Arch Intern Med Res 2022; 5 (2): 199-202 *DOI: 10.26502/aimr.0102*

Case Report

A Fatal Case of Thyroid Storm Precipitated by Methamphetamine Use

Matthew Nunley[1], Adrian Renaldi[1], Ross Turner[2], Mark Bannon[1], Adrita Ashraf[1], Creticus Marak[3*], Prashant Kaushik[4]

[1]Department of Internal Medicine, House Staff, Northeastern Health System, Oklahoma, USA

[2]Department of Internal Medicine, Attending Physician, Northeastern Health System, Oklahoma, USA

[3]Department of Medicine, Division of Pulmonary and Critical Care Medicine, Northeastern Health System, Oklahoma, USA

[4]Department of Medicine, Division of Rheumatology, Northeastern Health System, Oklahoma, USA

[*]Corresponding author: Creticus Marak, Department of Medicine, Division of Pulmonary and Critical Care medicine, Northeastern Health System, Tahlequah, Oklahoma, 74464, USA

Received: 16 May 2022; **Accepted:** 24 May 2022; **Published:** 30 May 2022

Citation: Matthew Nunley, Adrian Renaldi, Ross Turner, Mark Bannon, Adrita Ashraf, Creticus Marak, Prashant Kaushik. A Fatal Case of Thyroid Storm Precipitated by Methamphetamine Use. Archives of Internal Medicine Research 5 (2022): 199-202.

Abstract

A thyroid storm is an acute, life-threatening complication in patients with inadequately controlled hyperthyroidism. Fortunately, this condition is rare and occurs mainly in patients with poorly controlled hyperthyroidism with additional risk factors. Commonly cited risk factors include abrupt discontinuation of antithyroid medicines, emergent thyroid and non-thyroid surgeries, trauma, infections, medications (amiodarone, salicylates), parturition, and use of iodinated contrast medium. There have also been reports of methamphetamine use precipitating a thyroid storm, albeit only a few. We are reporting a case of thyroid storm in a patient with untreated hyperthyroidism that we believe was precipitated by recent methamphetamine use.

Keywords: Thyroid Storm; Sympathetic Toxidrome; Methamphetamine; Hyperadrenergic; Risk Factors

1. Introduction

Manifestations of thyroid storm or thyroid crisis are due to a hyper-catecholaminergic state, likely caused by the synergistic effects of thyroid hormones and endogenous catecholamines. Even though this is a well-described entity, making the correct diagnosis can sometimes be challenging, even for astute clinicians, due to overlapping clinical features. The clinical presentation of thyroid crisis is similar to the toxidrome caused by sympathomimetic agents. Various illicit drugs, (amphetamine, pseudoephedrine, and cocaine), in addition to conditions that increase the body's adrenergic response, (sepsis, trauma, burns, surgery, CVA, ACS, and DKA), have been known to precipitate thyroid crisis. A high index of clinical suspicion is needed to timely diagnose and treat thyroid storm in patients who abuse sympathomimetic agents that demonstrate similar overlapping clinical features.

2. Case Report

Our patient was a 43-year-old male, a polysubstance abuser (nicotine, marijuana, and Methamphetamine) with hypertension, untreated hyperthyroidism, and renal carcinoma, post radical nephrectomy. He initially presented to an outside facility for evaluation of dyspnea that started on the day of hospitalization. His initial BP was 152/124, HR was 160/min, RR was 20/min, Spo2 was 100% on RA, and Temp was 36.7 C. Physical examination revealed a somnolent patient who was tachycardic and tachypneic and had grade 3 bilateral exophthalmos. The remainder of the physical examination, including the heart and the lungs, were unremarkable. His initial blood workup revealed WBC 8.1 K, Hb 14 gm/dl, Platelets 189 K, Na 135 mEq/L, Bun 16 mg/dL, Cr 0.7 mg/dL, Ca 8.2 mg/dL, BG 169 mg/dL, ALP 146 IU/L, AST 32 IU/L, ALT 26 IU/L, TB of 1.3 mg/dL, BNP of 1800 pg/ml (normal < 100pg /ml), Troponin I of 0.08 ng/ml, FT4 3.22 (reference range: 0.9 to 2.3 ng/dl), TSH < 0.016 (reference range: 0.5 to 5.0 µIU/mL) and TSI 486% (normal < 140%).

The urine drug screen was positive for tetrahydrocannabinol, benzodiazepines, and Methamphetamine. Chest X-ray revealed cardiomegaly with venous congestion consistent with CHF, and his EKG showed an atrial flutter with RVR. He was diagnosed with atrial flutter with RVR and acute CHF, started on IV Cardizem for rate control and transferred to our hospital for further evaluation and management. He was admitted to the medical ICU for close monitoring. Stat echocardiogram revealed a dilated cardiomyopathy with an EF of 25%, moderate mitral regurgitation, biatrial enlargement, right ventricular enlargement with a PASP of 45mm Hg, and RAP of 15 mmHg; consistent with biventricular failure. While in the ICU, he had intermittent episodes of agitation requiring IV lorazepam to control suspected methamphetamine-induced behavioral disturbances. Stat CT of the head showed no acute findings. His Burch Wartofsky score was 60, highly suggestive of thyroid storm. He was started on IV Hydrocortisone, IV Esmolol (IV Cardizem was discontinued), PO Methimazole, PO Supersaturated Potassium Iodide, and PO Cholestyramine. Within a few hours of his arrival in the ICU, he developed apneic episodes and severe hypoxemia, with oxygen saturations dipping into the 70s, requiring emergent tracheal intubation for mechanical ventilation. Shortly after the intubation, he became bradycardic and developed PEA, requiring an ACLS protocol, with which he regained ROSC after two rounds of compressions. Post resuscitation, he developed refra-

ctory shock with multiple organ failure. Despite aggressive medical treatment, his condition deteriorated, and after acknowledging his overall grim prognosis, his family requested to place him on comfort measures. He expired on the fifth day of hospitalization.

3. Discussion

Illicit Methamphetamine (MA) drug use in the United States remains a major public health concern. Methamphetamine use has been on the rise in the United States since the 1990s, with the latest data showing an estimated 4.7 million Americans who had reportedly tried MA at least once in their lives [1]. Despite ongoing work by the drug enforcement agencies, unfortunately, the rates of MA use in the United States and other countries continue to rise. The data from a United Nations publication reports that the rate of methamphetamine use ranges from 0.2 to 1.3 percent of the population aged 15 to 64 years.

A thyroid crisis precipitated by sympathomimetic agents (Methamphetamine, Cocaine, and Pseudoephedrine) is probably underreported [2-5]. A thorough literature search revealed only 2 previously reported cases of a thyroid storm precipitated by Methamphetamine use [3, 4]. The pathophysiology of Methamphetamine precipitating a thyroid storm is still not clear. Methamphetamine produces its effects by increasing the release of neurotransmitters (dopamine, norepinephrine, and serotonin) from the nerve terminals, inhibiting the neurotransmitters' breakdown and blocking the neurotransmitters' reuptake in the presynaptic nerve terminals [6]. These various mechanisms of action of MA eventually result in increased levels of the neurotransmitters with resultant increased adrenergic effects.

Methamphetamine can also dysregulate the hypothalamic-pituitary-thyroid axis and cause an elevation in TSH and T4 levels and impair the feedback mechanism. This can result in an unregulated production of thyroid hormones [7]. Thyroid hormones can potentiate the effects of endogenous catecholamines, possibly by increasing the number of alpha and beta-adrenergic receptors, and vice versa, catecholamines can increase the production of thyroid hormones [8]. During pathological states, this synergistic interaction between the thyroid and the sympathoadrenal system can create a self-sustaining chain reaction resulting in a hyperadrenergic crisis [9]. Methamphetamine, by increasing the production of thyroid hormones and causing a hyperadrenergic state, can create a perfect condition for the generation of the storm, i.e., the thyroid storm. Considering thyroid storm in patients with hyperadrenergic states with a prompt institution of effective and appropriate treatment can be a matter of life and death for these patients. Clinical features of methamphetamine toxicity and thyroid crisis are similar, but the management is different for these two potentially life-threatening conditions.

4. Conclusion

Medical providers should consider thyroid crisis in patients with sympathomimetic toxidrome like methamphetamine toxicity. A simple blood test for TSH is all that is needed. This case should help add to the growing literature on thyroid storm triggered by methamphetamine use.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Funding

The authors declare that there was no funding for this study.

References

1. Anglin MD, Burke C, Perrochet B, et al. History of the methamphetamine problem. J Psychoactive Drugs 32 (2000): 137-141.
2. Lacy ME, Utzschneider KM. Cocaine Intoxication and Thyroid Storm: Similarity in Presentation and Implications for Treatment. J Investig Med High Impact Case Rep (2014)
3. Viswanath O, Menapace DC, Headley DB. Methamphetamine Use with Subsequent Thyrotoxicosis/Thyroid Storm, Agranulocytosis, and Modified Total Thyroidectomy: A Case Report. Clin Med Insights Ear Nose Throat (2017).
4. Lee JSG, Brown IE, Semrad AM, et al. Getting around the gut: a unique management challenge of thyroid storm precipitated by amphetamine-associated duodenal ischemia leading to compromised enteric absorption. BMJ Case Rep 14 (2021): e238889.
5. Wilson BE, Hobbs WN. Case report: pseudoephedrine-associated thyroid storm: thyroid hormone-catecholamine interactions. Am J Med Sci 306 (1993): 317-319.
6. Fleckenstein AE, Volz TJ, Riddle EL, Gibb JW, Hanson GR. New insights into the mechanism of action of amphetamines. Annu Rev Pharmacol Toxicol 47 (2007): 681-698.
7. Jacobson T, Steckler T, Wilson BE. Sympathomimetic drug abuse masking an endogenous hyperadrenergic state, Graves' disease. Behav Med 20 (1994): 91-94.
8. Bilezikian JP, Loeb JN. The influence of hyperthyroidism and hypothyroidism on alpha- and beta-adrenergic receptor systems and adrenergic responsiveness. Endocr Rev 4 (1983): 378-388.
9. Silva JE, Bianco SD. Thyroid-adrenergic interactions: physiological and clinical implications. Thyroid 18 (2008): 157-165.

This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC-BY) license 4.0