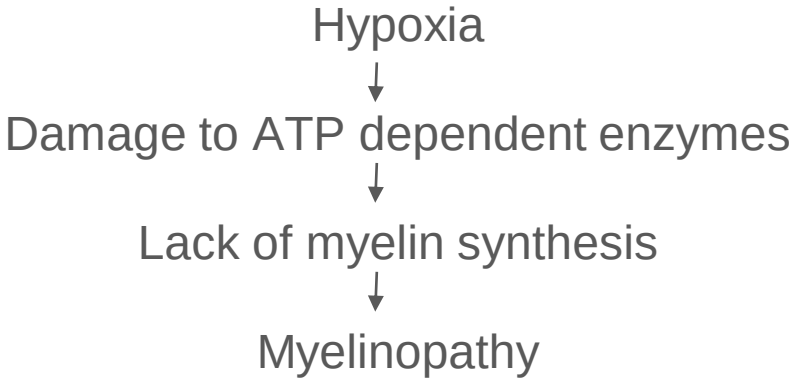
Delayed posthypoxic leucoencephalopathy (Grinker myelinopathy)

- Sana Rajani

Pathophysiology



Clinical course

- Biphasic course

Altered mental status (d/t hypoxic injury)

↓

Lucid interval (7-21days)

↓

DPHL

Past clinical history of

Global cerebral hypoxia due to

- CO poisoning
- Narcotic poisoning
- General anesthesia

} Anoxic anoxia

- Cardiopulmonary arrest
- Strangulation

} Ischaemic anoxia

- Hemorrhagic shock-

Anemic and ischaemic anoxia

Clinical presentation

Parkinsonism

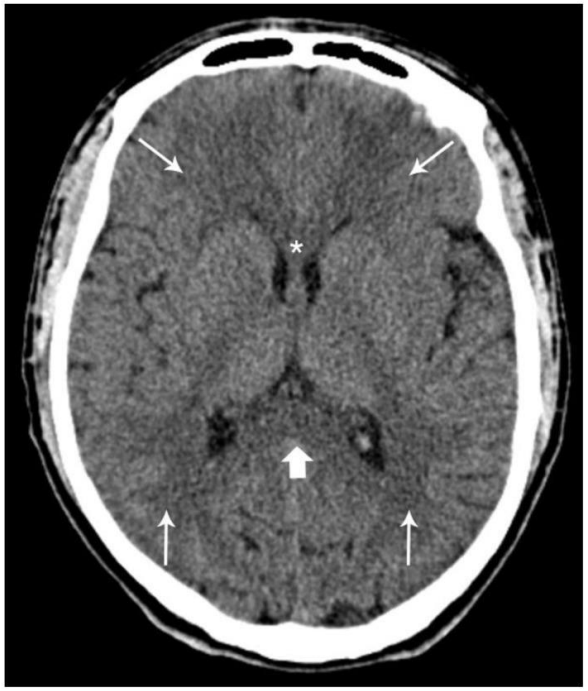
- Psychomotor retardation
- Akinetic mutism

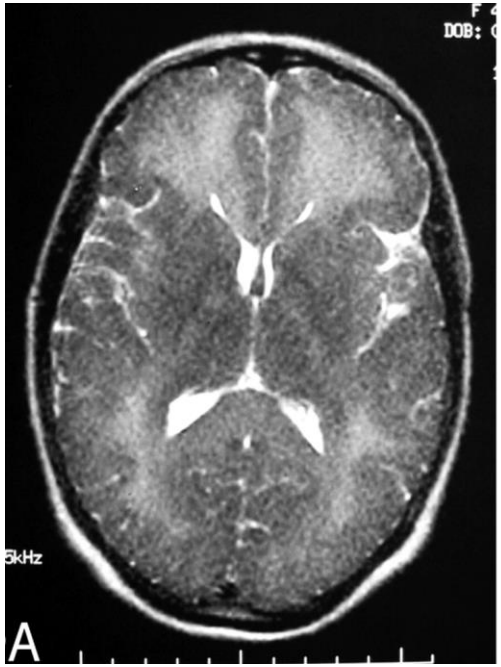
Diagnosis

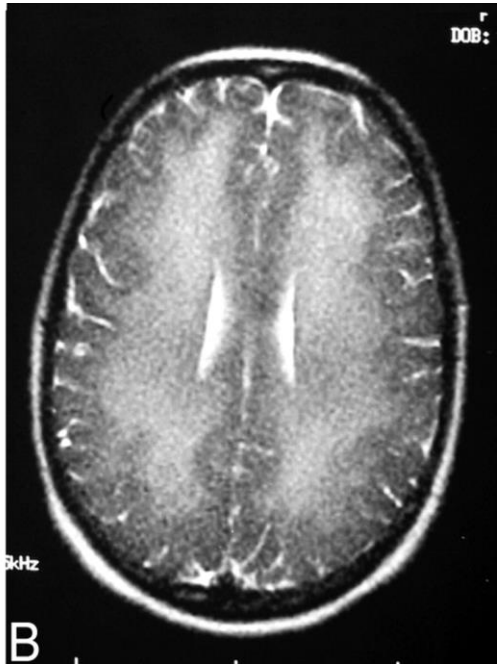
Biphasic course = History of hypoxia +neurological relapse + **MRI findings**

Investigation- MRI

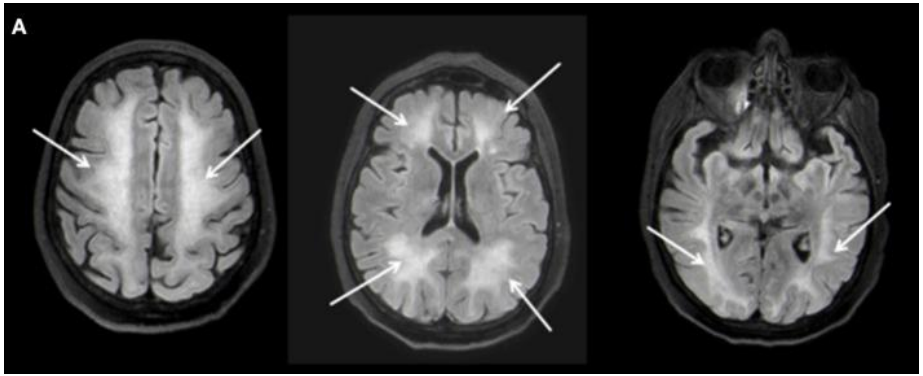
- T2 weighted: Bilateral, diffuse hyperintensity of white matter





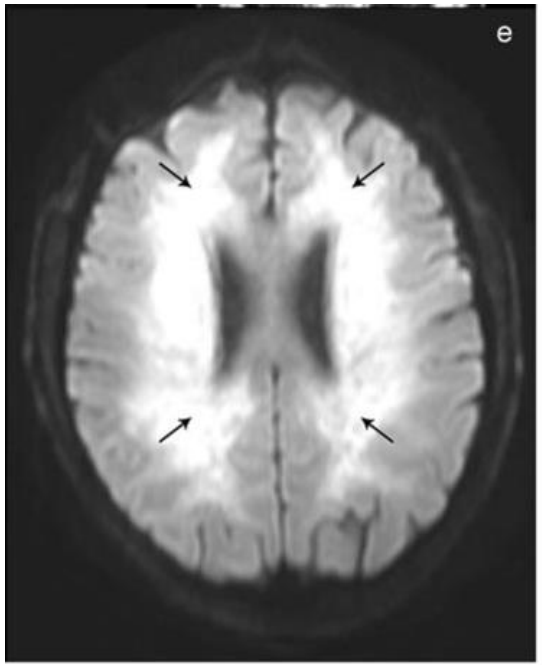


- FLAIR- subcortical hyperintensities

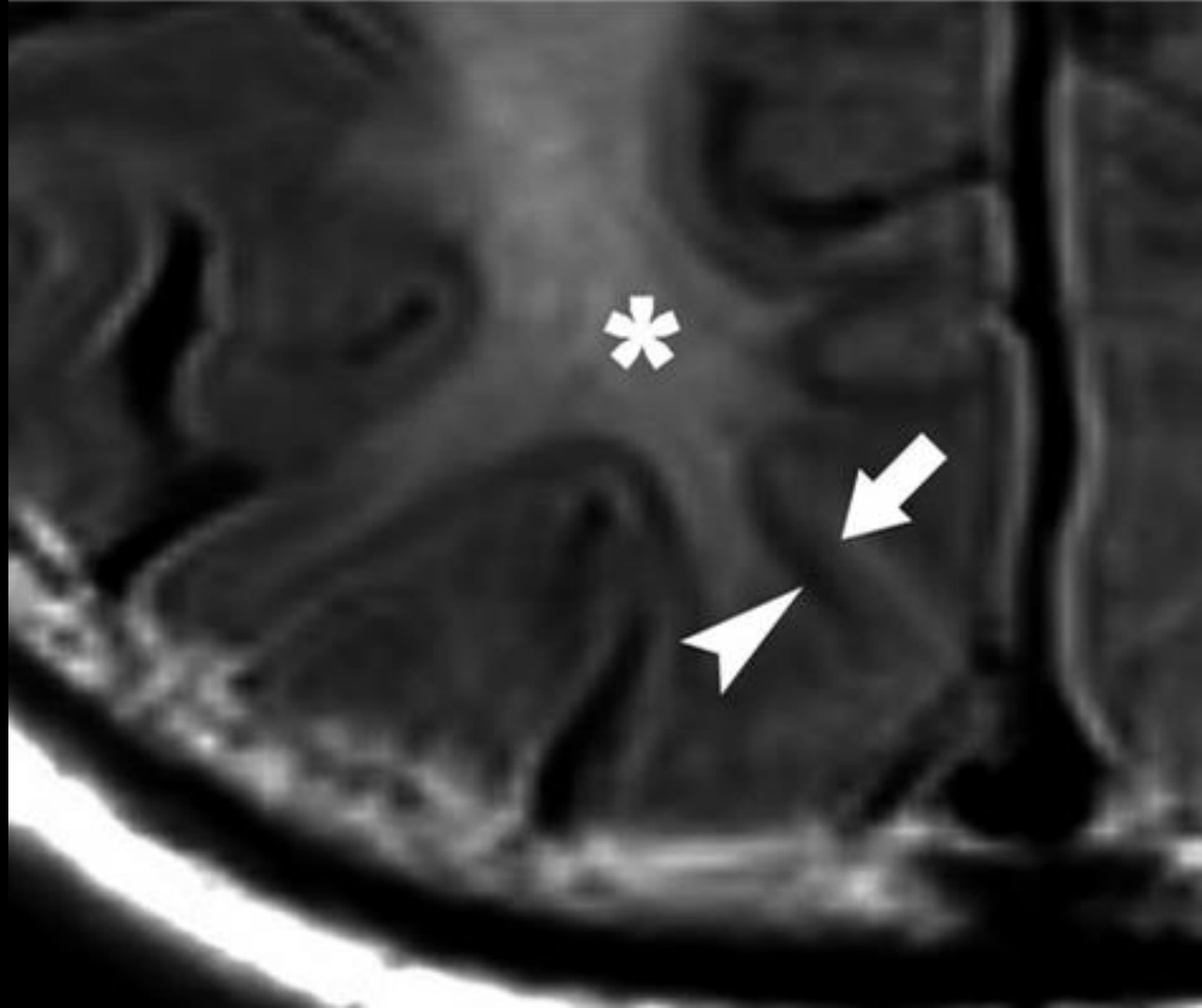


Investigation- MRI

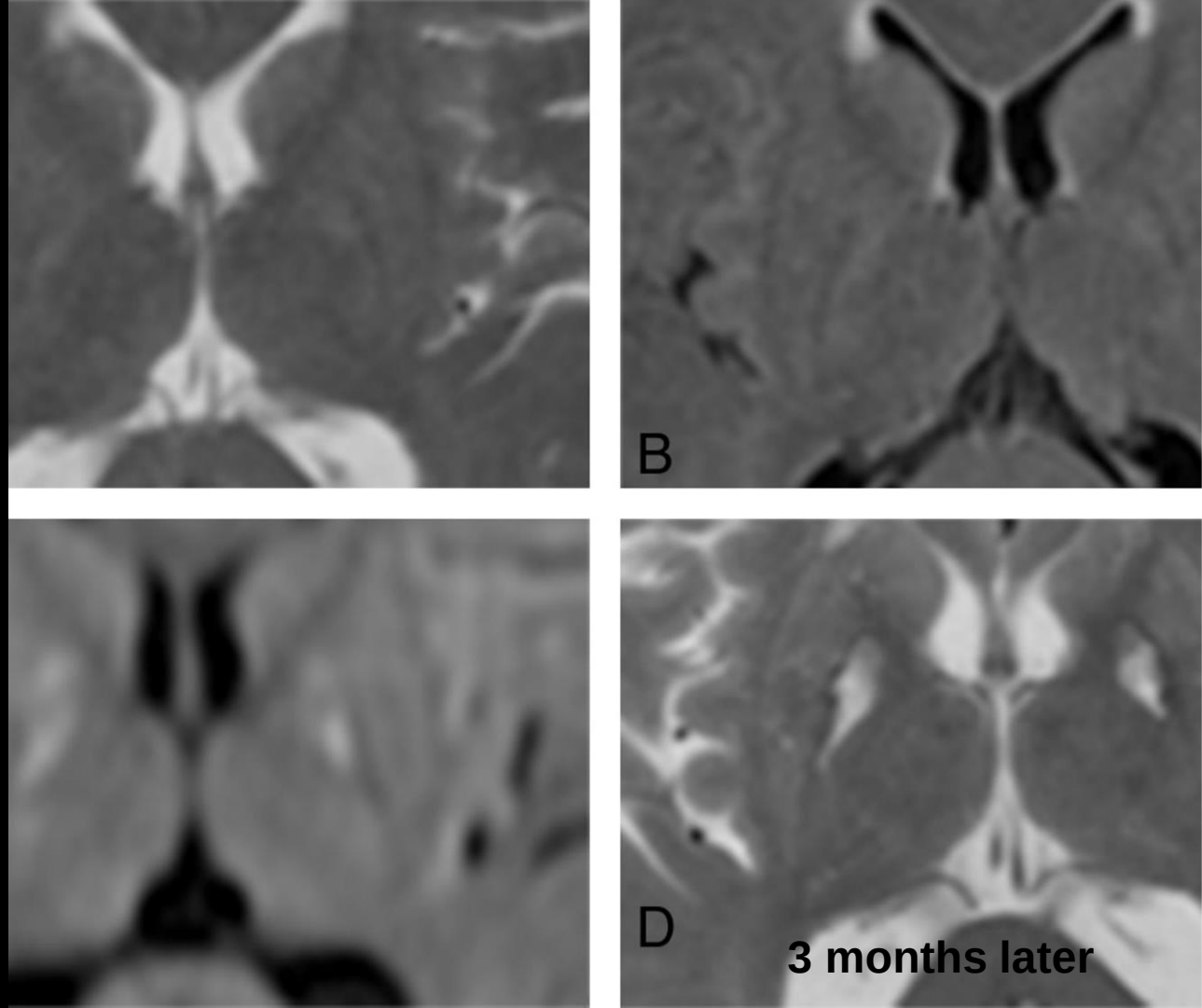
- DWI- Restricted diffusion



Sparing of U fibres

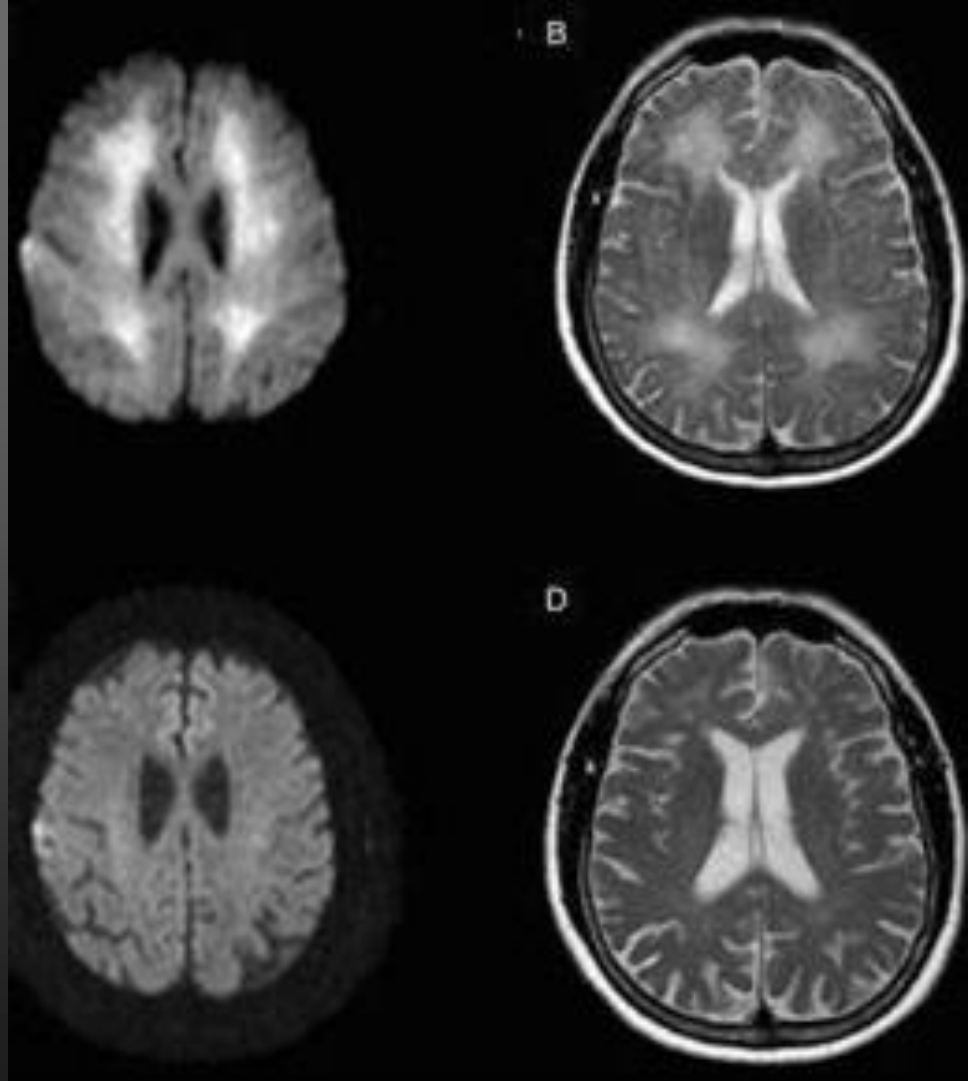


Hyperintensity in Globus Pallidus



During the episode of relapse

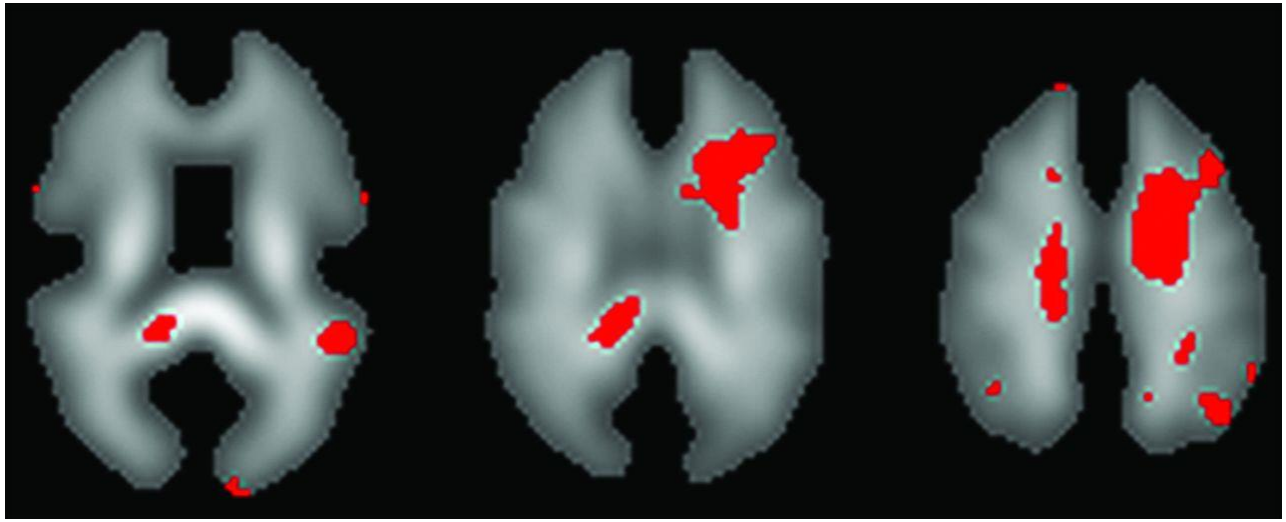
One year later



Other investigations

MR spectroscopy- choline peak, decreased NAA, Lactate (+)

DTI- structural integrity resolves in 5 months

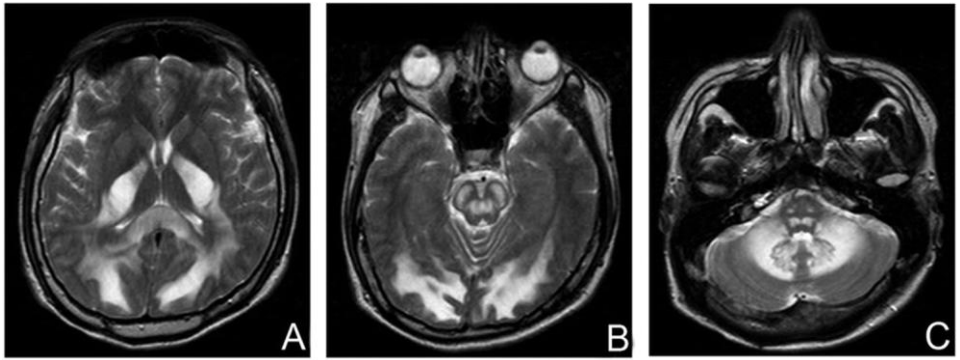


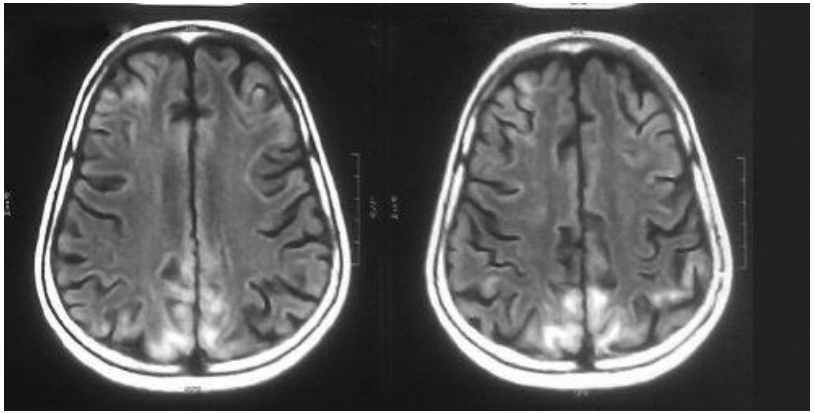
Differential diagnosis

- T2 hyperintensity and restricted diffusion seen within 48 hours of a hypoxic episode in case of stroke
- Alternative diagnoses should be considered if white matter lesions are non-continuous, enhancing, extend to overlying cortex, and are accompanied by seizures

Differential diagnosis

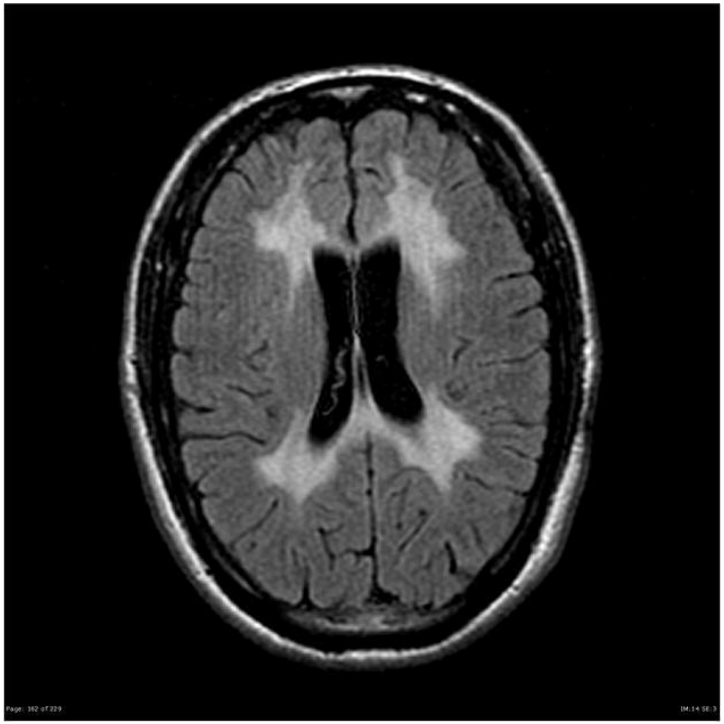
- Inhaled heroin- spongiform leukoencephalopathy, T2 hyperintensity sparing frontal and temporal white matter.
- PRES- white matter supplied by anterior circulation





Inherited disorder or myelination

Metachromatic leucodystrophy - deficiency of arylsuphatase-A



CSF

- Raised myelin basic protein

Prognosis

- Mixed results.

  Fatal ⟷ Recovery with some neurodeficit

- Relatively good prognosis

Treatment

- Steroids
- Levodopa
- Amantidine
- Antioxidant therapy with vitamin E, vitamin C, B-complex vitamins, and coenzyme Q10

Thank you

Summary table for delayed hypoxic-ischemic demyelination.

Etiology	Prolonged moderate hypoxic-ischemic event (++ CO intoxication)
Gender/Age	No predilection
Incidence	~10% of CO intoxications
Time of Presentation	2–40 days after the initial event
Clinical Presentation	Parkinsonism or Akinetic-Mutism after a complete clinical recovery
Diagnosis	Clinical history + suggestive imaging findings; exclusion of other causes
CT	Subtle bilateral hypodensity in the peri-ventricular and deep cerebral white matter
Conventional MR sequences	Confluent, bilateral and symmetrical peri-ventricular and deep white matter (++ centrum semiovale) hyperintensity on T2/FLAIR; absence of involvement of gray matter or infratentorial structures; also absence of mass effect or enhancement
DWI	Prolonged restricted diffusion (lasting longer than usually seen in ischemic cytotoxic edema)
MRS	Decreased NAA and increased Cho peak; presence of a lactate peak
Prognosis	generally good, with both clinical and radiologic improvement
Treatment	++ symptomatic/supportive; in the context of CO intoxication, hyperbaric oxygen therapy is controversial