

# Visual Hallucinations in a Patient with Myxedema Coma

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## Background

- Myxedema coma is a severe and life threatening form of decompensated hypothyroidism. It presents with multiple organ dysfunction and progressive mental deterioration.
- Cases of myxedema coma usually occur in women with significant mortality rate, 25-60% even when they are under proper treatment.
- In the peer-reviewed literature, the incidence of myxedema coma is 0.22 million cases per year.
- This case describes a male patient with myxedema coma associated with visual hallucinations.

## Case Presentation

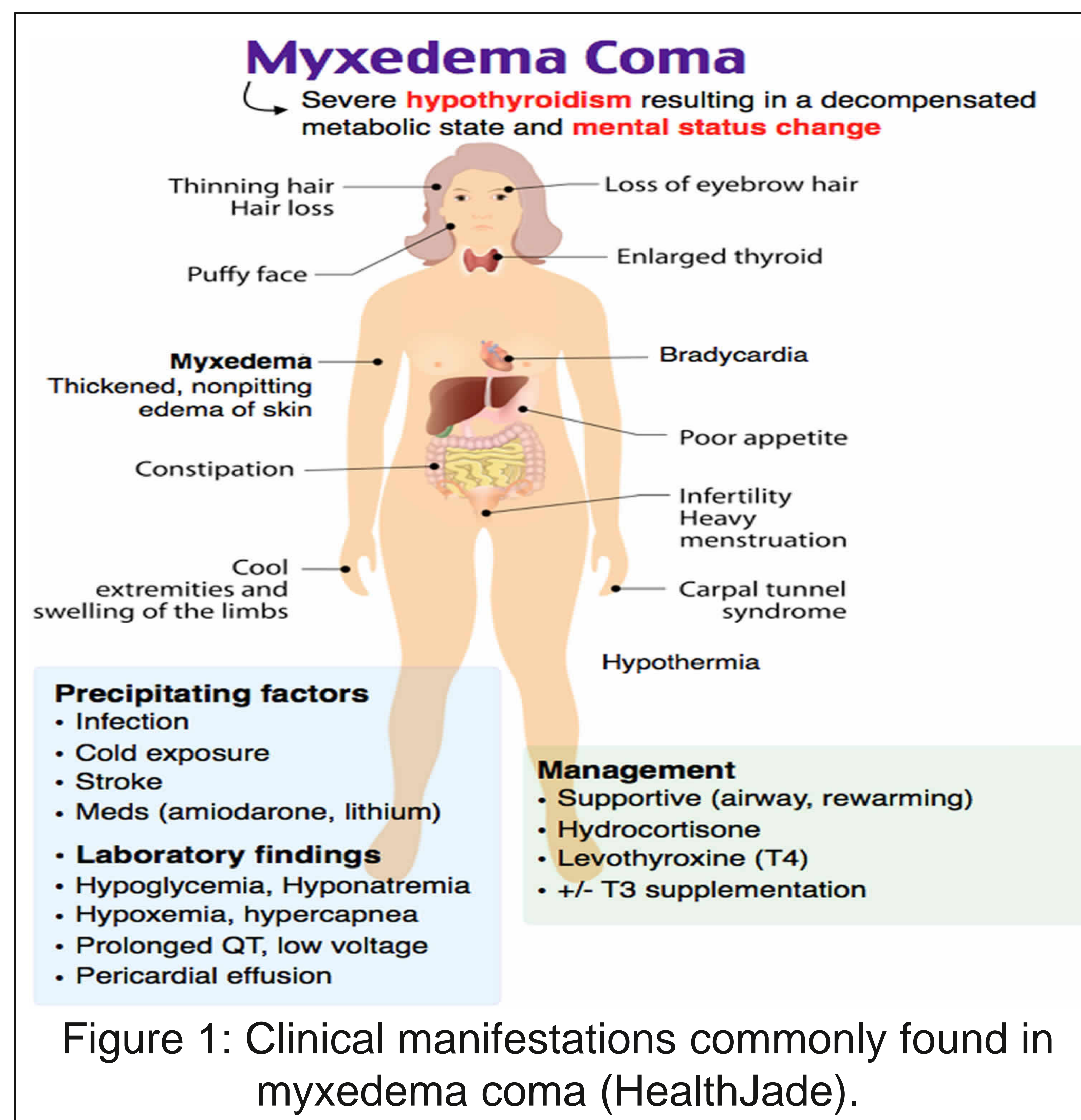
A 72-year-old Caucasian male with past medical history of hypothyroidism, coronary artery disease status post one stent, essential hypertension, right renal cell carcinoma status post nephrectomy on chemotherapy, and chronic kidney disease stage 4 had presented to the ED with weight gain, visual hallucinations for a week. He was taking levothyroxine 100 mcg daily, lisinopril 40 mg daily, clopidogrel 75 mg daily, aspirin 81 mg daily, and metoprolol 25 mg daily. On arrival to the emergency department, he was alert but not oriented and was confused. Physical examination revealed generalized edema more prominent bilaterally in lower extremities. His vital signs were stable with blood pressure 130/80 mmHg, heart rate 72 beats/minute, respiratory rate 18 breaths/minute, and temperature 97.9 F.

Patient was subsequently admitted to the intensive care unit. Intensivist and nephrologist were consulted. The nephrologist started the patient on intravenous (IV) 3% sodium chloride. At the same time, the intensivist placed him on thyroxine 50 mcg IV daily, liothyronine 5 mcg orally (PO), and hydrocortisone 80 mg every six hours PO to rule out adrenal insufficiency. Both the thyroid hormone and mineralocorticoid were continued for three days. Cortisol level returned within normal limits and hydrocortisone was discontinued.

On the third day, the sodium levels, liver enzymes, and mental status improved, and his thyroid hormone medication was switched to PO. Patient was intensive care unit for four days and then transferred to the medical floor. He was discharged home on PO thyroxine.

## Results

- Laboratory results revealed a sodium level of 117 mEq/L, creatinine 2.4 mg/dl, aspartate aminotransferase 189 U/L, thyroid stimulating hormone (TSH) 75.70  $\mu$ U/mL, and free thyroxine (T4) 0.47  $\mu$ U/mL.
- Cortisol level returned within normal limits .



## Discussion

- One of the most remarkable clinical manifestation of our patient was visual hallucination.
- From peer-reviewed literature, few cases of myxedema coma reported hallucinations. In these cases, patients have history of Hashimoto's thyroiditis, thyroidectomy or medication noncompliance<sup>6,7,8</sup>.
- In our case, a possible cause of myxedema coma could have been due to his concomitant treatment with levothyroxine and the chemotherapy drug for renal cell carcinoma.
- Most of the time, in the emergency department, thyroid stimulating hormone test is not ordered because it is not considered as part of the tests ordered in an acute setting.
- It is important to check for thyroid abnormalities as part of the differential diagnosis in patients who present with acute changes in behavior or cognition as they can prompt treatment quicker.

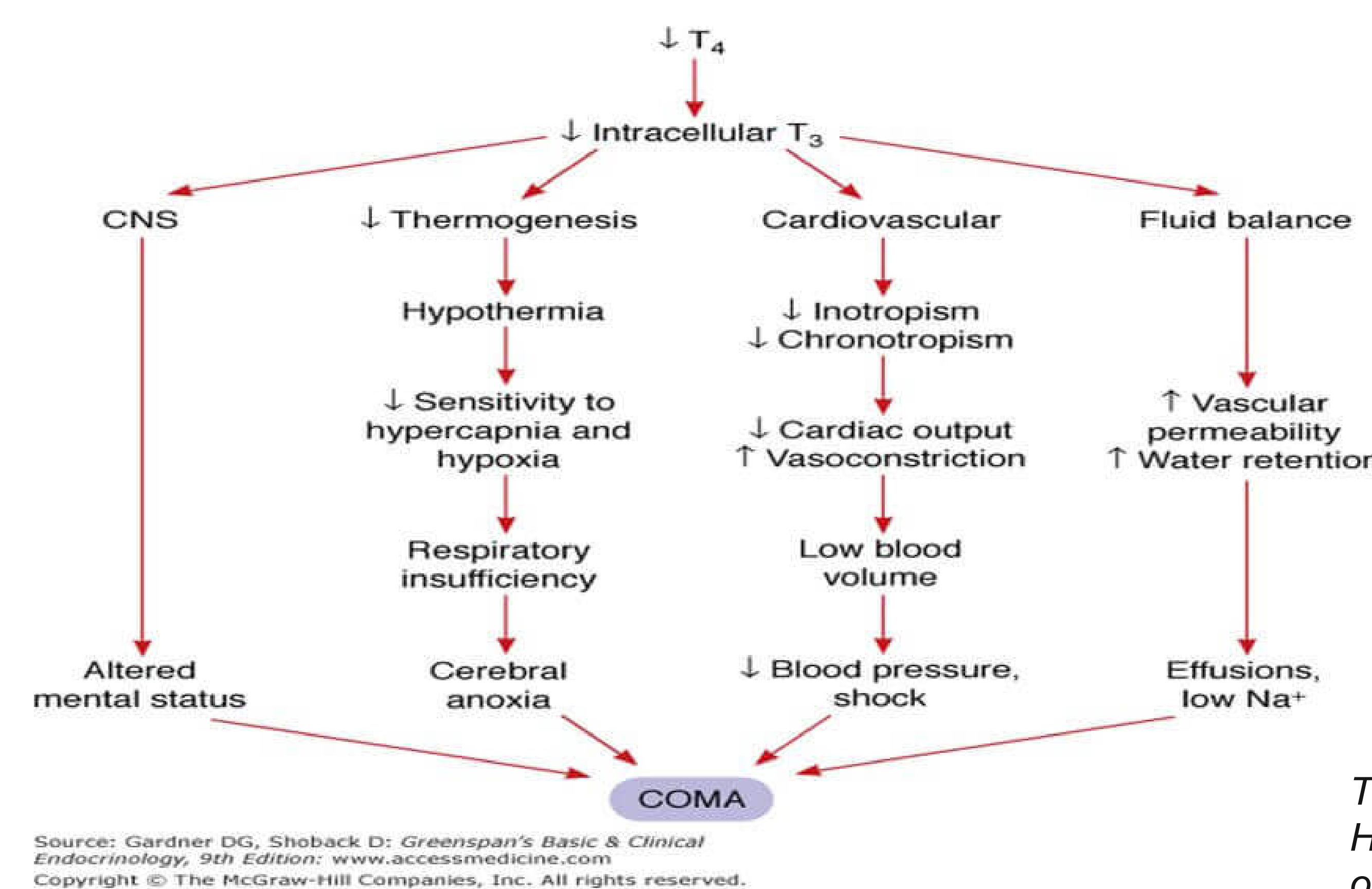
## Conclusion

- The prognosis for myxedema coma is difficult to establish due to the small number of cases reported.
- This disorder is best managed by an inter-professional team to enable prompt diagnosis and management. Identifying a precipitating cause is imperative.
- Endocrinology consult is necessary on clinical suspicion.
- This case draws attention that prompt diagnosis by checking thyroid function tests and rapid intervention are essential in reducing mortality.

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## Pathogenesis of myxedema coma



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