Wernicke Encephalopathy

- thiamine deficiency impairs dependent enzymes and results in glutamate accumulation/cell death
- 50% of WE cases occur in nonalcoholic population, including children
- nonalcoholic WE has same pathophysiology but different etiology (eg. malnutrion, hyperalimentation)

Wernicke Encephalopathy

WE = triad of ataxia, oculomotor abnormalities, confusion

- ocular involvement includes:
  - horizontal and vertical nystagmus
  - uni- or bilateral CN VI paresis common
  - retinal hemorrhages may occur
- also polyneuropathy in 80% of cases

Wernicke Encephalopathy

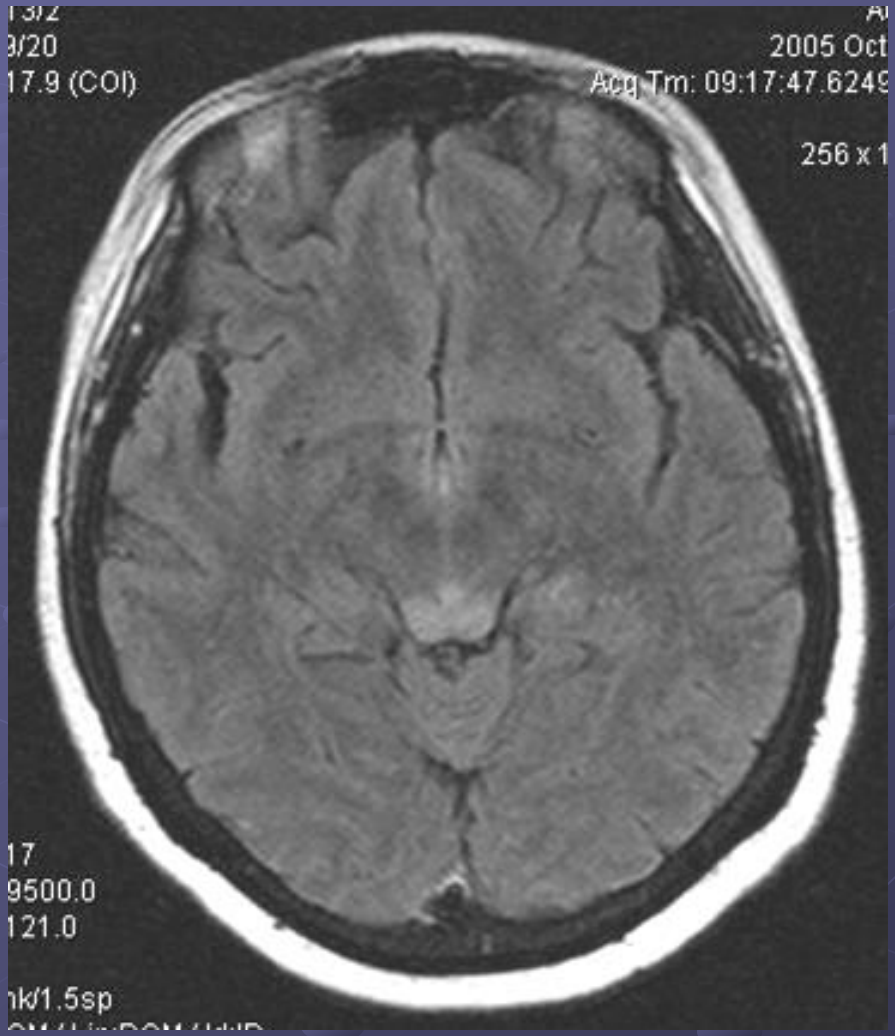
Treatment = immediate administration of IV thiamine

- ocular palsies respond first.
- ataxia, apathy, confusion clear more slowly (weeks)
- 50% left with slow shuffling gait

Wernicke Encephalopathy

MR imaging findings:

- T1W – may see ↓SI in periaqueductal gm, mamillary bodies, hypothalamus and medial thalami
- T2W - ↑SI around 3rd ventricle, mamillary bodies, hypothalamus, medial thalami and midbrain (periaqueductal grey matter)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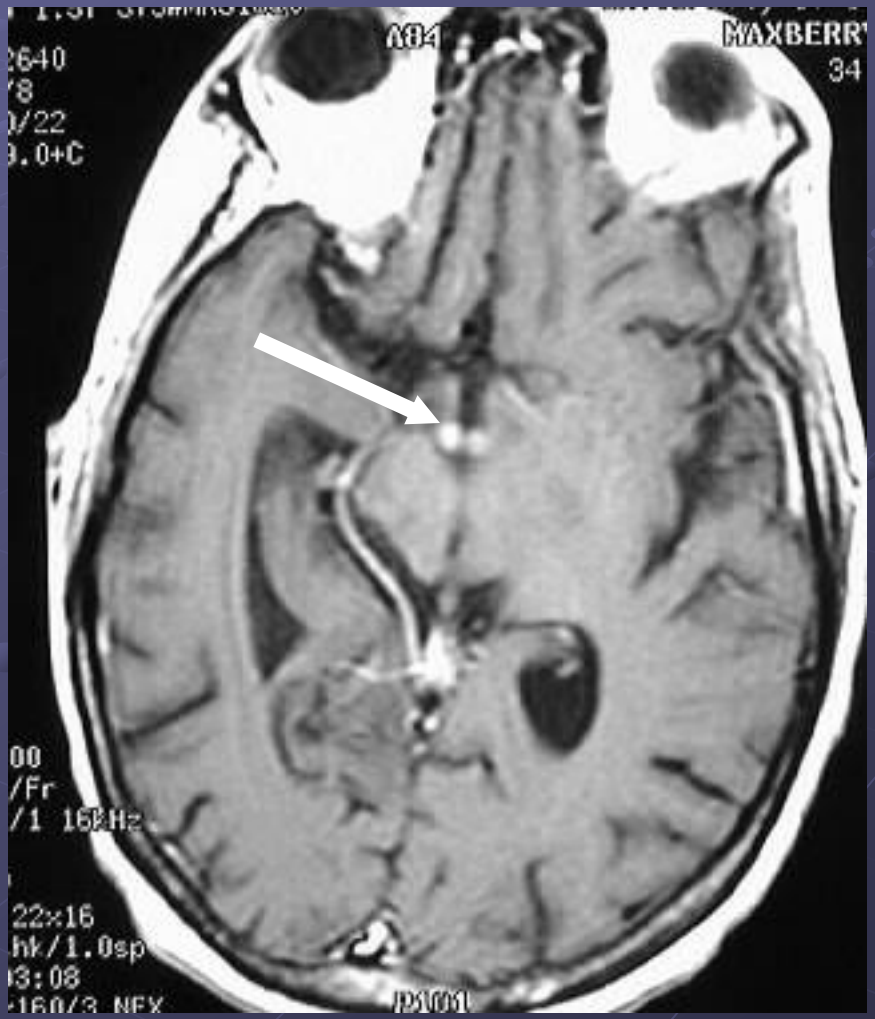
Wernicke's syndrome

Can effect peri-aqueductal gray and Thalamus



Wernicke's syndrome

- DDX: bilateral thalamic lesions
  - Systemic disease (toxic, metabolic, etc)
  - Central brain process (midline herniation, Internal Cerebral Vein thrombosis)
  - Astrocytic neoplasms
  - Lymphoma