An Unusual Case of Toxic Leukoencephalopathy
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Background:
• Toxic leukoencephalopathy (TLE) represents a group of CNS white matter disorders with varying presentations that depend on the causative agent.
• The two TLEs that have been identified as having distinct characteristics are "chasing the dragon" heroin inhalation leukoencephalopathy and delayed post-hypoxic leukoencephalopathy (DPHL).
• We describe an unusual case of TLE with features combining both entities.

Case Presentation:
• A 48 year old female with history of polysubstance abuse (alcohol, opiates and benzodiazepines) who presented with worsening mentation and bizarre behavior. Her history was positive for recent Morphine and Oxycodone ingestion.
• Initial exam was notable for disorientation, apraxia, aphasias, patellar hyperreflexia, and wide based gait. She was treated initially with IV corticosteroids with no improvement.
• Her symptoms progressed and she developed significant rigidity, fever and hemodynamic instability. She was intubated, treated with Lorazepam, Acetaminophen and cooling blankets. She continued to decline and eventually became comatose.

Results:
• CT (Head) showed no acute intracranial abnormalities.
• EEG showed intermittent, generalized, reactive polymorphic delta slowing.
• MRI (Brain) revealed confluent supra-tentorial white matter signal changes with mild diffuse cortical swelling suggestive of diffuse encephalitis. Repeat MRI brain showed similar findings.
• CSF studies and cerebral angiograms were normal.
• Brain biopsy was consistent with spongiform leukoencephalopathy.

Conclusion:
➢ Here we report a case of spongiform leukoencephalopathy that has clinical, radiological and pathological features of both DPHL and heroin-induced TLE.
➢ As with DPHL, her clinical course was delayed and her MRI findings were more periventricular, sparing the U fibers and cerebellum. However, the brain biopsy findings of spongiform vacuolization without demyelination or axonal involvement were more suggestive of heroin-induced TLE.
➢ Her symptoms were unusual and combined both cortical and cerebellar deficits. We believe this may suggest an overlap between both entities.

References: