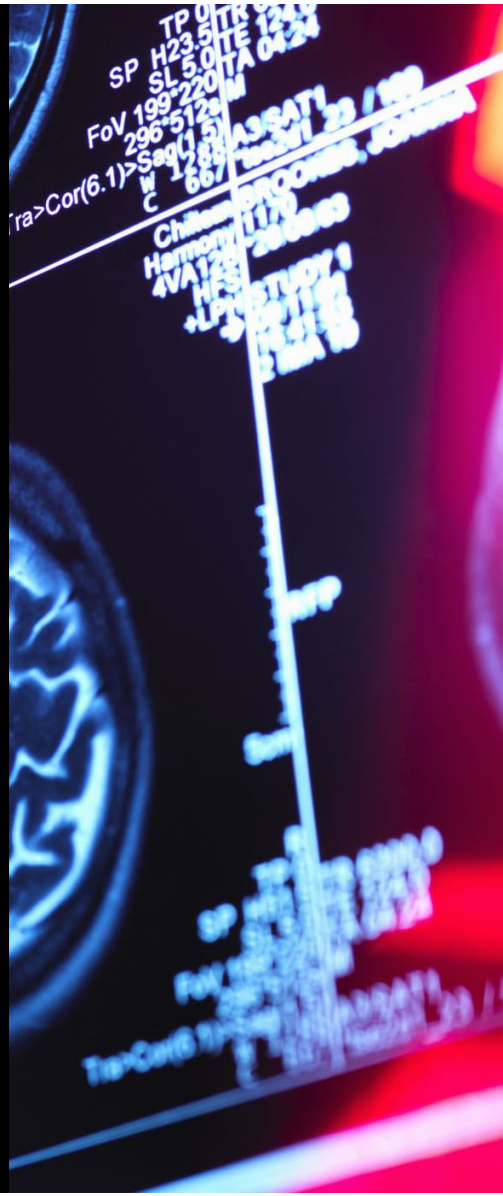
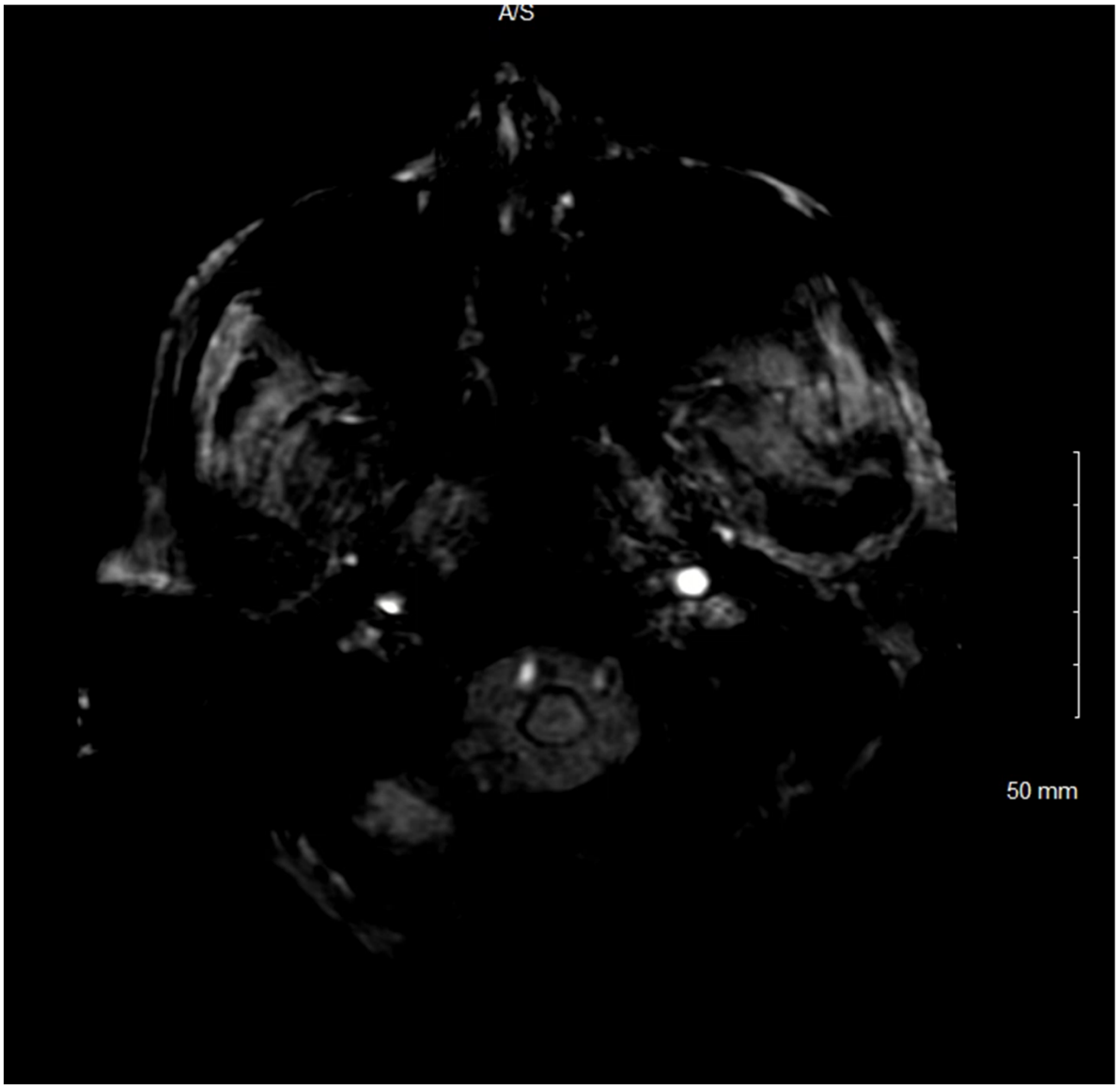
Neuro Imaging





HPI
60s male with a past medical history of subdural hematoma, ESRD on dialysis, and epilepsy, and a gradual cognitive decline over 1 year, admitted for status epilepticus

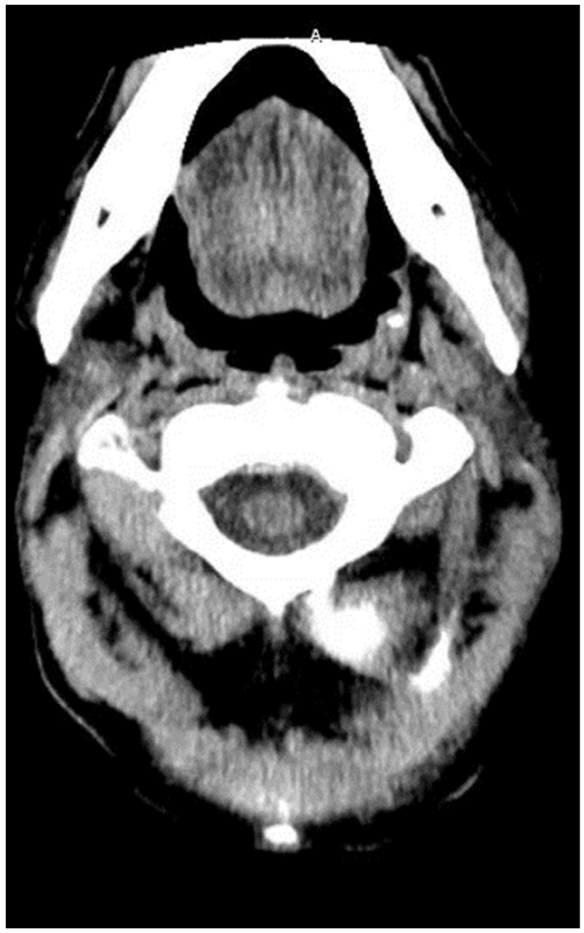
Imaging:
Enhanced susceptibility weighted imaging (eSWAN sequences) were performed. Compounds that have paramagnetic, diamagnetic, and ferromagnetic properties (e.g. deoxyhemoglobin, calcifications) all interact and distort the local magnetic field causing "blooming" artifact. In this eSWAN MRI, there is a rim of low signal on the surface of the brain, indicative of hemosiderin deposition.

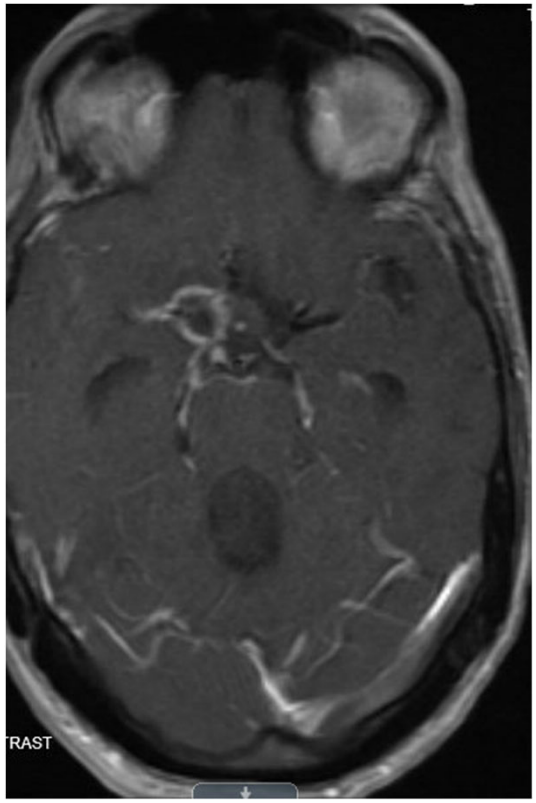
Diagnosis: Superficial siderosis

Discussion:
Superficial siderosis is a rare condition of hemosiderin deposit along the leptomeninges. It is associated with recurrent or extensive subarrachnoid hemorrhage (SAH) and instrumentation. On MRI, it is characterized by a rim of low signal on surface of brain or spinal cord, particularly noted on GRE or SWI.

Clinical presentation is diverse but may include: gradual bilateral sensorineural hearing loss (most common), cerebellar dysfunction, and pyramidal signs. Less common signs include dementia, incontinence, CN dysfunction, sensory deficits. The degree of imaging abnormality does not correlate with the clinical manifestations.

Pathologically, it is thought to result from recurrent occult subarachnoid hemorrhages but the source of bleeding is not usually identified on imaging. Conventional angiography is often unrewarding for source of bleed. The etiologies may additionally include spinal dural defects, intracranial neoplasms, vascular abnormalities, cerebral amyloid angiopathy, or idiopathic (45% cases).





HPI
60s male with a history of strokes, failure to thrive, and malnutrition who presented after a fall with altered mental status.
Lumbar puncture performed with normal opening pressure, glucose 20, protein 60, and a positive culture.

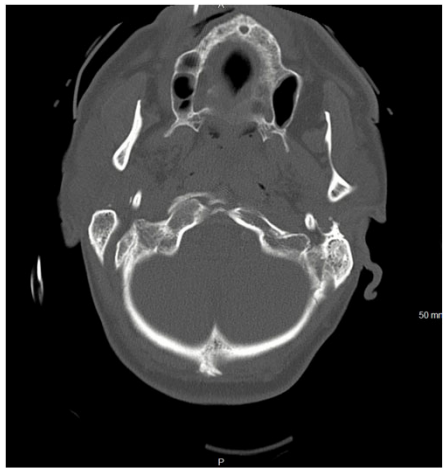
Imaging:
CT Head (left): note prominent fourth ventricle, temporal horn of lateral ventricle, and third ventricle

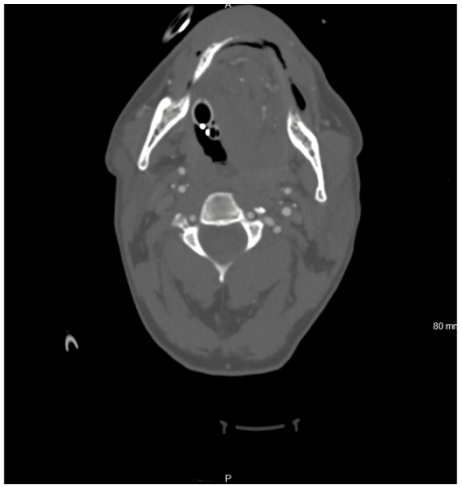
MRI brain with contrast (right):
Note the utility of contrast – areas of inflammation will take up contrast notable for irregular enhancement of the basilar meninges

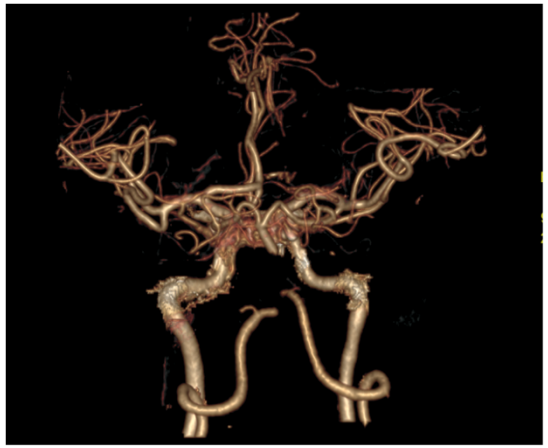
Diagnosis: Cryptococcal meningitis

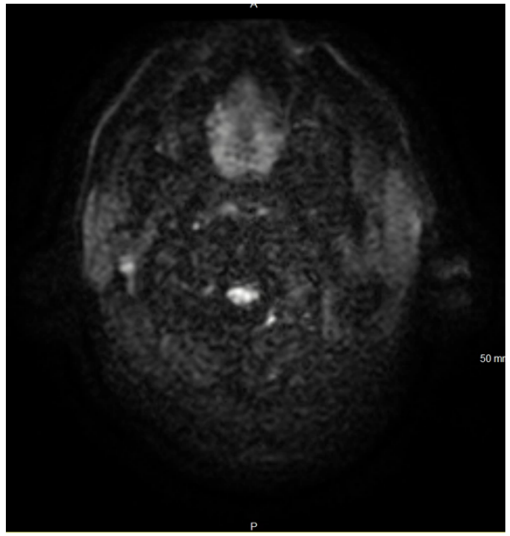
Discussion:
Cerebral cryptococcosis often present with signs and symptoms of meningitis or meningoencephalitis which may include headaches, altered mental status, focal neurological deficits, blurred vision (due to increased ICP), and seizures.

Pulmonary involvement is usually the primary site of cryptococcosis but hematogenous spread to lead to central nervous system involvement, particularly in HIV/AIDS patients when the CD4+ count drops below 100 cells/µL. On gross examination, a grayish mucinous exudate can be seen with meningeal involvement.









HPI
Middle-aged male fell down a flight of stairs, fracturing his clivus

Imaging:
CT bone window (top left): the bone window of the CT head demonstrates a fracture of the clivus

CTA (top right): CT angiography demonstrates a traumatic occlusion of the distal vertebral arteries leading to lack of opacification of the proximal basilar with distal reconstitution proximal to the PCAs

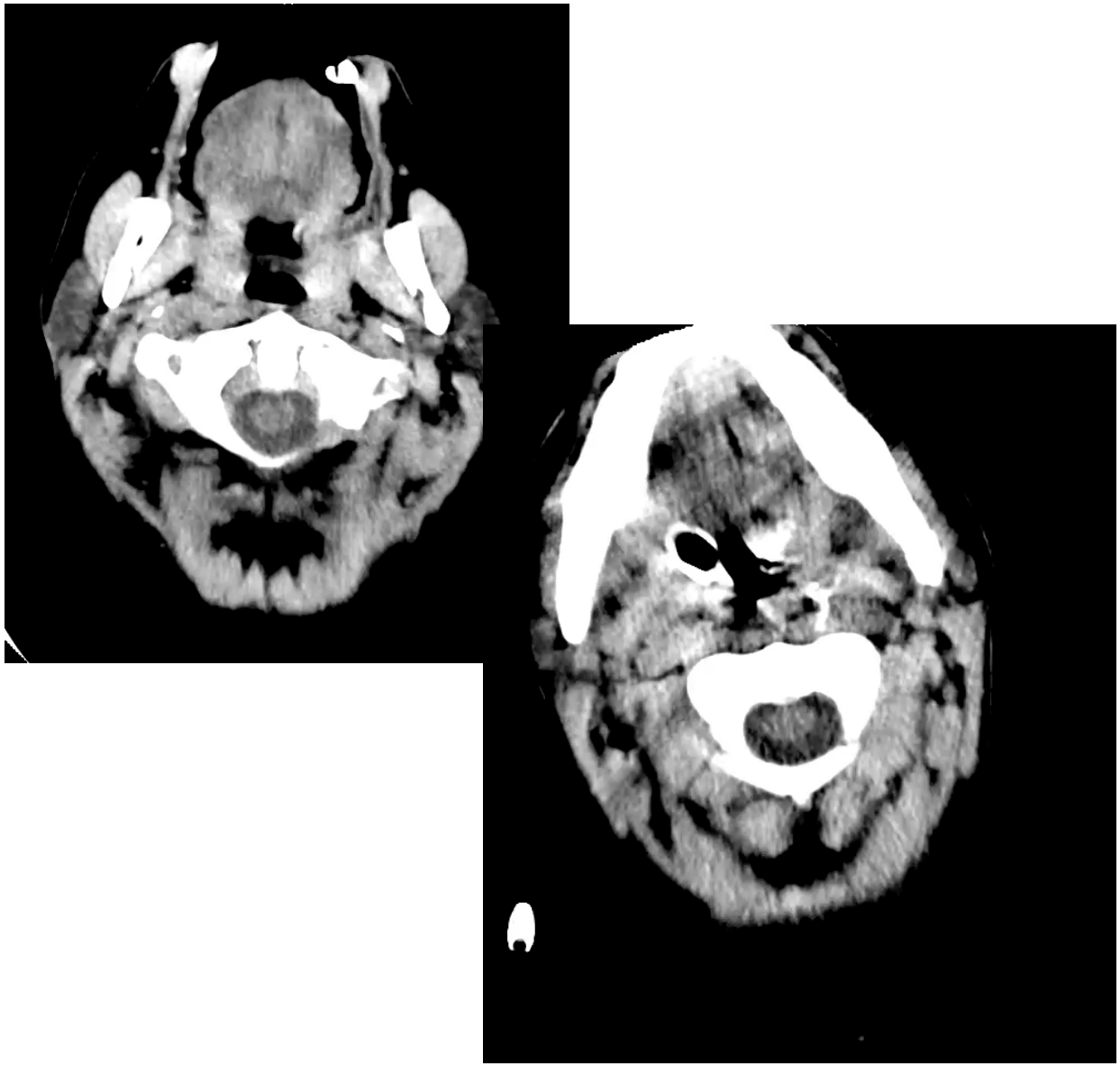
CTA recon (bottom left):the reconstruction of the CTA demonstrates the level of the basilar occlusion

MRI DWI (bottom right):there is diffusion restriction in the right cerebellum, pons, and midbrain

Diagnosis: Locked-in syndrome

Discussion:
Clivus fractures are an uncommon injury but, when they occur, are often due to falls and blunt force trauma. The fractures can be classified into three types: longitudinal, transverse, and oblique. Oblique and transverse fractures can lead to multiple cranial nerve palsies, Horner's syndrome, carotid-cavernous sinus fistulas, and stneosis of the carotid canal. Longitudinal fractures trap the vertebral and basilar arteries causing occlusion that can lead to a locked-in syndrome.

Locked-in syndrome occurs when there is complete paralysis of all voluntary muscles due to damage of the motor pathways that run through the pons-- except those of vertical gaze and blinking as the supranuclear ocular motor pathways run dorsally and are spared. Consciousness and cognition are preserved. Locked-in individuals may be able to communicate by their eye movements.



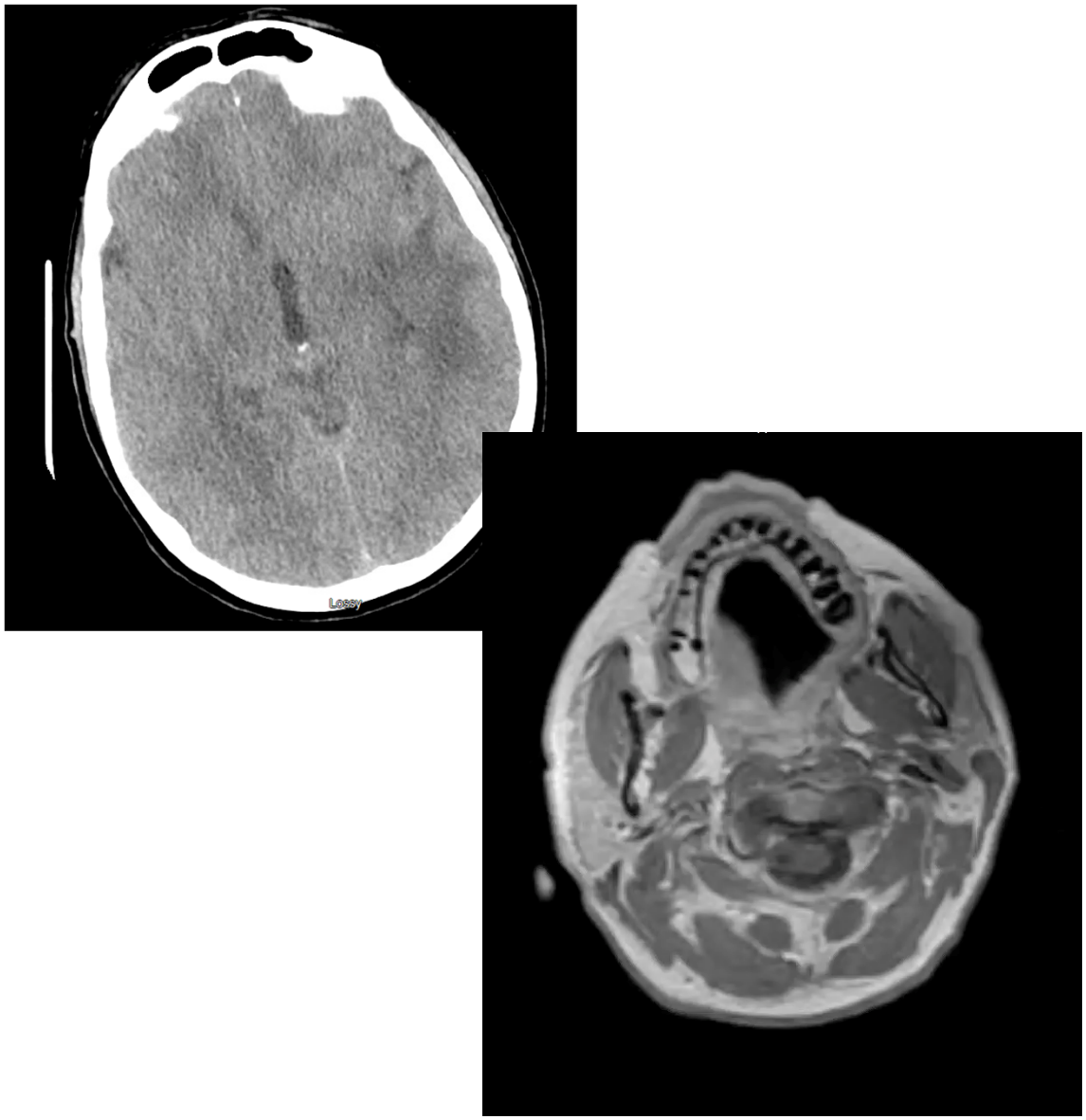
HPI
Young adult presents after a prolonged asthma attack during which she lost consciousness. On arrival, her pCO2 was 250. She was intubated for status asthmaticus and airway protection.

Imaging:
CT #1 (top left): 2 weeks prior to presentation, a normal CT head for age
CT #2 (bottom right): this CT head demonstrates diffuse cerebral edema, crowding of foramen magnum, and sulcal effacement

Diagnosis:
Cerebral edema due to hypercapnia

Discussion:
Hypercapnic respiratory acidosis causes cerebral vasodilation via perivascular extracellular pH changes. While it is rare to precipitate global cerebral edema in the absence of acute brain injury, there are scattered documentation cases of this occurring. Correction of the respiratory acidosis reverses the neurological symptoms and physiology leading to cerebral edema.

In this case, the respiratory acidosis was rapidly corrected and the next day, the patient woke up neurologically intact.



HPI:
50s year old male with a past medical history of IV drug abuse presents with altered mental status, including hallucinations, and headache

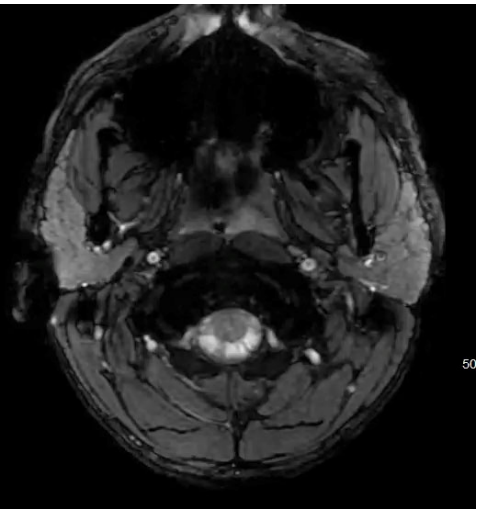
Imaging:
CT Head (top left): Focal lesion left temporal lobe with associated moderate vasogenic edema. Hypodense material in the ventricles with associated hydrocephalus

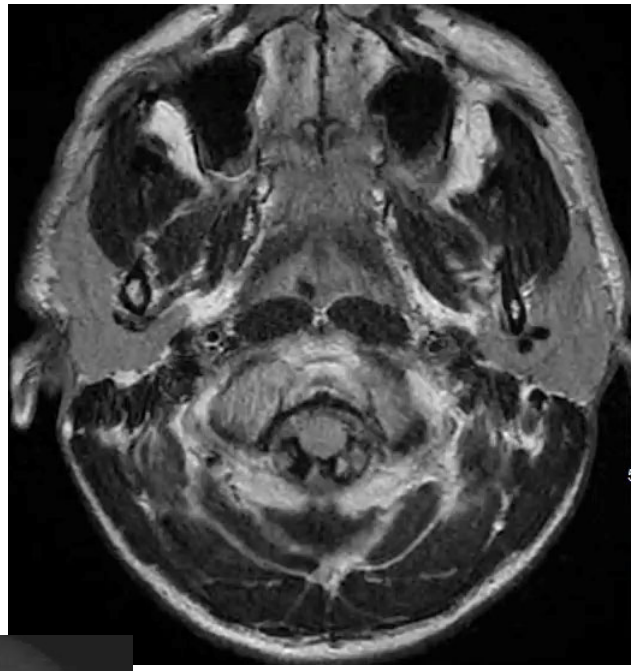
MRI T1 post-contrast (bottom right): left temporal lobe abscess communicating with the temporal horn and an extensive amount of purulent material in the lateral ventricles. Diffuse leptomeningeal enhancement consistent with associated meningitis.

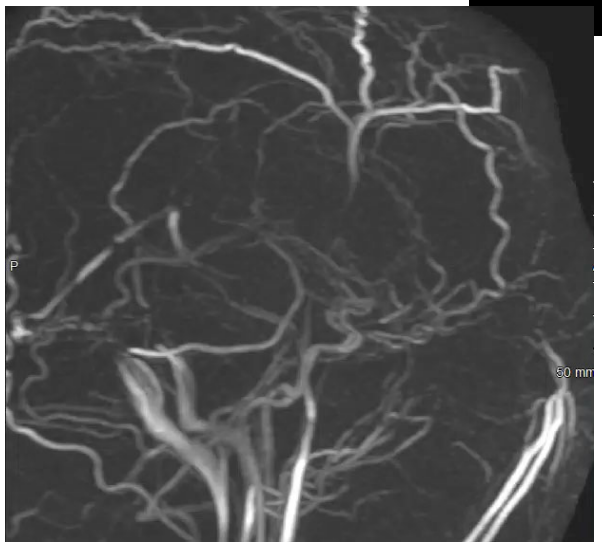
Diagnosis:
Left temporal lobe abscess with rupture into ventricles, ventriculitis

Discussion:
Cerebral abscesses can be causes by bacteria, fungi, or parasites that accumulate within a localized zone of necrosis with a surrounding membrane. An abscess may be caused by direct inoculation – such as a traumatic brain injury with skull fracture—or hematogenous spread from a primary infection elsewhere in the body. The most frequent microbial pathogens are Staphylococcus and Streptococcus but empiric coverage should also consider additional patient risk factors.

Ventriculitis is a rare infection of the ventricular ependyma, often resulting from a ruptured intracranial abscess –as in this case—or iatrogenic from instrumentation. Prognosis is often extremely poor, with mortality rates quoted between 40-80% per literature review.







HPI

Middle-aged male with a past medical history of hypertension and IV drug use presents with altered mental status that gradually worsened over several days

Imaging:

SWI (top left): Multifocal hemorrhage staining and multiple dilated cortical veins

DWI (top right): widespread and bilateral cortical infarctions involving the bilateral frontal, bilateral, and left occipital lobes, but sparing of the deep nuclei

MRA(bottom): widespread dural venous thrombosis involving the superior sagittal, straight, and bilateral transverse sinuses.

Diagnosis:

Bilateral venous thrombosis with infarctions

Discussion:

Venous sinus thromboses are venous blood clots in the major veins of the brain that can occur provoked or unprovoked, similar to venous clots elsewhere in the body. Hypercoagulable states, trauma, or antecedent infections in nearby locations (I.e. sinuses, meningitis) may all be contributing factors.

If there is an antecedent infection with spread into the sinuses – and not just localized inflammation, it may be a septic dural sinus thrombosis. This is an uncommon condition with mortality of 30-78%, varying by which sinuses are involved.

Overall, venous sinus thromboses are uncommon with literature estimates ranging from 13.2 to 15.7 per million patient-years.