Oxycodone abuse: An atypical cause of limbic encephalopathy

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Background

This report highlights a case of limbic encephalopathy with acute amnesia in a patient with suspected oral opioid abuse. Several cases of patients with amnestic syndromes due to synthetic opioid abuse, particularly fentanyl, have been documented. There has not been a known case reported with isolated oral oxycodone use. The proposed mechanism is thought to be due to toxic-metabolic injury to limbic structures with high density mu-opioid receptors.

Case

Patient: 60-year-old Caucasian transgender female
Past medical history: bariatric surgery 2015, gender reassignment surgery 2002
Past psychiatric history: unspecified mood disorder on multiple psychotropics
Medications: Oxycodone-Acetaminophen, Paliperidone, Cariprazine, Lamotrigine, Citalopram, Venlafaxine, Bupropion, Clonazepam, Methylphenidate, Diltiazem, Spironolactone, Omeprazole, Estradiol

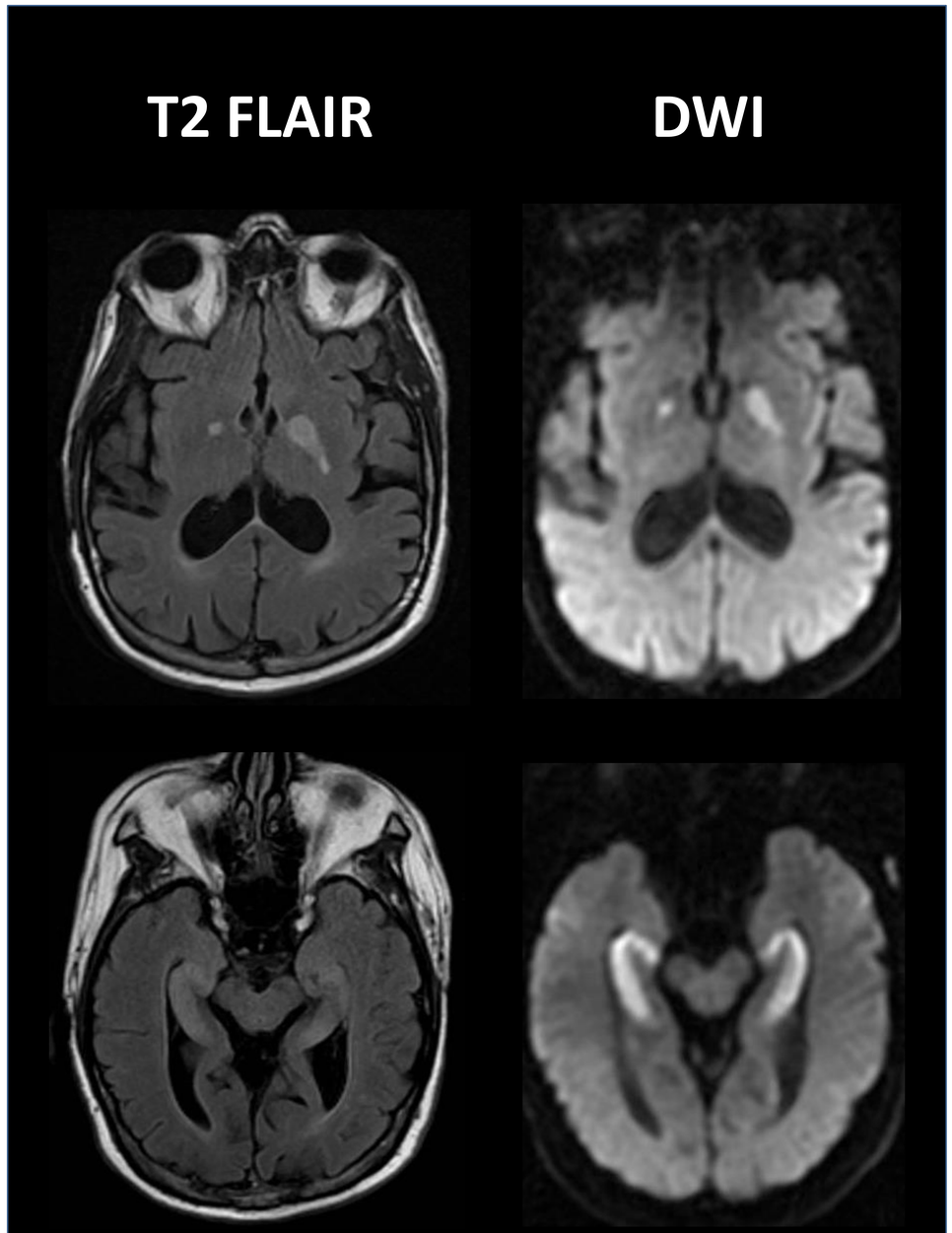
HPI

Patient was found unresponsive at home. Spouse reported seeing patient consume alcohol at an unspecified time prior and noticed multiple missing oxycodone pills.
At baseline, patient is alert and oriented, and she is the sole caretaker of her spouse with medical disability.
On daily visits in the hospital, she displayed severe anterograde amnesia and significant deficits in orientation, attention, and recall without clinical improvement.
Medication washout was done.
Patient was discharged requiring 24-hour care.

MSE

Behavior: Psychomotor retardation, withdrawn
Mood: Depressed; Affect: Flat
Speech: Slow, latency
Thought process: Blocked; Thought content: Poverty of content
Orientation: Person and place only
Memory: Impaired recent, anterograde amnesia
MOCA 15/30
Deficits in orientation, registration, and recall.

MRI brain with and without contrast



Axial T2 Fluid attenuation inversion recovery (T2 FLAIR) imaging on the left and axial diffusion weighted imaging (DWI) on the right demonstrating increased signal intensity and restricted diffusion in the bilateral globus pallidus and parahippocampal gyri.

Case (continued)

Imaging

EEG: generalized slowing consistent with nonspecific cerebral dysfunction without epileptiform discharges
CT chest, abdomen, pelvis: no findings consistent with neoplasm

Labs

CBC, CMP, TSH, UA: within normal limits
Respiratory panel, including COVID: negative
Antibody panel, including HSV: negative
Vitamin panel: within normal limits
Utox: pos. for opiates, benzodiazepines, cannabis; neg. for fentanyl
BAL: <10
LP: no findings consistent with infectious, autoimmune, or metabolic etiologies

Discussion

- It is hypothesized that patient's globus pallidus pathology was due to hypoxic injury, as patient was unresponsive for unknown amount of time; while the limbic encephalopathy was secondary to chronic oxycodone use/abuse.
- Isolated hippocampal/parahippocampal injury is not specific for a particular etiology. Limbic encephalopathy can present with a wide range of clinical characteristics, particularly neuropsychiatric symptoms related to memory, emotions, and behavior.
- Most commonly, MRI findings involve cortical thickening and increased T2/FLAIR signal intensity of bilateral regions of the limbic system. There are a variety of causes for these findings, including infectious, autoimmune, and paraneoplastic. There are also known cases tied to synthetic opioids.
- It is important to consider other opioids beyond synthetics as causes for limbic pathology with acute amnestic syndrome, as this area has a high density of opioid receptors. Additionally, longitudinal studies should be considered for surveillance of cognitive improvement and potential therapies for this syndrome.

References

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