Comatose Deception, Benzodiazepine masquerade of Myxedema Coma

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Introduction

Myxedema coma is a life-threatening condition due to severe hypothyroidism or long-standing untreated hypothyroidism. This condition can be triggered by factors such as infection, exposure to certain medications, or other stressors on the body. Symptoms of myxedema coma include hypothermia, altered mental status, and fluid accumulation. This condition can be life threatening and requires immediate medical attention.

Benzodiazepines are often prescribed to address various conditions such as anxiety, sedation, and seizures. However, they have significant side effects including drowsiness, sedation, low blood pressure, and slow heart rate. These side effects can mimic symptoms of other conditions such as myxedema coma. In this case we describe a patient who initially appeared to have overdosed on benzodiazepines but was later diagnosed with myxedema coma.

Case Presentation

A 49-year-old lady presented to the emergency department in an obtunded state and decreased respiratory drive. Earlier that day, she accidentally ingested a fish burger despite having a fish allergy, and experienced wheezing, facial swelling, and vomiting. She self-administered an epinephrine pen to manage the allergic symptoms. She was found unconscious by her daughter, who promptly called emergency medical services (EMS). Although no signs of an allergic reaction observed, EMS noted bradycardia and a reduced respiratory drive and administered naloxone and flumazenil which mildly improved her symptoms.

The patient's medical history included post-traumatic stress disorder, generalized anxiety disorder, bipolar type one, fibromyalgia, and hypothyroidism with inconsistent medication (levothyroxine) adherence. Her medication regimen included clonazepam (2mg twice daily), trazodone (100mg twice daily), tizanidine (4mg twice daily), fluoxetine (20mg daily), and temazepam (50mg twice daily). She was not taking levothyroxine at that time daily. Her daughter reported a progressive decline in strength, fatigue, weight gain, hoarse voice, dyspnea on exertion, and cold intolerance over the recent months.

During assessment, vital signs indicated a temperature of 97 degrees Fahrenheit, bradycardia (56), respiratory rate (10), blood pressure 93/38, oxygen saturation of 85% on room air, and body mass index of 55. Physical examination revealed obesity, acute distress, constricted pupils (2-3), a short supple neck, audible inspiratory stridor, tenderness in the left upper quadrant of the abdomen, and 2+ pitting edema in bilateral lower extremities.

Laboratory findings revealed hypercarbia (31.7 mmol/L), reduced glomerular filtration rate (39.4 ml/min/1.73 mm2), elevated thyroid-stimulating hormone (179 uIU/ml), and low free thyroxine (0.1 ng/dl) levels. With a Popoveniuc score of 80, she met the diagnostic criteria for myxedema coma and was promptly started on levothyroxine.

Conclusion

Initial diagnosis of benzodiazepine overdose was questioned due to consistent use, confirmed by pharmacy refill dates, and most importantly lack of typical symptoms of overdose and response to treatment. Later it was proved that noncompliance with levothyroxine led to myxedema coma triggered by benzodiazepines which worsened her condition. This case emphasizes the need for vigilance in distinguishing between benzodiazepine side effects and other conditions. Timely recognition and appropriate treatment, such as levothyroxine initiation, are crucial in managing myxedema coma and preventing life-threatening complications.