

# A Unique Presentation of Cerebritis, A Case Report

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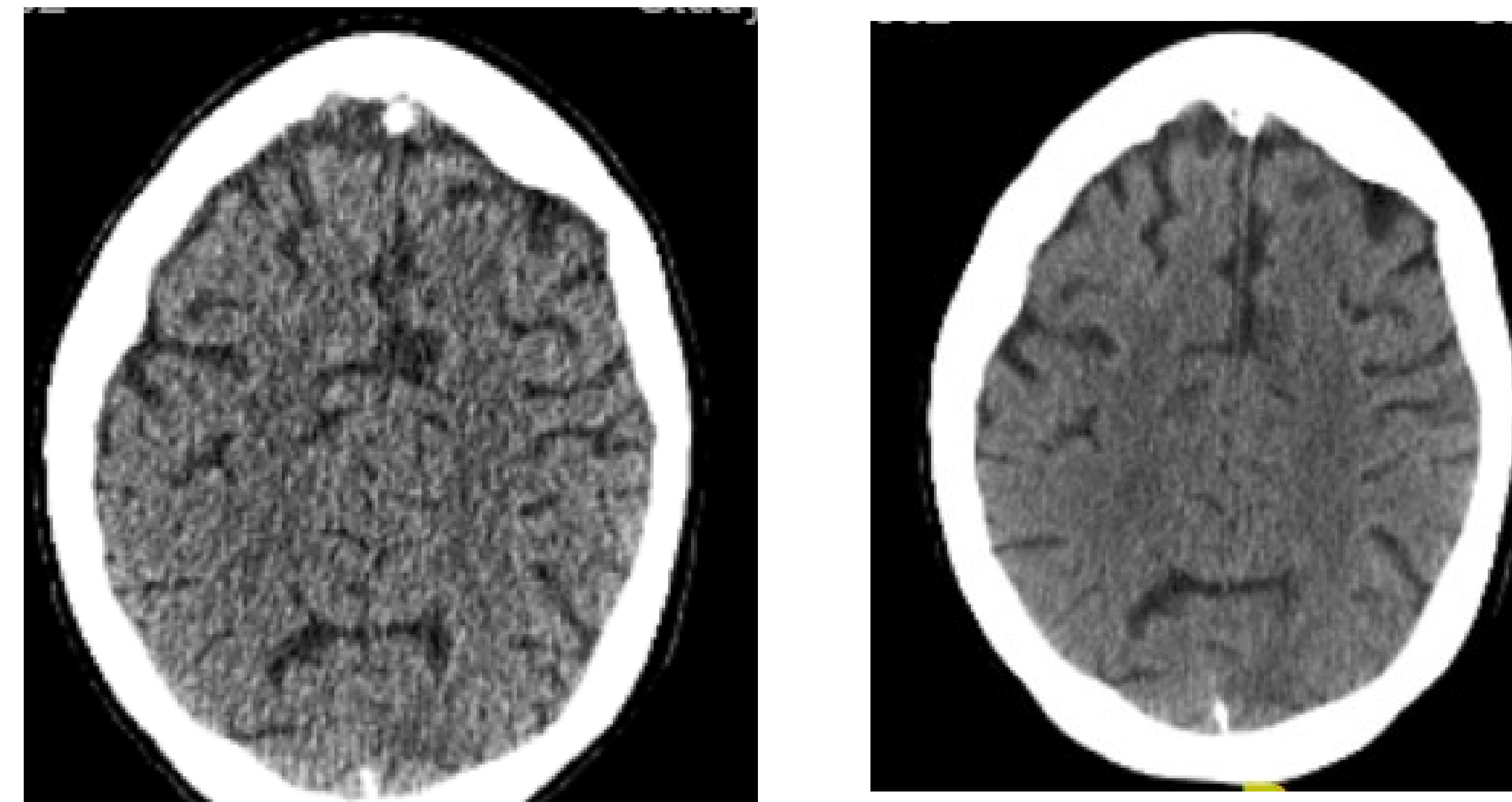
## History of Present Illness

- **Chief Complaint:** altered mental status and sepsis
- 57F presented as a transfer from an outside hospital. The patient has had a subacute mental decline in the prior 2 months endorsing syncopal episodes, weight loss, and weakness. Initial preliminary workup at local hospital was unremarkable but after transfer to a higher care facility, a left renal cell carcinoma was discovered and a nephrectomy was performed. Afterwards, she was discharged back home and reportedly at baseline.
- Days later, she was found by family to be covered in blood (from nosebleeds and biting her lips), vomit, and urine. She returned to the same local hospital where the workup was unremarkable and infectious disease presumed it was bacterial/viral encephalitis. She was started on antibiotics, stable, & sent to a nursing home where her symptoms of confusion and vomiting worsened at which point she was transferred to our facility.
- Other pertinent history: No known medical diagnosis. No known sexual contact or travel history. Has not seen a PCP in 10 years

## Diagnostic Testing

- **On admission:** WBC 14.7 and lactic acid 2.2. She was admitted for sepsis workup. CT head found possible stroke. CTA head and neck showed a vessel irregularity of the distal intracranial vasculature which may be secondary to atherosclerotic disease versus vasculitis. Code stroke was called on this patient.
- **Extensive workup mostly unremarkable:**
  - Blood cultures were negative
  - CSF: 0 WBC, 0 RBC, 49 glucose, 47 total protein
  - HSV1/2 negative, West Nile IgM Ab
  - B12, Folate
  - ANCA, rheumatoid factor, dsDNA
  - **Brain Biopsy**
- **Pertinent results:**
  - ESR 41, ANA positive
  - MRI did not show ischemia. Findings in next section.
  - EEG: moderate to severe diffuse encephalopathy of nonspecific etiology with periodic lateralizing epileptiform discharges over the left temporal area, which may be secondary to underlying structural abnormality, such as stroke and cerebritis
- **Hospital course:** Patient was given empiric antibiotics, antivirals, and steroids. Hospitalization was complicated by psychosis, seizures, and thrombocytopenia. Eventually anti-epileptics were weaned or reduced. Anti-psychotic dosing decreased. Now stable, long term prognosis unclear, but at this time patient has limited capability. On discharge, patient has improving mentation. Able to respond in full sentences appropriately to most questions, though not entirely oriented to time or situation.

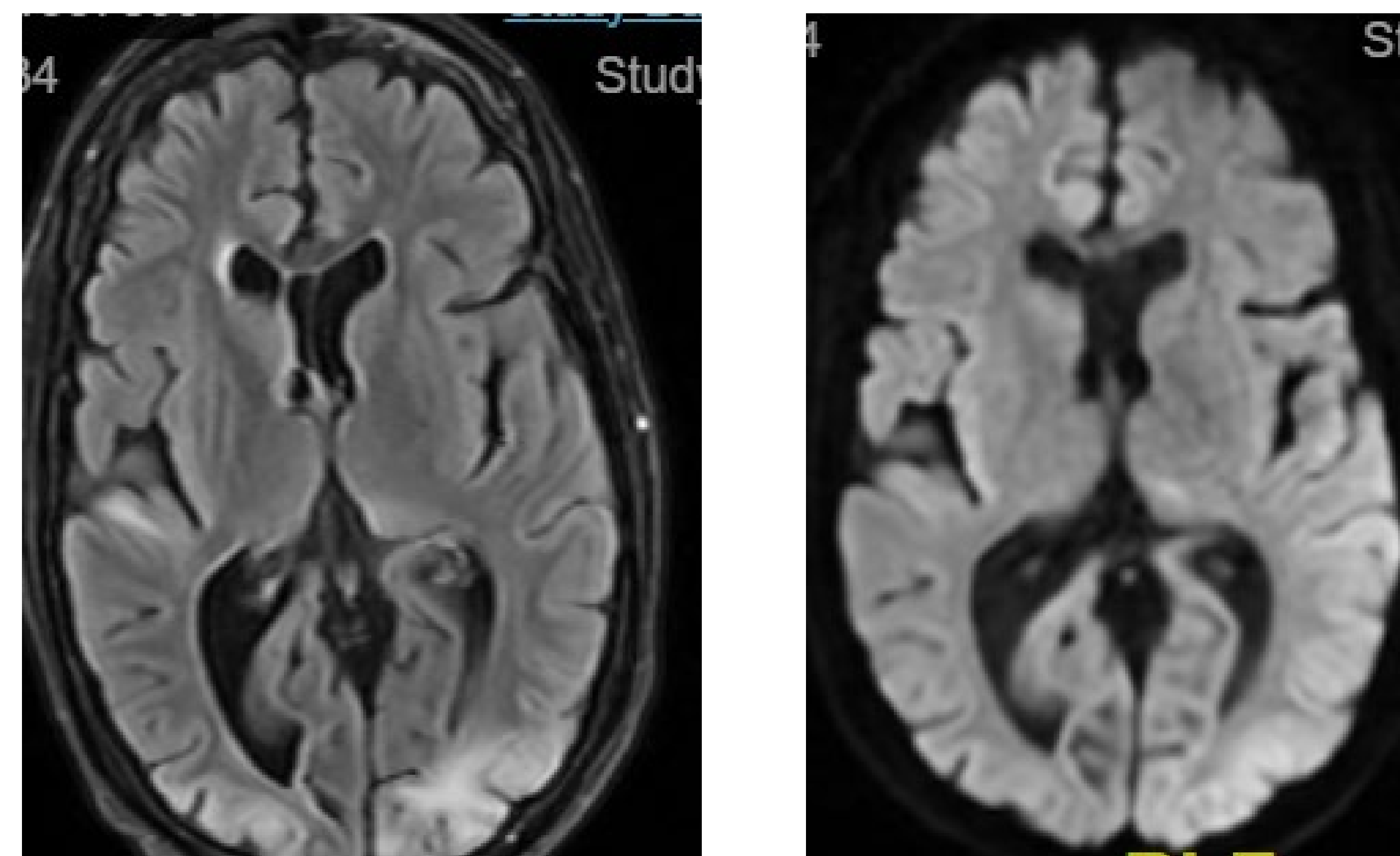
## Imaging



**Images Above:** Initial Head CT showed Indeterminate left occipital lobe infarct. No hemorrhage. Mild chronic microvascular ischemic changes and atrophy.

**Images Below:** MRI of Brain showed no discrete underlying masses in the previously described areas of signal abnormality. Punctate foci of enhancement in bilateral hippocampi are nonspecific, but likely related to vascular blush. There is also minimal focal cortical enhancement in the left occipital lobe. Findings suggestive of infectious etiology of cerebritis.

Diffusion-weighted images show no hyperacute, acute or early subacute infarction.



## Discussion

Acute encephalopathy is a common presentation in the hospital setting. Oftentimes, there are straightforward etiologies (illustrated below). However, in some situations, patients remain encephalopathic despite an unremarkable initial workup. Our particular patient presented with acute encephalopathy and a mixed presentation of symptoms with fluctuating cognition and was not improving despite many different types of management which ultimately led to an extensive workup which yielded diffuse inflammation as a sequelae of viral encephalopathy. Exploring this unique case allowed us to better understand the appropriate diagnostic approach (in particular the value of radiographic imaging) and broaden our differential diagnosis when trying to elicit the etiology of acute encephalopathy.

### AEIOU TIPS - Causes of Altered Mental Status

- A** Alcohol, Acidosis, Ammonia, Arrhythmia
- E** Electrolytes, Endocrine, Epilepsy
- I** Infection
- O** Overdose, Oxygen, Opiates
- U** Uremia
- T** Temperature, Trauma, Thiamine
- I** Insulin (hypoglycemia)
- P** Psychiatric, Poisoning
- S** Stroke, Seizure, Syncope, Space Occupying Lesions, Shunt (VP) Malfunction, SAH



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## Conclusion

The differential for acute encephalopathy is quite broad. This case report illustrated an extensive diagnostic approach to an ultimately idiopathic etiology of acute encephalopathy which yielded a diagnosis through the use of radiological imaging and clinical presentation when other approaches such as lumbar puncture, brain biopsy, and viral panels proved to be inconclusive. Furthermore, this could reveal a potential point of further research into refining imaging techniques as it may hold the key to further elucidating idiopathic presentations of acute encephalopathy.

## References

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