



# Reversible Splenic Lesion Syndrome

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# Introduction

- ▶ Reversible splenial lesion syndrome (RESLES) is a clinical radiological syndrome characterized by the presence of a reversible lesion involving the splenium of the corpus callosum (SCC).
- ▶ First described in 1999, by Kim et al.

# Etiology: Epilepsy & AED related

- ▶ Epilepsy related:

Seizures (type and frequency are irrelevant)

AED ( Carbamazepine, phenytoin, Lamotrigine)

Withdrawal (Most Relevant)

- ▶ AED related: when used as mood stabilizer, migraine prophylactic, neuropathic pain

# Etiology: Infections

- ▶ Clinical picture; encephalopathy, and encephalitic features. Rarely: Visual hallucinations.
- ▶ Viral infections were the most common: Influenza virus, EBV, and HSV.
- ▶ 50% encephalopathy with normal CSF composition.
- ▶ MRI normalized after 1-2 weeks, with no clear correlation with clinical recovery.
- ▶ Few patients with Viral encephalopathy reported extra corpus callosum lesions, which is associated with less favorable clinical outcomes

# Etiology: Metabolic conditions

- ▶ Clinical features consisted of abnormal sensorium and focal neurologic deficits.
- ▶ Causes: Hypoglycemia, hyperglycemia, and hypernatremia.
- ▶ Clinical outcomes:  
Hypernatremia + seizures + AED led to vegetative state  
Hypoglycemia led to hemiparesis; MRI shows bilateral extension of the CC lesion to the corona radiata.
- ▶ Clinical outcomes; favorable unless presence of poor prognostic indicators.

# Etiology: Medications \ Toxins

- ▶ Methyl bromide.
- ▶ Chemotherapy: cisplatin and carboplatin.
- ▶ Combination of escitalopram, and olanzapine.
- ▶ Clinical outcomes; favorable with cessation of the offending agent.
- ▶ No MRI follow up, Diagnosis can not be fully established.

# Etiology: Miscellaneous Conditions

- ▶ Anorexia nervosa.
- ▶ Malnutrition.
- ▶ Vitamin B12 deficiency.
- ▶ Charcot Marie-Tooth disease.
- ▶ High-altitude cerebral edema.
- ▶ Systemic lupus erythematosus.
- ▶ Eclampsia.
- ▶ Rabies and Mumps Vaccine
- ▶ Follow up MRI: Recovery within 1-4 months

# Pathophysiology

- ▶ Why the SCC? Lack of adrenergic tone, more prone to hypoxic vasodilation and autoregulation failure with resultant overperfusion.
- ▶ The brief and reversible failures of cellular fluid regulation that occur in convulsions.
- ▶ Antiepileptic drug level fluctuations and changes in electrolytes hemostasis led to myelin sheath edema.
- ▶ Hypoglycemia led to reduction of cell membrane ionic pump activity and a consecutive shift of cerebral water from extracellular to intracellular space.



# Clinical picture

- ▶ RESLES is an asymptomatic disorder
- ▶ The clinical manifestation is related to the underlying pathology.
- ▶ It may or may not include encephalopathy.

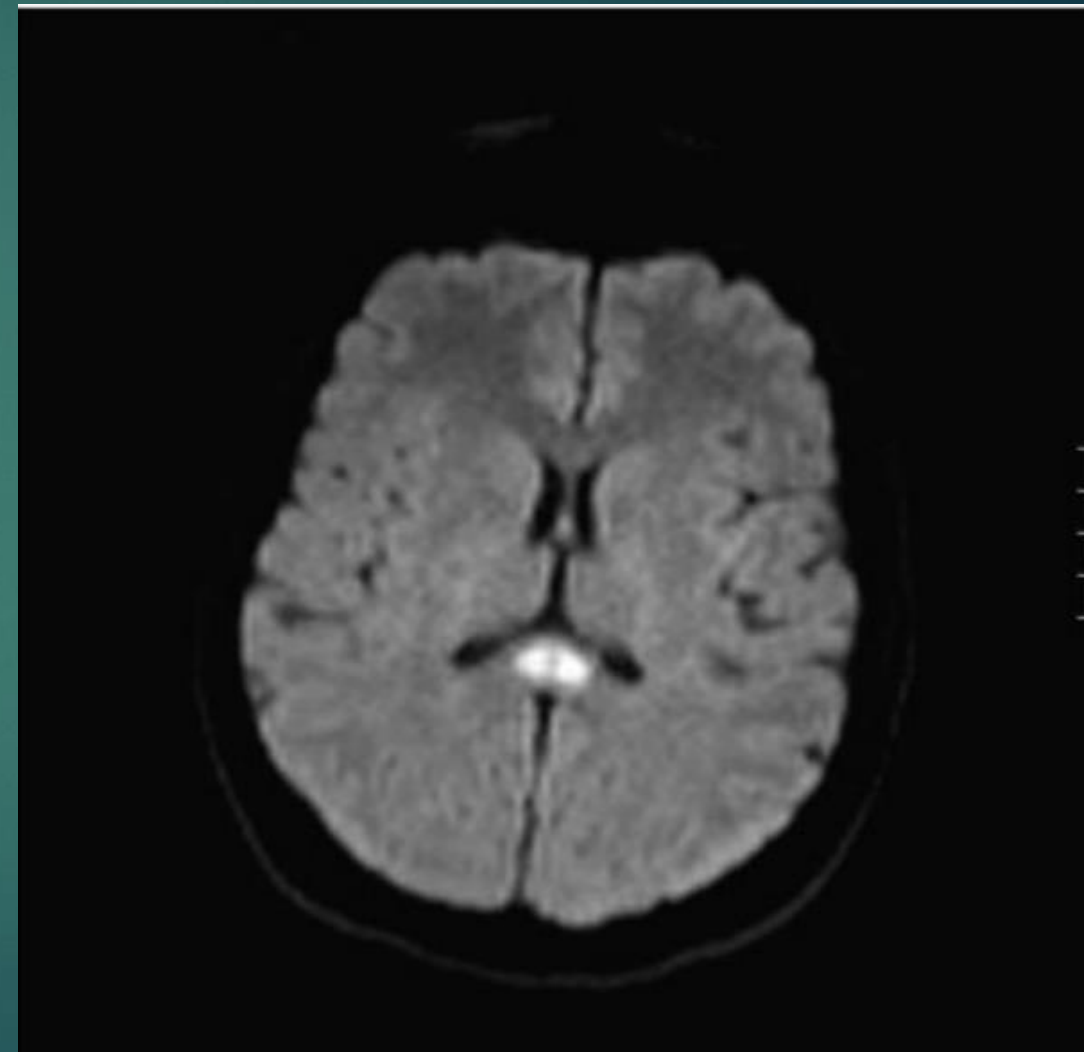
# Diagnosis

## Revised inclusion criteria of Garcia-Monco et al. (2011)

1. Lesions must involve the SCC as shown by MRI
2. Lesion must be symmetrical and oval shape
3. Lesion must disappear on follow up MRI
4. The main lesion must be centered on the SCC.
5. The absence of disseminated encephalopathy
6. Absence of concurrent demyelinating disorders.

# MRI Findings

DWI  
round lesions  
Earliest sign



# MRI Findings

T2-weighted  
round lesions



# MRI Findings

FLAIR  
round lesions



# Potential Biomarker

- ▶ Urinary  $\beta$ 2-microglobulin reported to be elevated in few patients with RESLES.

# Prognosis

- ▶ Full and quick recovery in majority of cases.
- ▶ Poor prognosis indicators are:
  - 1- Severe disturbances in consciousness at the onset of the disease
  - 2- Diffuse slow waves on electroencephalogram (EEG) findings
  - 3- Extracallosal lesions

Questions?