A Mysterious Case of Unresponsiveness that was found to be Myxedema Coma

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Background

Myxedema coma is an extreme complication of hypothyroidism in which patients exhibit multiple organ abnormalities and progressive mental deterioration.[1] Myxedema coma occurs when the body's compensatory responses to hypothyroidism are overwhelmed by a precipitating factor, such as infection.[1] This condition affects women and the elderly disproportionately, and is exacerbated during the winter months. The cardinal feature is the deterioration of the patient's mental status. It can be recognized on exam by the classic myxedematous face (generalized puffiness, macroglossia, ptosis), goiter or indication of thyroid surgery, non-pitting edema in the lower extremities, and altered mental status. Patients will be hypothermic and bradycardic, with resulting hypotension. In the case below, we discuss presentation, management, and hospital course of a patient who presented with myxedema coma. Additionally, we discuss the treatment of resistant bradycardia with Dopamine.

Case Report

A 63-year-old female presented to ED after being found unresponsive at her nursing home. Patient was bradycardic in the 20s with SBP 50/palpable when EMS arrived. Transcutaneous pacing was started en route to hospital.

PMHx: a-fib (Eliquis), diastolic CHF, OSA, COPD, and hypothyroidism, with chronic VDRF. On arrival she was paced to 71 bpm and BP 125/77. Physical exam demonstrated a lethargic woman who responded to pain. Head CT was negative for intracranial bleed. TSH was ordered and returned at 85.7. Upon records review, a TSH >50 was noted a week prior. She was given Atropine, calcium gluconate, insulin, kayexalate, and started on fluids in the ED. Bradycardia was resistant to Atropine, and Dopamine drip was initiated with admission to the ICU.

The following day T4/T3 were low on morning labs. AM Cortisol was elevated, likely due to the patient's history of chronic steroid use. This raised our suspicion for myxedema coma in the setting of altered mental status, hyponatremia, hypothermia, and bradycardia. Both T4 and T3 replacement therapy was initiated. Patient improved clinically over the next two days. She was weaned off Dopamine drip and subsequently downgraded. Patient was discharged back to nursing home after six days in the hospital.

Vitals

	Initial Vitals on Scene	ED Vitals	Day 1 ICU	Day 2 ICU
BP	50/palpable	125/77	124/72	130/81
HR	20s	70 (paced)	70 (paced)	64
RR & Pulse Ox	100% on 40% FiO2	18 / 100% on 40% FiO2	18 / 100% on 40% FiO2	18 / 100% on 40% FiO2
Temp (C)		35.7	36	36.5

Labs/Imaging

Na 133	**BUN 41**	WBC 9.5	Glucose 109	**TSH 85.7**	Blood Cx: neg
K 8.1	**Cr 6.6 (1.2)**	Hgb 8.7	Mg 4.1	Lactic 2.0	COVID swab: neg
Cl 99	AST/ALT 77/31	Hct 27	P 10.1	PT/INR 22.7/2.07	U/A: neg
HCO 29.1	Alk Phos 116	Plt 300	Ca 7.9		Troponin: neg x2

Radiology

Head CT negative for bleed

CXR & CT A/P: right lower lobe opacity

References

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Discussion

Myxedema coma is a life-threatening endocrine condition resulting from severe thyroid hormone deficiency. It develops out of a long-standing undiagnosed or untreated hypothyroidism, and is precipitated by an acute event, such as: infection, trauma, or surgery. Cardinal symptoms of myxedema coma are hypothermia, decreased mental status, and hypoventilation with risk of pneumonia and hyponatremia. There is no screening tool for myxedema coma, rather it is avoided by proper monitoring of thyroid levels, good physician-patient communication, and avoidance of acute life stressors.[2]

Diagnosis is based on clinical criteria as measurements of thyroid hormone levels do not differ between uncomplicated severe hypothyroidism and myxedema coma. Prompt initiation of multimodal treatment is critical. In addition to substitution of thyroid hormones and glucocorticoids, treatment focusses on critical care measures to treat hypoventilation and hypercapnia, with correction of hyponatremia and hypothermia.[2]

Additionally, Dopamine has been considered as therapy for unstable bradycardia in patients who are refractory to atropine. A RCT feasibility trial was conducted in 2008 that showed initial promise that Dopamine therapy was equivalent to transcutaneous pacing (TCP) for 30-day mortality in patients with unstable bradycardia and hypotension; much like our 63-year-old patient.[3] The conclusion of this study was that a larger multi-center trial would be needed; however, the 2018 ACC guidelines included Dopamine as a level IIb recommendations for bradycardia attributable to AV block.[4]

Conclusion

Myxedema coma can occur as the culmination of severe, longstanding hypothyroidism or be precipitated by an acute event in a poorly controlled hypothyroid patient. Myxedema coma can occur in patients with central or drug-induced hypothyroidism (lithium, amiodarone, etc). Hypothyroidism can exist with normal cortisol levels, but an elevated TSH will further suppress the adrenal axis.

Dopamine is not a first line therapy for bradycardia but can be used in Atropine resistant patients. Also, it showed the same impact on 30-day mortality as transcutaneous pacing in patients with hypotension.