Bitemporal T2 FLAIR Hyperintensities

Presentation, Differential, and Workup

Mark Newcomb

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Summary

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HPI

80 y/o F with a PMH significant for hypothyroidism, atrial fibrillation (on ASA and metoprolol), and hypertension presents with CC of altered mental status and amnesia.

The patient was in her usual state of health and went to church on the morning of presentation. She drove herself to church, sang in the choir, and interacted with friends as usual. However, around 11:30 am she became increasingly confused. She was talking to the minister and realized that she could not remember most of the morning. She was driven to the hospital by a friend.

No recent travel, weight loss, night sweats, weakness, nausea/vomiting/diarrhea, headaches, chest pain/palpitations, or shortness of breath. She does endorse increased anxiety as her husband recently had a stroke, a friend died of cancer, and her daughter just had a baby. Additionally, the patient had been feeling under the weather with "flu-like symptoms" in the two days prior to admission, but on the day of admission felt as though these symptoms had resolved.

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PMH/PSH

- Anxiety
- Hyperlipidemia
- Atrial Fibrillation (not on anticoagulation)
- Impaired glucose tolerance
- Hypothyroidism (subclinical)
- GERD
- Appendectomy (distant)
- Hysterectomy (distant)
- Excision of basal cell carcinoma (distant)

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FH/SH

- Mother - breast cancer (unknown age of onset)
- Father - coronary artery disease (unknown age of onset)
- Aunt (maternal) - breast cancer (unknown age of onset)
- 3 daughters and 3 sons, all healthy
- Previously was a stay at home mom and worked at a daycare. Now retired for many years.
- Occasional alcohol
- Former smoker (quit several decades ago)
- No illicit drug use

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Medications/Allergies

Medications

- Metoprolol Succinate 50mg daily
- Aspirin 81mg daily
- Flovent PRN
- Pro-Air PRN

Allergies

- Trimethoprim/Sulfamethoxazole (rash)
- ciprofloxacin (itching)
- codeine (itching)
- hydrocortisone (rash)
- atorvastatin (myalgias)
- meperidine (hives)
- nabumetone (unknown)
- penicillins (rash)
- naproxen (palpitations)
- prednisone (eye swelling)
- diltiazem (headache, nausea)
- verapamil (headache, nausea)

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Physical Exam

Vitals

T 97.9, HR 70, BP 134/75, RR 16, SpO2 94% on RA

Exam

General: Pleasant elderly woman, appears younger than stated age
HEENT: PEERLA, EOMI, no conjunctival abnormalities, atraumatic, oropharynx clear, no cracked teeth
Cardiovascular: heart regular rate and rhythm
Pulmonary: Lungs CTAB
Abdomen: soft, non-tender, nondistended
Extremities: warm, dry, no lower extremity edema
Neuro: able to recall objects, no recollection of events from 11am to about 2pm. Pt is slightly ambivalent about the memory loss.
MSK: appropriate bulk and tone strength 5/5 b/l upper and lower extremities
Skin: no rash or bruises

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Data

Na 139, K 4.0, Cl 100, CO2 23, BUN 16
Cr 0.87, Glu **122** (H)

WBC 7.96, Hb 13.7, Hct 43.2, Plt 272

Tbili <0.2, DBili <0.2, AST 25
ALT 20, Alk Phos 66
Total Protein 6.7, Albumin 4.6

PTT **36.7** (H), INR 1.0

Ca 9.1
Troponin <0.01
TSH 3.28

UA showing 1+ leukocyte esterase, otherwise WNL

EKG: normal sinus, rate 71, normal axis, normal PR, QRS, and QTc intervals, no ST-T segment changes

CT Head: no CT evidence of acute intracranial abnormality

Lumbar Puncture: clear, 0 RBCs, 2 WBC per uL, 70% lymphocytes Glucose 63 (LP not done at time of blood draw), Total protein 30

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MRI

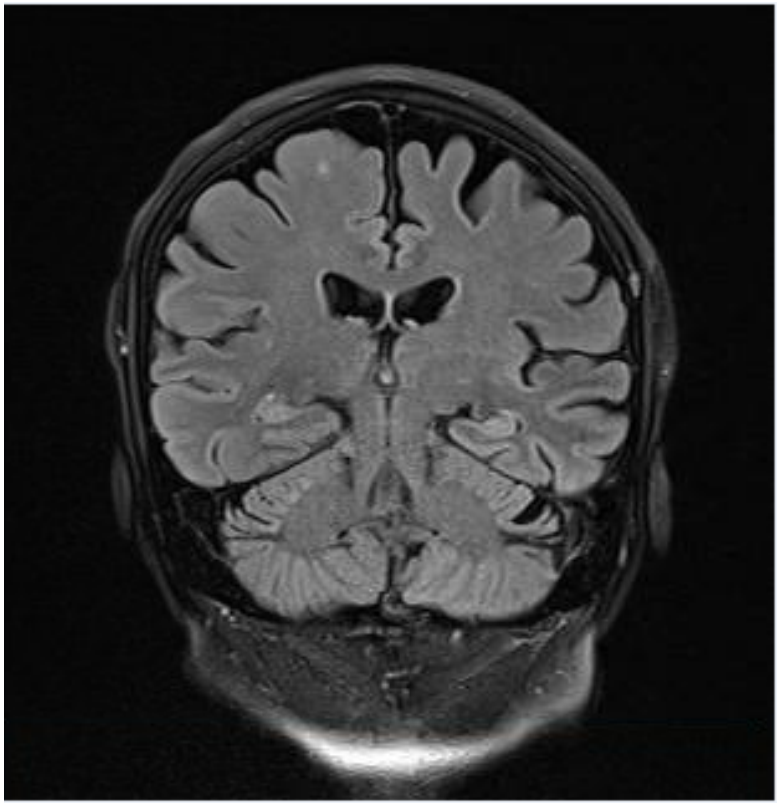

Figure: Mesotemporal FLAIR/T2 Abnormality

MRI Brain

The patient's MRI showed: Mild FLAIR/T2 signal hyperintensity involving hippocampal formations and limbic pathways bilaterally, left greater than right without evidence of qualitative atrophy or enlargement of the temporal horns.

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Temporal Lobe

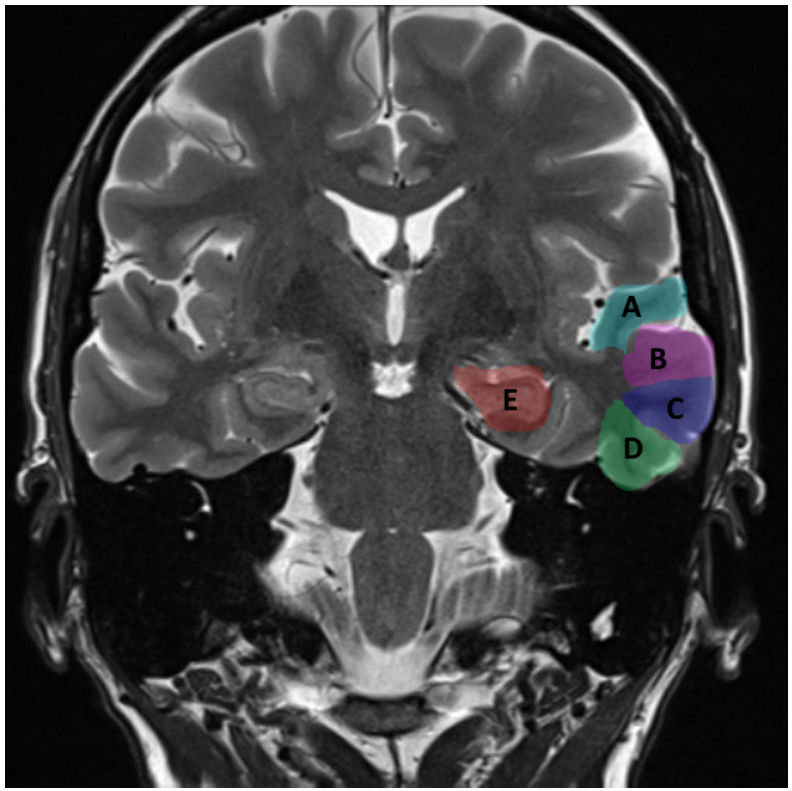

Figure: A=transverse temporal gyrus, B,C,D=superior,middle,inferior gyri, E=parahippocampal and hippocampal gyri

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Factors in Bilateral Temporal Lobe Disease

- connection via anterior and hippocampal commisures and corpus callosum
- optic tracts and radiations
- amygdala and hippocampal sensitivity to hypoxia
- unique immunologic signature within the limbic tract
- dependency on patent basilar artery for bilateral hippocampal blood supply

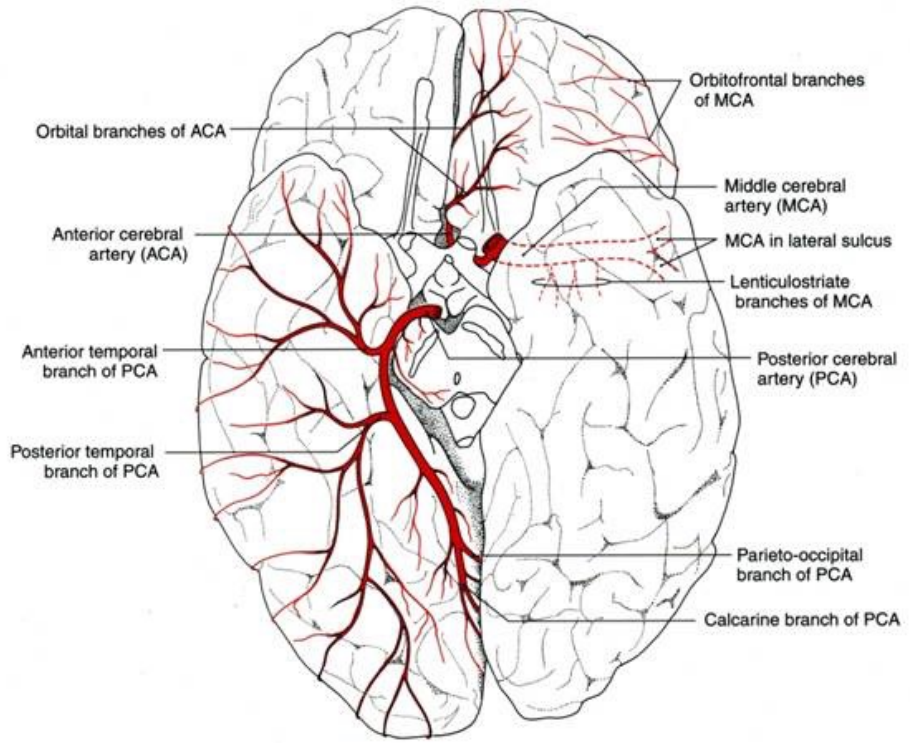

Figure: Hippocampal dependency on PCA brances of basilar artery

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HSV, HHV-6, Bacterial/Fungal Infection

- **HSV** - Usually acute presentation with fever, AMS, headaches. Etiology is reactivation of latent virus in the trigeminal ganglion. MRI often showing T2 signal enhancement in the cortex and white matter of the temporal lobes as well as possible cortical microbleeds on T2 imaging. CT scan may show edema.
- **HHV-6** - typical etiology is reactivation of latent virus approx 3 weeks after an immunocompromising event such as bone marrow transplant. AMS, memory impairment, and seizures are typical. CT will usually be normal. MRI may show T2/FLAIR enhancement, but typically not in the mesial temporal lobes.
- **Bacterial/Fungal** - bilateral otomastoiditis can spread to the bilateral temporal lobes. Other infections such as mucormycosis can spread from the ethmoid/maxillary sinuses, but will typically involve the entire cerebrum and exhibit edema, hemorrhages, and irregular enhancement on MRI.

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HSV, HHV-6, Bacterial/Fungal Infection

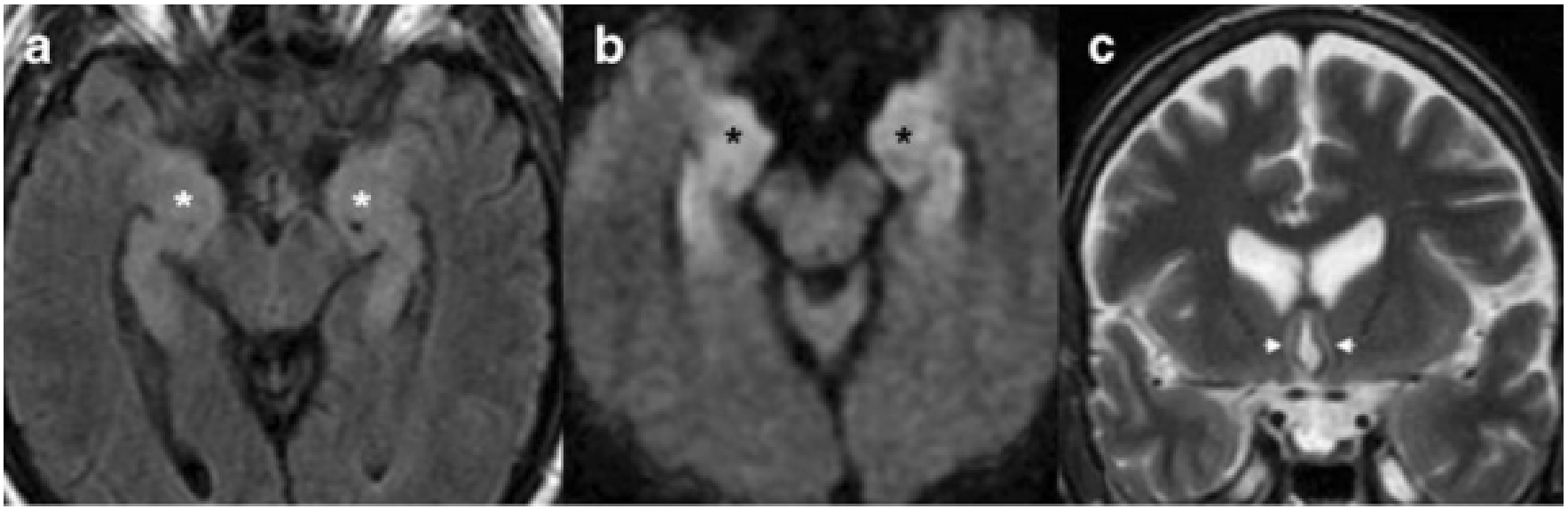

Figure: 66 y/o M with CC confusion, 10 days post BM transplant. MRI showing T2/FLAIR enhancement with restricted diffusion in the bilateral temporal lobes (a,b) and forniceal columns (c).

Eran, Hodes, and Izbudak, 2016

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Limbic Encephalitis

- **Limbic Encephalitis** - A family of autoimmune disorders which may be paraneoplastic or non-paraneoplastic. Presentation is similar in all types, with insidious personality changes, irritability, depression, dementia, and short-term memory loss.
- Anti-neuronal antibodies are found in 60% of these patients - (e.g. Anti-Hu Ab, Anti-Ta Ab, Anti-Ma Ab)
- In the paraneoplastic form, most often associated with small cell lung carcinoma and germ cell tumors.
- Can be seen up to 4 years before diagnosis of a malignancy.
- Non-neoplastic form is diagnosed only after thorough workup to exclude malignancy.

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Dementia

- **Alzheimer's Disease** - Hippocampal formations and entorhinal cortex are thought to be more susceptible to neuritic plaque deposition, thus bilateral volume loss in the mesial temporal structures is a known feature of the disease.
- **Fronto-temporal Dementia** - characterized by preferential atrophy of the frontal and temporal lobes, often asymmetrically.

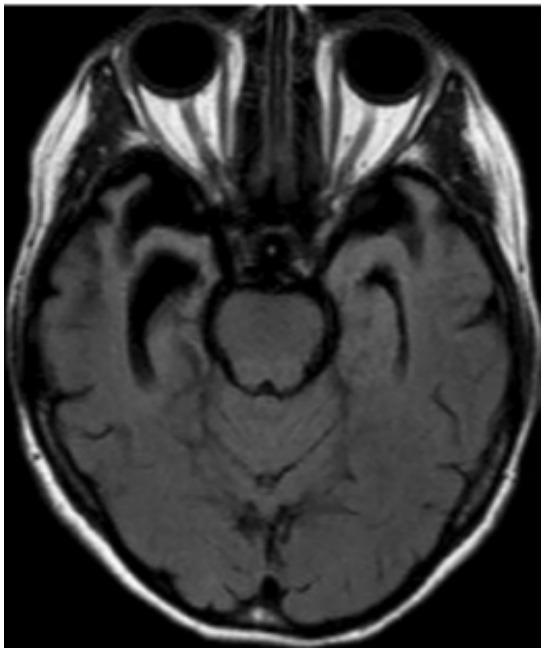

Figure: 63 y/o F with CC 2 yr history of cognitive decline. Imaging showing asymmetric temporal lobe atrophy, right greater than left, consistent with fronto-temporal dementia.

Eran et al., 2016

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Adult-Onset Ornithine Transcarbamylase Deficiency

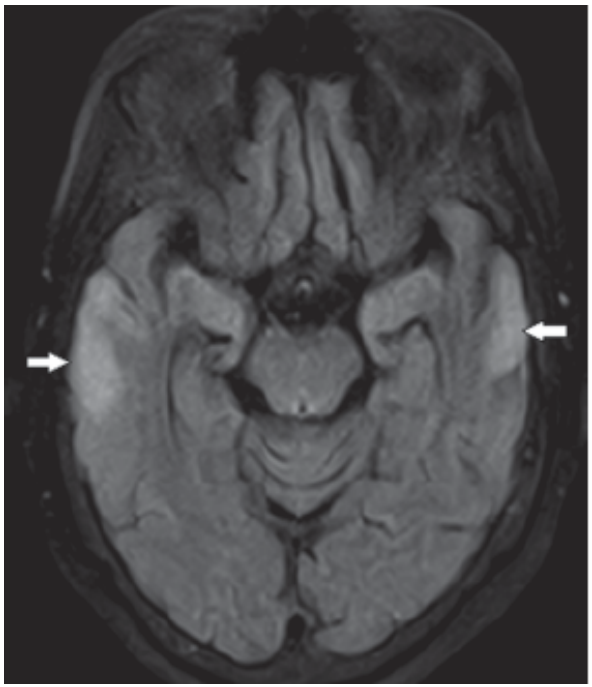

Figure: 61 y/o F with hyperammonemic encephalopathy

Sureka and Jakkani, 2012

- An X-linked disorder which manifests as headaches, dysarthria, ataxia, blurred vision, and acute altered mental status.
- Late-onset variant can present as late as 40-50 years of age.
- MRI characteristically shows swelling, increased T2 signal and diffusion restriction symmetrically in the insular and cingulate cortex.
- Symmetry and lack of hemorrhage help distinguish from HSV encephalitis.

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Sclerosis

Mesial temporal sclerosis is the most frequently implicated structural disorder in partial complex seizures. Up to 10% of cases involve bilateral sclerosis. Limbic encephalitis can also present with seizures but will lack hippocampal atrophy and will show extra-temporal increased signal.

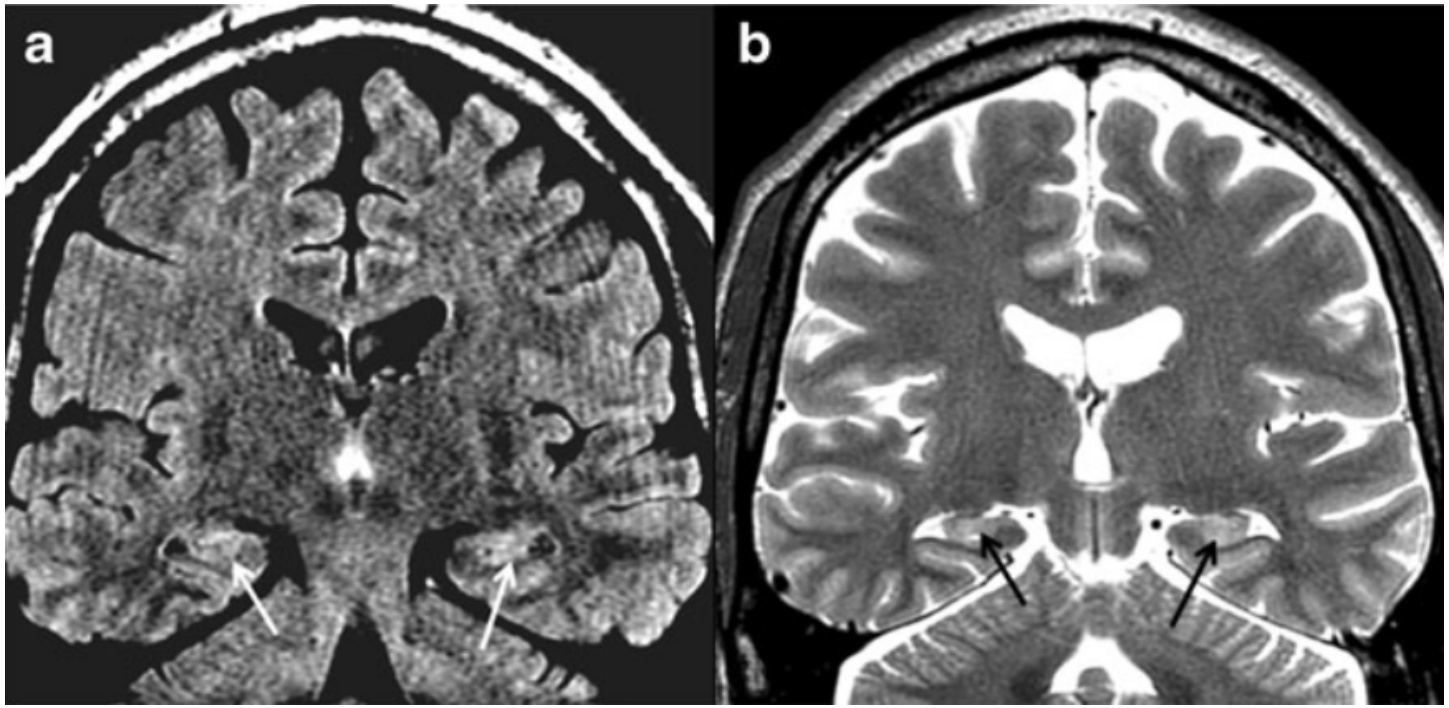

Figure: 27 y/o F with recurrent partial complex seizures. MRI showing bilateral hippopcampal atrophy and increased T2/FLAIR signal indicative of bilateral mesial temporal sclerosis.

Eran et al., 2016

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Glioma

Neoplasms, particularly gliomas, often spread through white matter tracts (anterior/posterior commisures, corpus callosum) and can involve the bilateral temporal lobes. Spread through the **anterior commisure** is the most common path taken in disease isolated to the bilateral temporal lobes. Optic nerve gliomas can also spread bilaterally through the optic radiations or the optic chiasm.

The typical presentation of a neoplasm is insidiously progressive neurologic deficits. Imaging typically shows spread throughout the brain parenchyma (whereas encephalitis is often confined to cortex).

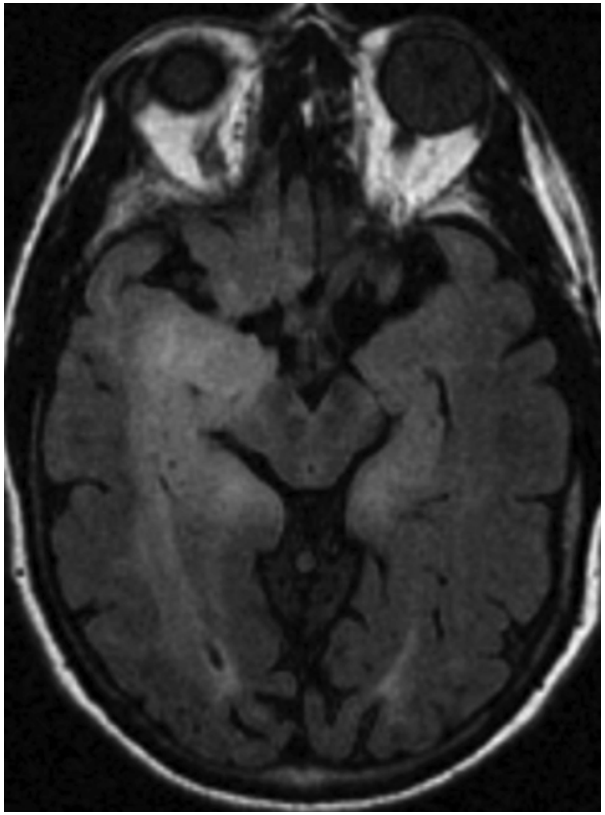

Figure: Gliomatosis Cerebri - extensive T2/FLAIR hyperintensities with minimal mass effect

Zapadka, 2015

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Miscellaneous

- **CVA** - basilar tip and bilateral PCA occlusion can cause bilateral temporal disease and presents with memory/visual deficits as well as altered mental status.
- **Trauma** - sudden deceleration can compress the temporal lobe against the sphenoid wings bilaterally.
- **Radiation** - radiation of head/neck malignancy, most commonly nasopharyngeal carcinoma, can cause bilateral temporal edema and necrosis.
- **Artifact** - pulsation of the carotids or natural relative T2 hyperintensity of the temporal lobes can mimic disease.

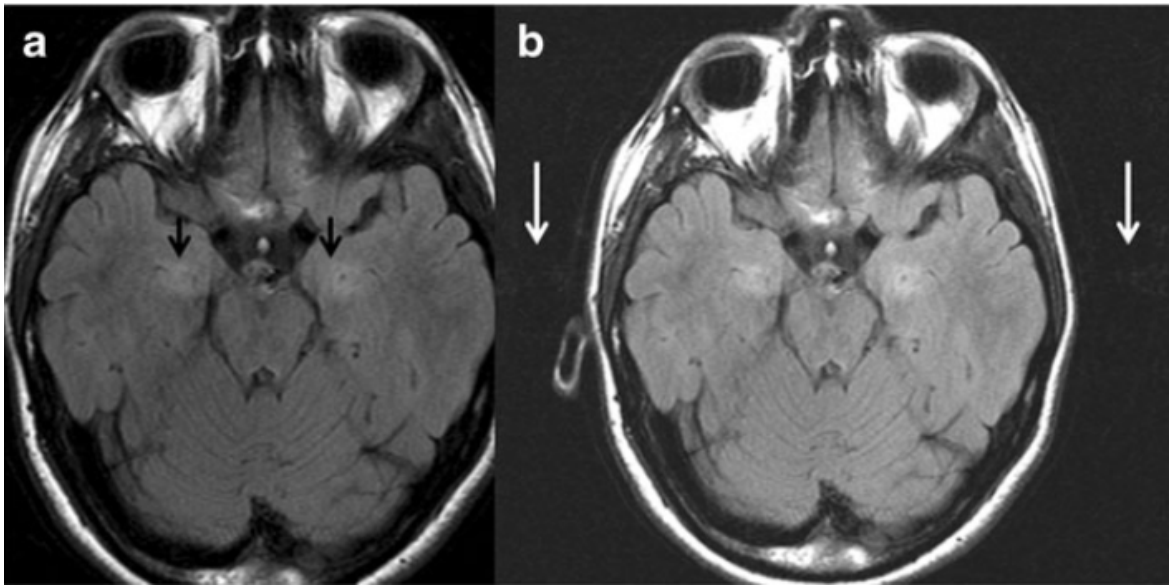

Figure: 25 y/o M with CC headaches, found to have bilateral temporal enhancement. Widened window (right) showed repetitive ghosting indicating a pulsation artifact.

Hospital Course

After admission, the patient felt roughly back to her baseline and began to remember bits and pieces of her amnestic episode.

- HSV PCR of the CSF - **negative**.
- CSF gram stain and bacterial culture - **negative**
- CSF viral cultures - **pending**
- EEG - **normal**

Limbic encephalitis was considered unlikely given the patient's rapid improvement after presentation. After a discussion with the patient, it was decided not to pursue an extensive malignancy workup given that she was up-to-date on her routine age-appropriate preventative screening tests.

The patient's presentation was felt to be consistent with transient global amnesia. She was discharged on Day 3 and has been doing well according to PCP notes (now roughly one week post-discharge).

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Thank You

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Sureka, J. & Jakkani, R. (2012). Clinico-radiological spectrum of bilateral termporal lobe hyperintensity: A retrospective review. **The british journal of radiology**, **85**, 782–792.

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