

Posterior Reversible Encephalopathy Syndrome Masked by Hepatic Encephalopathy

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Introduction

- Posterior reversible encephalopathy syndrome (PRES) is a rare clinical and neuroradiologic diagnosis
- PRES is commonly associated with hypertension, autoimmune conditions, or medications
- Symptoms include, but are not limited to, altered mentation, visual disturbances, headaches, and seizures
- Treat supportively by correcting underlying conditions

Case Presentation

A 73-year-old male with hepatitis C and alcohol use disorder presents with altered mental status and visual hallucinations.

Vital Signs / Notable Exam Findings

- Temp 37.5°C, HR 104, BP 172/85, Pox 93% on RA
- AAOx0, agitated, neck stiffness, bilateral clonus, tremors, rest of exam unremarkable

Labs

- CBC and BMP unremarkable
- UDS negative
- Acetaminophen, salicylate, ethanol levels not elevated

AST (IU/L)	ALT (IU/L)	Alk Phos (IU/L)	Total Bilirubin (mg/dL)	Hep C RNA (IU/mL)	Ammonia (umol/L)
100	46	145	1.21	767,000	68

Diagnostic work-up

- Lumbar puncture ruled out meningoencephalitis
- CT head: No acute intracranial abnormalities (Figure A)
- Abdominal US: Cirrhosis of the liver (new diagnosis)

Initial Management

- Clinical Institute Withdrawal Assessment for Alcohol protocol
- Initiated lactulose for hepatic encephalopathy (HE)

Imaging

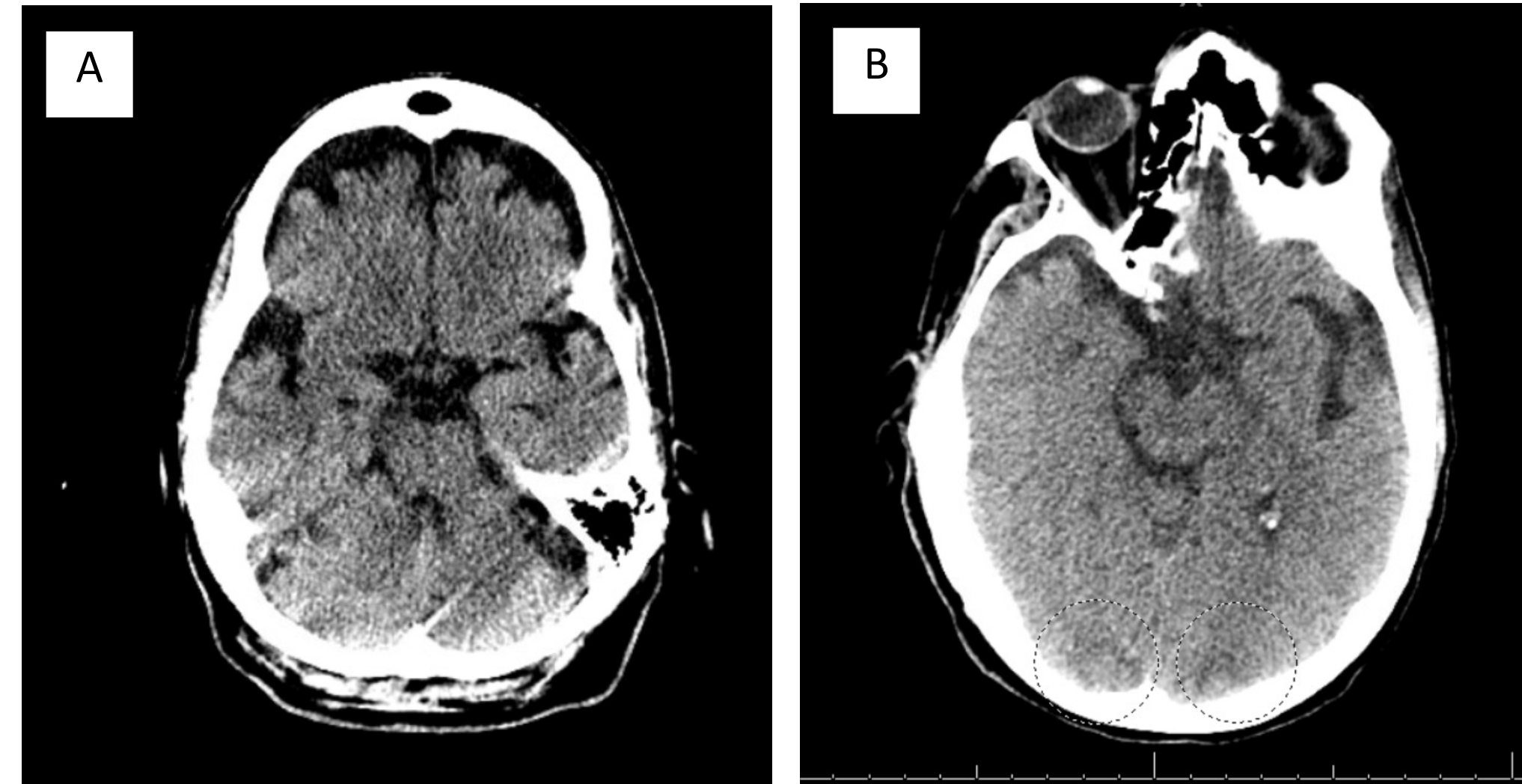


Figure A. Computed tomography scan of the head on day 1 did not show any intracranial abnormalities

Figure B. Computed tomography scan of the head on day 7 showed new bilateral occipital lobe hypodensities (dotted circles on imaging)

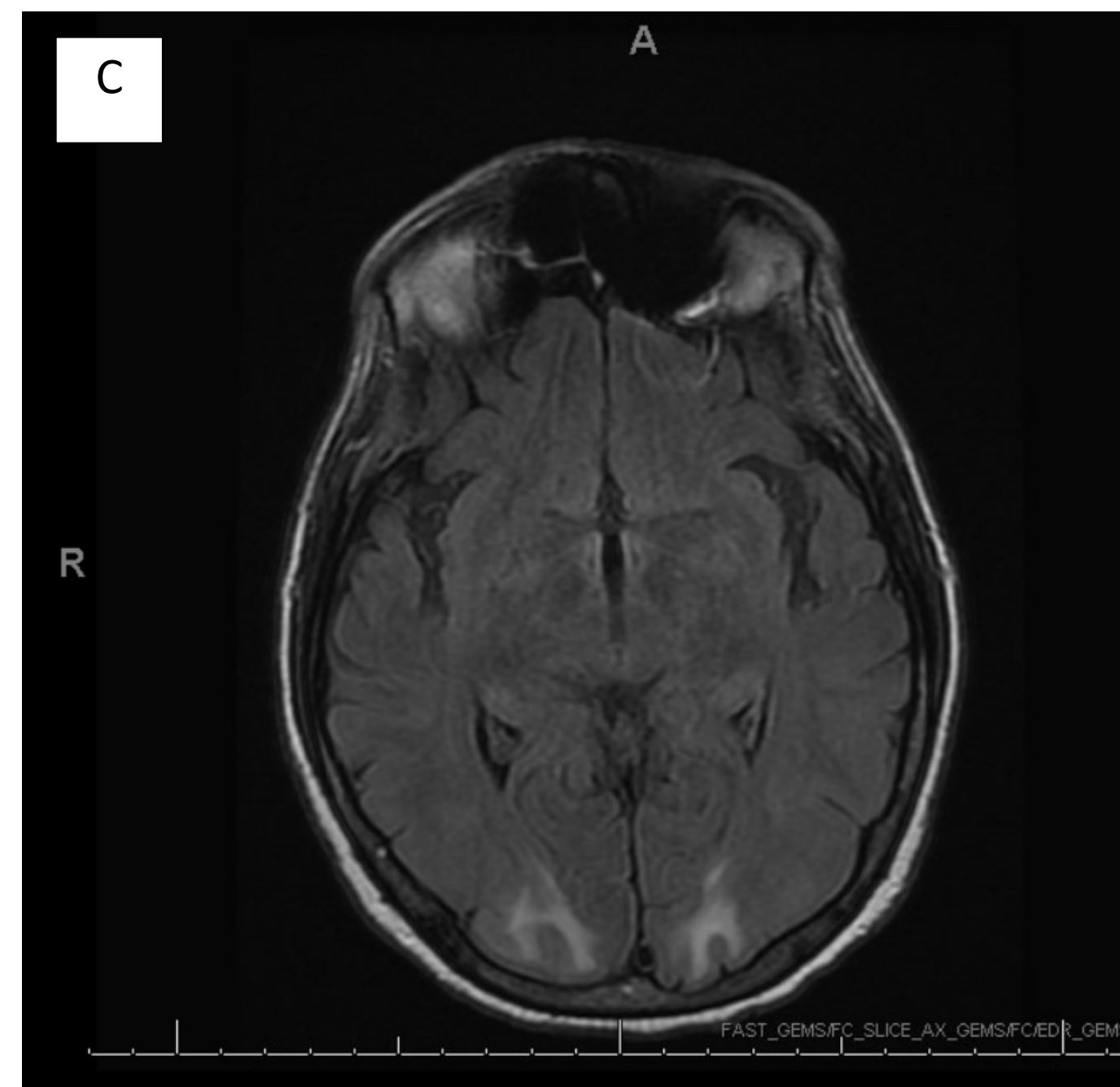


Figure C. Magnetic Resonance T2/Flair imaging showed bilateral occipital subcortical white matter changes consistent with PRES, and without evidence of an acute stroke

Hospital Course

- Repeat CT head was performed on day 7 due to ongoing encephalopathy and concerns for new visual impairment; Results were concerning for PRES (Figure B), which was confirmed on MRI (Figure C)
- Blood pressure trend was reviewed, patient was relatively normotensive except one reading of 172/85 on admission
- Autoimmune work-up sent, results were negative
- Repeat serum ammonia level showed a rise to 99 umol/L, Rifaximin was initiated
- Mental status and vision improved by day 19, patient eventually discharged home with family

Discussion

- A proposed mechanism of PRES is vasogenic edema leading to endothelial injury
- In animal models, elevated ammonia has been shown to cause vasogenic and cytotoxic brain edema through inflammation of cells, increased glutaminase activity, and disruption of the blood-brain barrier
- Serum ammonia is usually elevated in the setting of HE, and may have raised the risk for developing PRES in this patient
- It is interesting to consider whether initiating Rifaximin (reduces ammonia production) helped reduce ammonia levels which may have helped with this patient's recovery

Conclusion

Clinicians should consider PRES and head imaging in patients with ongoing hepatic encephalopathy who do not improve with lactulose therapy.