dysfunction, encephalopathy, cognitive symptoms, consciousness reduced/coma, EEG findings of extreme delta brush, and orolingual dyskinesias	Mostly ovarian teratoma in females (40%), testicular germinoma, neuroblastoma	Yes	Yes
Less Common				
AMPAR	Cognitive disorders, confusion, limbic encephalitis, and psychiatric disorders	Breast, SCLC, thymoma	Yes	Yes
CASPR2	Brainstem disorders, cognitive disorders, limbic encephalitis, Morvan's syndrome, peripheral nerve hyperexcitability, psychiatric disorders, and sleep disorders	Thymoma, SCLC	Yes	Yes
$GABA_BR$	Ataxia, behavioral and cognitive disorders, limbic encephalitis, and opsoclonus myoclonus	SCLC	No	Infrequent
Rare				
DPPX	Autonomic dysfunction, brainstem disorders, cognitive disorders, diarrhea, myoclonus, psychiatric disorders, sleep disturbances, tremor, sleep disturbance, and weight loss	B-cell neoplasms	No	Yes
$GABA_AR$	Behavioral disorders, cognitive disorders, consciousness decreased, movement disorders, multifocal cortical–subcortical MRI T2/FLAIR changes	Thymoma, SCLC	Yes	Unclear
Ganglionic AChR	Autonomic dysfunction, cognitive disorders, peripheral neuropathy, and psychiatric disorders	Adenocarcinoma	Yes	Unclear
GFAP	Ataxia, encephalitis, encephalopathy, headache, meningitis, myelitis, psychiatric disorders, and blurred vision	Varied, ovarian teratoma	Yes	Yes
Glycine	Autonomic dysfunction, cognitive disorders, encephalomyelitis (progressive with rigidity and myoclonus), spasms (axial/limb), stiff-person syndrome	Breast, lymphoma, leukemia, SCLC, thymoma	Yes	Yes
mGluR5	Ataxia, Ophelia's syndrome	Hodgkin's lymphoma	No	Unclear
VGCC	Ataxia, encephalopathy, Lambert-Eaton syndrome	Breast, SCLC	Yes	Unclear

Lindsay M. Higdon. Practical Neurology 2018



Antigen	Associated Features	Associated Tumors	Isolated Seizures?	Relapses?
INTRACELLULAR ANTIGENS				
Common				
ANNA-1 (Hu)	Brainstem/limbic encephalitis, sensory neuropathy	SCLC, neuroblastoma, prostate or bladder cancer	Yes	
CRMP-5	Ataxia, chorea, cognitive disorders, encephalopathy, myelopathy, neuropathy	Non-Hodgkin's lymphoma, SCLC, thymoma, tonsillar	Yes	
GAD65	Anxiety, ataxia, brainstem symptoms, limbic encephalitis, and stiff-person syndrome	Breast, colon, lymphoma, renal cell cancer, thymoma	Yes	
Ma/Ta	Brainstem/limbic encephalitis, encephalopathy, and hypothalamic dysfunction	Breast, colon, testicular	Yes	
Less Common				
Amphiphysin	Limbic encephalitis, myelopathy, and stiff person syndrome	Breast, SCLC, ovarian cancer	No	

Abbreviations: AChR, acetylcholine receptor; AMPAR, α-amino-3-hydroxy-5-methyl-4-isoxazole-proprionic receptor; CASPR2, contactin-associated protein 2; DPPX, dipeptidyl-peptidase-like protein 6; FBDS, faciobrachial dystonic seizures; FLAIR, fluid-attenuated inversion recovery; $GABA_AR$, γ-aminobutyric acid A receptor; $GABA_BR$, γ aminobutyric acid B receptor; GFAP, glial fibrillary acid protein; LGI1, leucine-rich glioma inactivated 1; mGluR5, metabotropic glutamate receptor 5; NMDAR, *N*-methyl-D-aspartate receptor; SCLC, small-cell lung cancer; VGCC, voltage-gated calcium channel.[3,8,13]

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APE score

TABLE 2. ANTIBODY PREVALENCE IN EPILEPSY OF UNKNOWN ETIOLOGY SCORE (APE)	
Autonomic dysfunction: atrial bradycardia or sustained tachycardia, blood pressure labile, bradycardia, cardiac aystole, hyperhidrosis, orthostatic hypotension, ventricular tachycardia.	1
Brain MRI: consistent with limbic encephalitis (medial temporal T2/FLAIR signal changes)	2
Seizure or cognitive changes: rapidly progressive mental changes over 1-6 week period or new onset seizure (within 1 year of evaluation)	1
CSF findings consistent with inflammation: protein > 50 mg/dL and lymphocytic pleocytosis > 5 cells/dL, if total number of red blood cells is < 1,000 cells/dL	2
Facial dyskinesia or faciobrachial dystonia	2
Malignancy (excludes cutaneous basal cell carcinoma or squamous cell carcinoma)	2
Psychiatric symptoms (agitation, aggression, emotional lability)	1
Seizure refractory to medical treatment	2
Viral prodrome (low-grade fever, sore throat, rhinorrhea); scored only if there is no underlying malignancy	2
NOTE. An APE Score of ≥4 (max: 15) predicts detection of neural autoantibody in autoimmune epilepsy (sensitivity: 97.7%,; specificity: 77.9%)[17] Abbreviations: CSF, cerebrospinal fluid; FLAIR, fluid-attenuated inversion recovery.	

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RITE score

TABLE 3. ADDITIONAL ITEMS FOR COMPLETE RESPONSE TO IMMUNOTHERAPY IN EPILEPSY SCORE (RITE) SCORE	
Initiation of immunotherapy within 6 months of symptom onset	2
Detected neural plasma membrane autoantibody (AMPAR, CASPR2, DPPX, $GABA_AR$, $GABA_BR$, LGI1, mGluR1, mGluR2, mGluR5, NMDAR.)	2
NOTE: A RITE Score, which consists of APE score + two additional variables, of ≥7 (max:19) predicts response to initial immunotherapy in autoimmune epilepsy (sensitivity: 87.5%,; specificity: 83.8%)[17] Abbreviations: AMPAR, α-amino-3-hydroxy-5-methyl-4-isoxazole-proprionic receptor; CASPR2, contactin-associated protein 2; DPPX, dipeptidyl-peptidase-like protein 6; $GABA_AR$, γ-aminobutyric acid A receptor; $GABA_BR$, γ aminobutyric acid B receptor; LGI1, leucine-rich glioma inactivated 1; mGluR, metabotropic glutamate receptor; NMDAR, *N*-methyl-D-aspartate receptor.	

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Suspected Autoimmune Epilepsy APE Score ≥ 4

Order Neural Ab Testing

Abs to intracellular antigens (see Table 1)

GAD65 Abs

Seronegative

Tumor screening (treat if present)

Abs to intracellular antigens (PNP associated)

Screen and treat tumor Consider cyclophosphamide

Initiate Trial of Immunotherapy (do not wait for Ab testing results)

RITE score ≥ 7 (see Tables 2 and 3)

IVMP 1 g for 3 to 5 days then weekly for 4 to 6 weeks
OR
IVIG 0.4g/kg for 3 to 5 days then weekly for 4 to 6 weeks

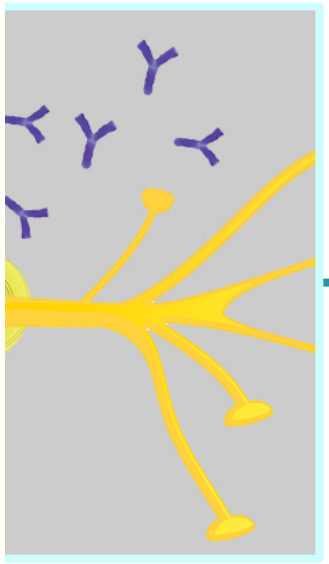
Improvement?

no

Consider second immunotherapy trial with other agent

yes

Supports autoimmune epilepsy diagnosis Taper IVMP/IVIG Consider maintenance immunotherapy



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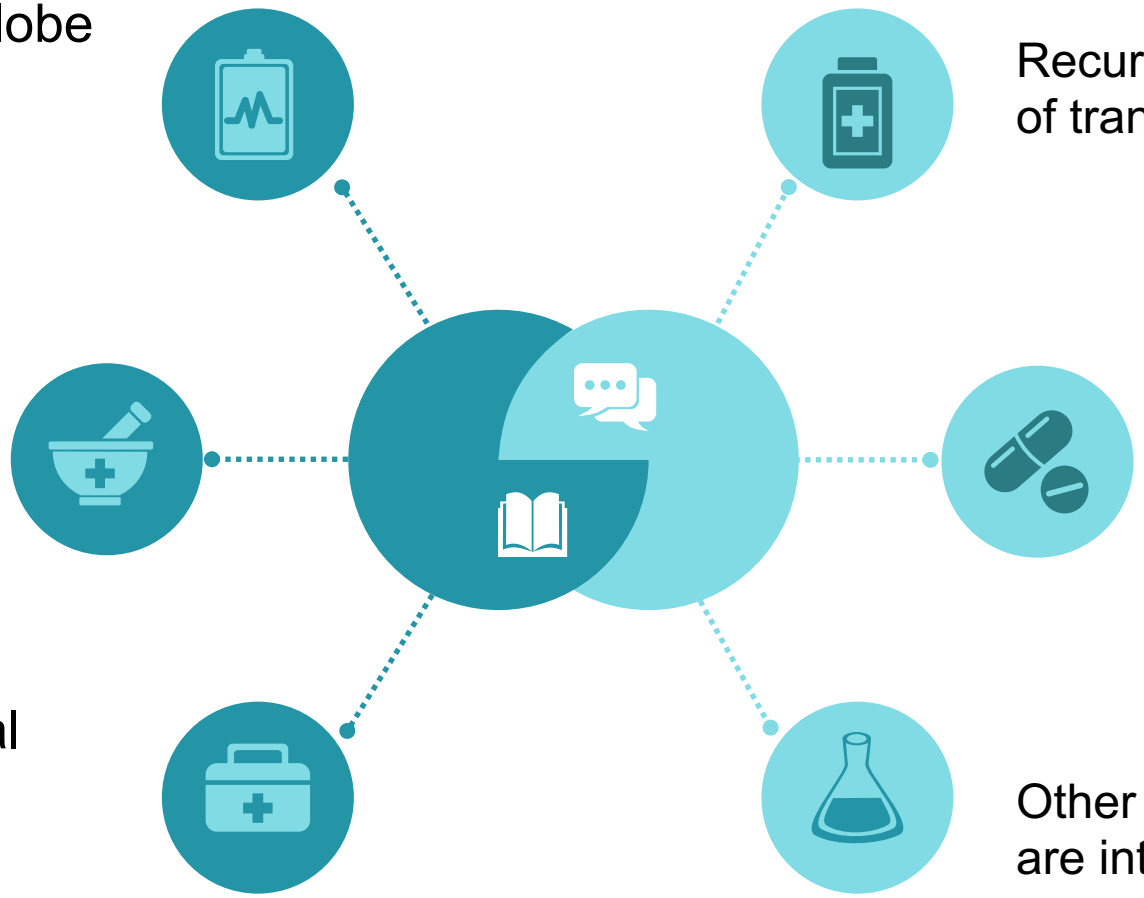
Transient Epileptic Amnesia

Subtype of temporal lobe epilepsy

Recurring episodes of transient amnesia

Amnestic attacks only or accompanied by other manifestations of epilepsy (olfactory hallucinations)



Lasting < 1hr
Often occurring on waking.

Loss of autobiographical accelerated forgetting and topographical memories

Other cognitive functions are intact

Adam Zeman.Epilepsy & Behavior 26 (2013)

Transient Epileptic Amnesia

Diagnostic criteria for Transient Epileptic Amnesia.

1. A history of recurrent witnessed episodes of transient amnesia
2. Cognitive functions other than memory judged to be intact during typical episodes by a reliable witness
3. Evidence for a diagnosis of epilepsy based on one or more of the following:
   a. epileptiform abnormalities on electroencephalography
   b. the concurrent onset of other clinical features of epilepsy (e.g., lip-smacking or olfactory hallucinations)
   c. a clear-cut response to anticonvulsant therapy

Core clinical features of Transient Epileptic Amnesia.

Demographics	Age at onset (y)	62.1 (range 44 to 77)
	Sex distribution	m = 34; f = 16
Amnestic attack characteristics	Number of attacks	median = 10 (IQR 6 to 30)
	Frequency (attacks per year)	median = 12 (IQR 5 to 20)
	Attack duration	median = 30–60 min (range <1 min to days)
	Cessation of attacks on AED	96%
	Some attacks on waking	74%
	Partial amnesia for attack	56%
	Repetitive questioning	50%
	Olfactory hallucinations	42%
	Motor automatisms	36%
	Brief unresponsiveness	24%
Interictal memory	c/o autobiographical memory loss	70%
	c/o accelerated forgetting	44%
	c/o topographical memory loss	36%
Investigations	Interictal epileptiform EEG abnormalities	37%
	Structural lesion on MRI	2%

c/o = complains of.

Neuroimaging features : the medial temporal lobes Sz focus

Adam Zeman.Epilepsy & Behavior 26 (2013)

Epileptic amnesic syndrome(EAS)

Late-onset (mean age=63 years)

Persistent memory difficulties (neuropsychological **test)**

Association with subtle temporal lobe seizures

Complaints of ALF and remote memory impairment

Adam Zeman.Epilepsy & Behavior 26 (2013)


THANK
YOU