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“Street Brownie” Chronicles: A Case Report Demonstrating the Impact of Alpha Two Antagonist in Combination with Fentanyl on Clinical Severity

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Abstract

Background: The evolving panel of illicit drug combinations is a dynamic public health concern with the latest trend to adulterate illicit fentanyl with alpha two agonists being an area of heightened attention. Xylazine and clonidine are arising as new additives. The combination of these drugs has an atypical presentation and is a medical emergency. The following case highlights the presentation, diagnosis, and management of two individuals presenting with similar symptomology with markedly different escalation in clinical severity after ingesting the same “street brownie”.

Case Presentation:

A 43-year-old woman with a past medical history of chronic kidney disease stage 3, Graves’ disease status post radioactive iodine ablation complicated by iatrogenic hypothyroidism, thyroid eye disease, polysubstance use disorder, anxiety, and depression presents to the Emergency Department with recurrent falls and profound weakness. Her partner, a 52-year-old male with a past medical history significant for migraines, GERD, anxiety, opioid use disorder, nicotine dependance, and chronic low back pain status post thoracolumbar spinal fusion joins her in presentation with the same chief complaints. Both endorse symptoms began soon after ingestion of a “street brownie”. On investigation, urine drug screen was positive for fentanyl and benzodiazepines.

Though presenting with similar symptoms, their clinical courses quickly diverged. The male was admitted to the general medicine floor, provided supportive care, and clinically improved. He was discharged on admission day two. While the female counterpart began to clinically decline soon after admission requiring higher level care in the intensive care unit. She was hemodynamically unstable necessitating pressor support to maintain adequate blood pressure. There was significant bradycardia with heart rates as low as 37 requiring prn atropine and dopamine infusion. Cardiology was consulted and recommended dopamine titration and electrophysiology evaluation.

Labs were ordered to rule out hypothyroidism as the confounding factor leading to the drastic differences in clinical course. Results included a TSH of 49.27 µIU/mL and a free T4 of 0.7 ng/dL serving to rule out this hypothesis. Given known ingestion of adulterants, Poison Control was contacted and recommended comprehensive send-out testing. Serum LC-QTOF/MS confirmed fentanyl/norfentanyl in both patients. However, the identification of clonidine was limited to the female sample.

Conclusion: The difference in severity of illness in this case illustrates the life-threatening effect of fentanyl and clonidine on the cardiovascular system. Independently, each drug causes bradycardia through distinctly different biochemical pathways. When used together, these discrete mechanisms of action compliment to create an additive effect of life-threatening proportion. Initial symptoms will typically present within thirty minutes to two hours post ingestion. Clinical presentation will likely include severe bradycardia, hypotension, CNS depression, and respiratory distress. This cardiovascular toxicologic emergency is difficult to reverse and requires a higher level of supportive care. Given this emerging public health concern, when interrogating potential sources of atypical combined toxidrome, it is important for clinicians to consider a comprehensive toxicology and a broader list of differentials.