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Psychiatry and Addictions Case Conference

UW Medicine | Psychiatry and Behavioral Sciences

IDENTIFYING AND ADDRESSING WERNICKE KORSAKOFF SYNDROME

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SPEAKER DISCLOSURES

No conflicts of interest to disclose.

PLANNER DISCLOSURES

The following series planners have no relevant conflicts of interest to disclose; other disclosures have been mitigated.

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OBJECTIVES

1. Define Wernicke Korsakoff syndrome as a spectrum of neuropsychiatric disorders caused by thiamine (vitamin B1) deficiency
2. Identify clinical populations at increased risk for thiamine deficiency and Wernicke Korsakoff syndrome
3. Apply evidence-based diagnostic criteria to improve clinical recognition of Wernicke encephalopathy
4. Initiate timely, guideline-concordant treatment, including appropriate dosing, route of thiamine administration, and electrolyte repletion
5. Describe the neurocognitive sequelae and prognosis associated with Korsakoff syndrome and relevant neuroanatomic correlates
6. Recognize and mitigate common management errors in the prevention, diagnosis, and treatment of Wernicke Korsakoff syndrome

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BIG PICTURE – WHAT IS WERNICKE KORSAKOFF SYNDROME?

Continuum of neuropsychiatric disorders due to thiamine (B1) deficiency

Wernicke Encephalopathy (acute) → **Korsakoff Syndrome (chronic)**

*Potentially **reversible** neurologic dysfunction*

*Profound, often **irreversible** amnesia and confabulation*

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EPIDEMIOLOGY

- **What predisposes to thiamine deficiency?**
  - **Patients with excessive alcohol consumption = primary risk factor (90-93% of cases)**
  - Malnutrition 2/2 gastric bypass surgery
  - Chronic illness
  - Patients with severe psychiatric disorders
  - Patients with other severe substance use disorders
  - Pregnant patients with hyperemesis gravidarum
- **Disproportionately affects men (approx. 80% of cases), but women appear to be more more susceptible**
  - Female-to-male ratio for WE higher than ratio for alcohol dependence *(Harper et al.)*
- **Significantly underdiagnosed**
  - Clinical detection: 0.04-0.13% prevalence in general population *(Sechi et al.)*
  - Autopsy studies: 0.4-2.8% prevalence in general population *(Li et al.)*

PATHOPHYSIOLOGY

- Poor dietary intake → inhibition of thiamine absorption in GI tract → disruption of thiamine transport across gut-blood-brain barrier → decreased synthesis of thiamine pyrophosphate → mitochondrial dysfunction
- EtOH inhibits key enzymes required for thiamine phosphorylation and utilization → decreased neuronal energy metabolism
- Upregulation of TLR4/MyD88 pro-inflammatory pathway in frontal cortex and cerebellum → selective neuronal vulnerability and damage

Mg deficiency → impaired thiamine utilization (may blunt response to thiamine)

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ACUTE IDENTIFICATION & DIAGNOSIS OF WERNICKE ENCEPHALOPATHY

- Diagnosis = clinical
- Classic triad (ophthalmoplegia, ataxia, confusion) present in < 1/3rd of cases
- Caine Criteria (need 2 of 4)*
  - Dietary deficiency
  - Oculomotor abnormalities
  - Cerebellar dysfunction
  - Altered mental status/memory impairment
- Nutritional history + lab assessment (magnesium)

*Sensitivity of ~85% (compared to ~22% with classic triad); not intended to apply to patients without hx of heavy EtOH use

Serum thiamine is not reliable for diagnosis; blood concentrations do not reflect tissue/brain levels!

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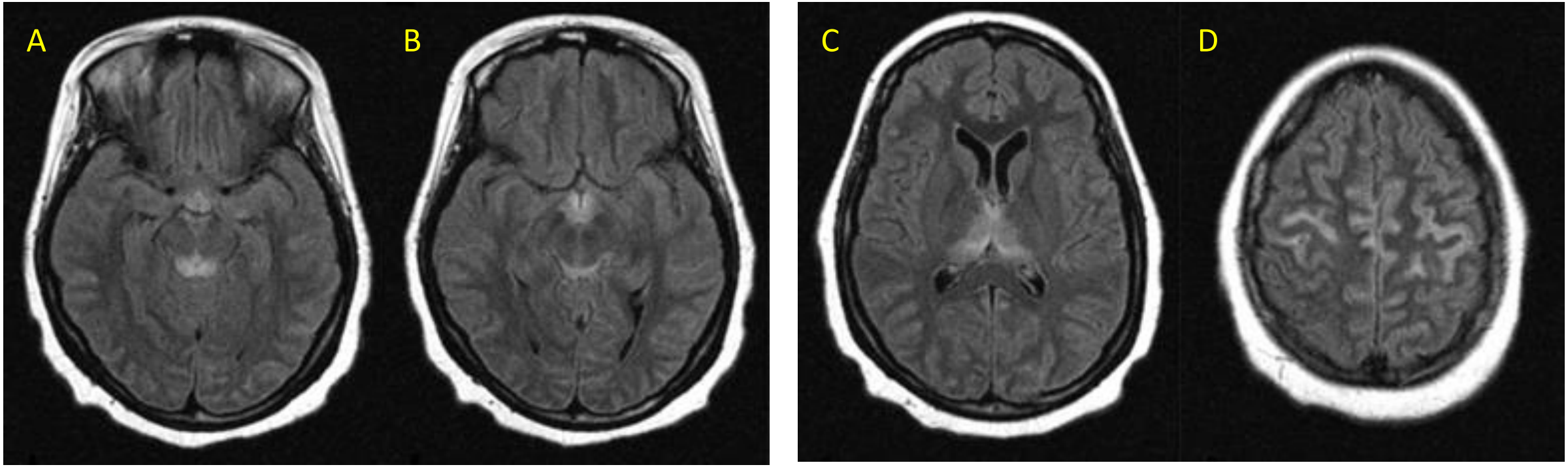
ACUTE IDENTIFICATION & DIAGNOSIS OF WERNICKE ENCEPHALOPATHY

MRI brain can be supportive, but is not necessary for diagnosis



20yo pregnant patient with HG and presumed WE

Hyperintensities in **A) periaqueductal region**, **B) mammillary bodies**, **C) thalamus**, and **D) frontal-parietal corticies**

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ACUTE MANAGEMENT OF WERNICKE ENCEPHALOPATHY

- Immediate administration of parenteral thiamine
- Administration of parenteral thiamine considered urgent; notable risk of progression to irreversible KS
- What about oral thiamine?
  - **Inadequate!** → very erratic GI absorption
  - Chronic alcohol use → to transcriptional suppression of thiamine transporter-1 → impaired dietary thiamine absorption in small intestine + impaired thiamine uptake from bacterial sources in colon
- 20% mortality rate and progression to KS in 85% of survivors when inadequately treated
- Supportive care: hydration, correction of electrolytes, management of alcohol withdrawal, folate supplementation (rare, but can be co-morbid)

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ACUTE MANAGEMENT OF WERNICKE ENCEPHALOPATHY

- Prompt administration of thiamine → ocular sx improvement in hours to days (though up to 60% may have residual horizontal nystagmus)
  - If no improvement in ophthalmoplegia, consider alternate diagnoses
- Vestibular function + ataxia improves around second week of treatment (in up to 40% of patients)
- Confusion improves over days to weeks
- Signal abnormality on MRI tends to resolve with clinical improvement

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THIAMINE DOSING AND ADMINISTRATION

- ASAM guidelines: give high-dose parenteral thiamine for pts w/ suspected WE/KS or severe AUD and risk factors for thiamine deficiency
- Typical regimen: 500 mg IV TID x2-3d, followed by 250 mg IV or IM qd x3-5 days or until clinical improvement plateaus
  - No specific ASAM guideline, but they generally support giving thiamine 200-500 mg IV TID, 64mg/kg Mg, and 400-1000 mcg IV folate for patients with s/sx WE
- Novo-Veleiro et al. - In patients with WE, only 20.9% received >300 mg/day of thiamine; delayed or insufficient treatment → poorer recovery + increased mortality

Overtreatment strongly preferred over undertreatment; no lit evidence of thiamine toxicity from over-administration!

Give thiamine and glucose in any order per ASAM

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PROPHYLAXIS

- 2020 ASAM guidelines, for inpatient alcohol withdrawal:
  - Thiamine: IV/IM preferred, 100 mg qd x3-5d; PO can also be offered
  - ICU patients: always give thiamine
  - Give Mg if serum levels low, arrhythmias, electrolyte issues, prior seizures
  - Give phos if serum <1 mg/dL; nutrition if 1-2 mg/dL
  - Consider folate
- 2020 ASAM guidelines, for outpatient alcohol withdrawal:
  - "Patients can be offered oral thiamine. Typical dosing is 100 mg PO per day for 3-5 days." (*Recommendation IV.9*)

***Controversial!**

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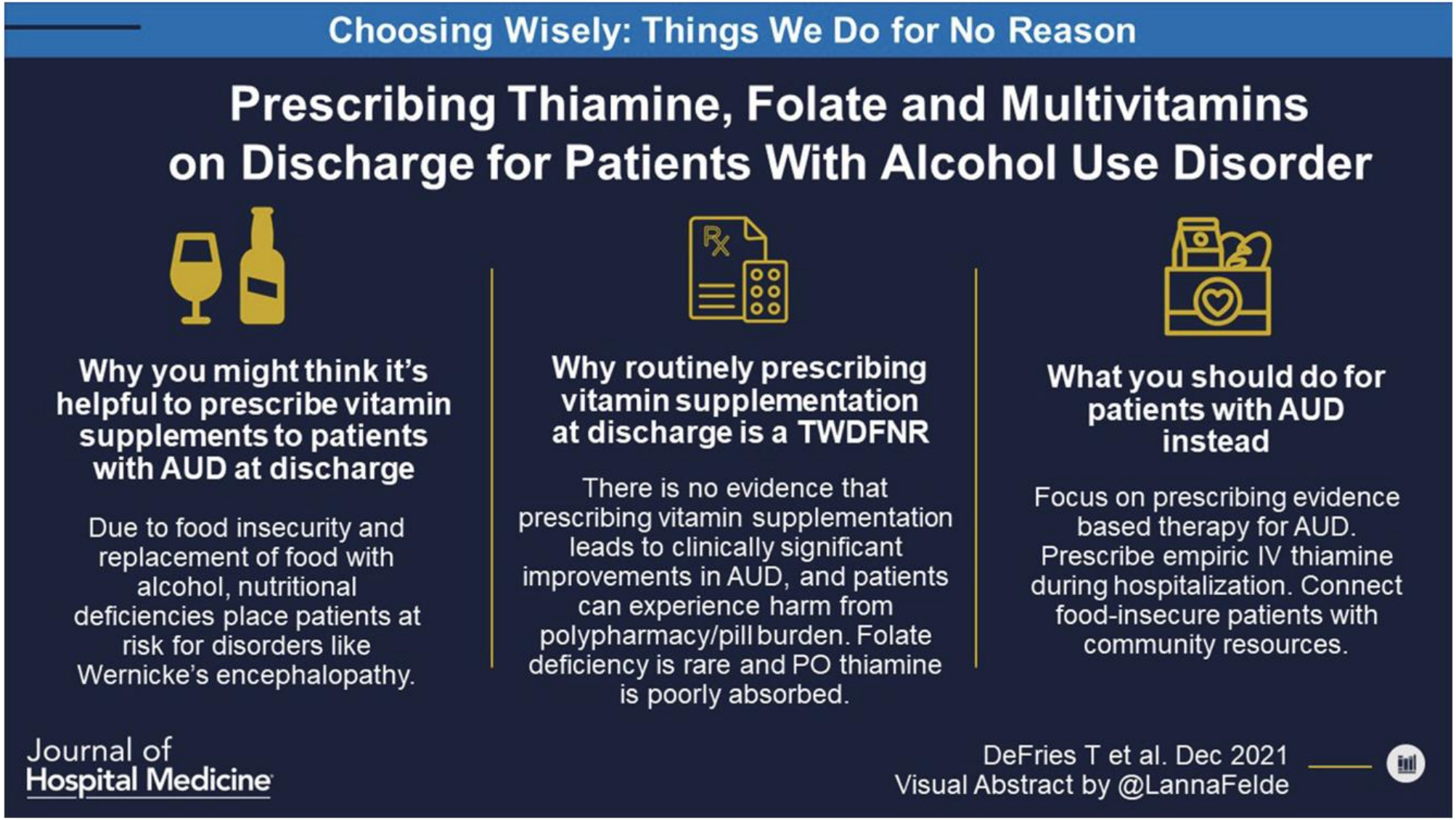

DeFries T, Leyde S, Haber LA, Martin M. Things We Do for No Reason™: Prescribing Thiamine, Folate and Multivitamins on Discharge for Patients With Alcohol Use Disorder. J Hosp Med. 2021 Dec;16(12):751-753. doi: 10.12788/jhm.3691. PMID: 34730500.

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QUICK REFLECTION

WHAT IS YOUR PRACTICE AND EXPERIENCE WITH PRESCRIBING THIAMINE TO PATIENTS WITH AUD IN THE OUTPATIENT SETTING?

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r/dementia • 2y ago

Anyone else caring for someone with Korsakoff's?

I feel like the resources and places discussing personal experiences with Korsakoff's are kind of scant, so wanted to pose the question. I am emotionally struggling to care for my mother with it for many reasons, but the fact that it was alcohol-inflicted (and the many years of awful behavior prior) make it feel impossible to reconcile my resentment with my sadness. Her constant violent com

Anything anyone wants to relate re: their personal expe
I feel pretty isolated navigating this.

23 · 52

r/AdultChildren • 3y ago

Mother is in a home with Korsakoff - what can we still do?

Hello everyone,

I thought I'd share my story of my mothers (and my own) alcoholism. I have some questions and a few tips from my own experiences that I thought I'd put down for others to read. (I apologise if this all reads as if it's been written with a longsword, I've been at it for a while now and I'm tired :P)

TL;DR: my tale is probably similar to many others from what I read. My mother (now 71) had drank pretty much all her life. There's a lot more to this story, the general gist of which is written below. The damage is now done, she has Korsakoff-Wernicke syndrome and is in a special care home. She has difficulty forming sentences and I'm wondering if there's anything we can still do to help her mind a bit or at least give her more peace; questions and

My sister has been in a memory care facility for 4 years because of Wernecky Korsakoff syndrome brought on by a chronic eating disorder and alcoholism. It's horrible to deal with, and I'm sorry you're in this situation! I'm trying to connect with others because there really isn't a lot of information about what happens to people after they quit. I know that it really depends on how far WK progressed, but it has been really helpful for me to hear the experiences of other people. I'd be happy to share my journey.

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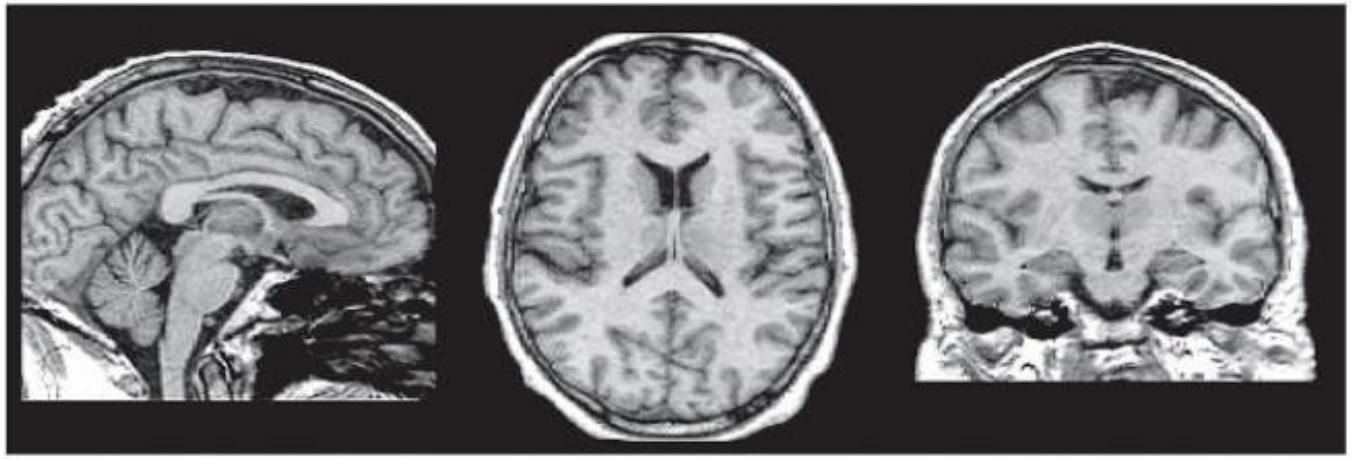
CHRONIC SEQUALAE

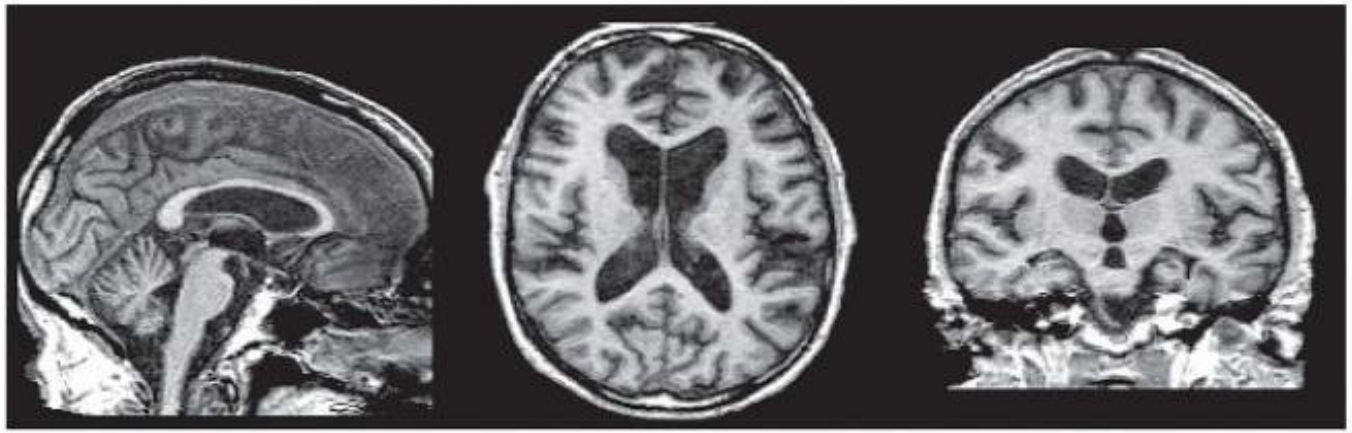
- Once KS develops, neurocognitive deficits are typically irreversible
  - Severe anterograde +/- retrograde amnesia
  - Apathy, poor insight (patients are unaware they have Korsakoff Syndrome)
  - Relative preservation of sensorium, social behavior, attention, long-term memory, other cognitive skills
  - Not all patients have confabulation
- Clinical manifestations stem from anterior thalamic lesions, hippocampal volume loss, mammillary body atrophy

GRADED BRAIN-VOLUME DEFICITS IN WERNICKE-KORSAKOFF SYNDROME

(T1-Weighted MRI scans)



A 63-year-old healthy control male



A 63-year-old man with Wernicke-Korsakoff Syndrome

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LONG-TERM MANAGEMENT AND PROGNOSIS

- Supportive care with focus on quality of life
- Cognitive rehabilitation, memory aids
- Occupational therapy
- Addressing co-morbid psychiatric and medical conditions
- Structure environment at home or in chronic care facility
- Often multidisciplinary approach between psychiatry, neurology, social work, rehabilitation services

- No established pharmacologic treatments beyond thiamine replacement
  - Anecdotal reports of possible attention/memory improvement with acetylcholinesterase inhibitors and memantine, but no controlled studies

Thiamine may prevent further deterioration, but rarely reverses established deficits

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COMMON PITFALLS

- Under-recognition of WE due to reliance on classic triad

Use CAINE criteria instead, and have very low threshold to give thiamine IV/IM empirically

- Using a serum thiamine level to guide risk stratification or diagnostics

A normal serum thiamine level does not exclude risk or presence of WE

- Giving thiamine in acute settings without repleting magnesium

Always check a serum magnesium level and replete, especially if a patient is not responding as expected to IV/IM thiamine

- Using oral or subtherapeutic doses of thiamine repletion in at-risk patients

Give high doses of IV/IM thiamine; there are no known vitamin B1 toxicity risks

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DISCUSSION QUESTIONS

1. What is your experience, if any, working with patients/clients who have Wernicke Korsakoff syndrome?
2. Given the controversial and variable guidance around outpatient oral thiamine for patients with AUD, how should clinicians weigh biologic plausibility, absorption concerns, and harm-reduction principles when deciding what constitutes adequate prevention outside the hospital setting?
3. In your own clinical setting, what most often delays giving IV or IM thiamine when Wernicke encephalopathy is possible, and what would make it easier to give it early?
4. For patients who develop Korsakoff syndrome, what has been most challenging in balancing prognosis, patient insight, and long-term care planning?

THANK YOU!

QUESTIONS? REFLECTIONS?

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