

Myxedema Coma due to Autoimmune Thyroiditis Presenting as Acute Encephalopathy in an Elderly Male: A Case Report

Dr Puratchi Kodi A

Abstract: Myxedema coma is a rare but life-threatening manifestation of severe hypothyroidism characterized by altered mental status, hypothermia, bradycardia, and metabolic disturbances. It predominantly occurs in elderly individuals and is associated with high mortality if not recognized early. We report the case of a 65-year-old male who presented with progressive drowsiness and altered sensorium for five days, preceded by a three-month history of constipation, excessive sleepiness, and weight gain. Clinical examination revealed coarse skin, non-pitting edema, hypothermia, bradycardia, and hypoventilation. Laboratory investigations demonstrated severe hypothyroidism with free T3 of 0.30 pg/mL, free T4 of 4.63 µg/dL, and thyroid-stimulating hormone (TSH) >100 mIU/L. Autoimmune thyroid markers were markedly elevated with anti-thyroid peroxidase antibodies of 484 U/mL and anti-thyroglobulin antibodies of 725 IU/mL, confirming autoimmune thyroiditis. The patient also had hyponatremia and metabolic encephalopathy. Imaging studies did not reveal any structural intracranial pathology. A diagnosis of myxedema coma secondary to Hashimoto thyroiditis was made. The patient was treated with thyroid hormone replacement therapy, hydrocortisone, electrolyte correction, ventilatory support, and vasopressor therapy with gradual improvement in neurological status.

Keywords: Myxedema coma, severe hypothyroidism, Hashimoto thyroiditis, encephalopathy, hyponatremia

1. Introduction

Myxedema coma represents the extreme manifestation of untreated or poorly controlled hypothyroidism and is considered an endocrine emergency with high mortality rates ranging from 30–60%. It typically occurs in elderly individuals and may be precipitated by infections, medications, exposure to cold, cardiovascular events, or metabolic disturbances.

The clinical presentation includes altered mental status, hypothermia, hypotension, bradycardia, hypoventilation, and electrolyte abnormalities such as hyponatremia. Autoimmune thyroiditis (Hashimoto thyroiditis) is the most common cause of primary hypothyroidism in adults. In severe untreated cases, progressive decline in thyroid hormone levels may culminate in myxedema coma. Due to its nonspecific presentation, the diagnosis is often delayed. Early recognition is essential to improve outcomes.

2. Case Presentation

A 65-year-old male presented with progressive drowsiness and lethargy for five days. Initially he responded to verbal commands but gradually developed reduced responsiveness to all forms of stimuli. The patient had a history of chronic constipation, excessive sleepiness, and progressive weight gain for three months prior to admission.

There was no history of fever, seizures, trauma, headache, vomiting, or focal neurological deficits. The patient had a past history of pancreatic surgery five years earlier and was known to have hypertension but was not on regular medications.

On examination the patient was drowsy with reduced responsiveness. Vital signs revealed hypotension and bradycardia. Physical examination showed hypothermia, cool extremities, coarse dry skin, and non-pitting edema.

Respiratory examination revealed hypoventilation. Neurological examination showed depressed sensorium without focal deficits. Fundus examination revealed no papilledema.

3. Investigations

Thyroid profile showed severe hypothyroidism with free T3 0.30 pg/mL, free T4 4.63 µg/dL and TSH >100 mIU/L. Autoimmune thyroid markers were markedly elevated with anti-thyroid peroxidase antibody 484 U/mL and anti-thyroglobulin antibody 725 IU/mL confirming autoimmune thyroiditis.

Additional investigations revealed hyponatremia with reduced serum osmolality (~240 mOsm/kg), hypercholesterolemia (>300 mg/dL), anemia, and elevated inflammatory markers. Liver and renal function tests were within acceptable limits.

Chest X-ray did not show significant abnormalities. CT brain did not reveal any acute intracranial pathology. Fundus examination showed no papilledema.

Diagnosis

Myxedema coma secondary to autoimmune hypothyroidism (Hashimoto thyroiditis) presenting with metabolic encephalopathy.

4. Treatment

The patient was admitted to the intensive care unit and managed with thyroid hormone replacement therapy. Levothyroxine (LT4) was administered as an initial loading dose followed by daily replacement therapy. In situations where intravenous preparations were unavailable, levothyroxine was administered through a nasogastric tube.

Empirical stress-dose corticosteroid therapy with intravenous hydrocortisone 50 mg every 6 hours was administered to prevent possible adrenal insufficiency.

Supportive therapy included oxygen supplementation, ventilatory support for hypoventilation, correction of electrolyte imbalance including hyponatremia, intravenous fluids, vasopressor therapy with norepinephrine and dopamine for hypotension, and passive warming measures.

Outcome and Follow-Up

Following initiation of thyroid hormone replacement and supportive management, the patient showed gradual improvement in neurological status. Blood pressure stabilized with vasopressor therapy and metabolic abnormalities were corrected. The patient's sensorium improved progressively during hospitalization.

5. Discussion

Myxedema coma is a rare but life-threatening complication of severe hypothyroidism characterized by multisystem involvement. Altered mental status is a hallmark feature and may range from confusion to coma.

Hyponatremia in hypothyroidism results from impaired free water clearance due to increased antidiuretic hormone secretion and decreased renal perfusion. Cardiovascular manifestations such as hypotension and bradycardia occur due to reduced cardiac output and impaired autonomic regulation.

Autoimmune thyroiditis is the most common cause of primary hypothyroidism worldwide. Elevated anti-thyroid peroxidase and anti-thyroglobulin antibodies confirm the autoimmune etiology. Early recognition and aggressive treatment significantly improve survival.

6. Conclusion

Myxedema coma should be considered in elderly patients presenting with unexplained altered sensorium, particularly when accompanied by features suggestive of hypothyroidism. Early diagnosis and prompt initiation of thyroid hormone replacement therapy with intensive supportive care are essential to reduce mortality.

7. Learning Points

- 1) Myxedema coma is a rare endocrine emergency associated with high mortality.
- 2) Elderly patients may present with altered sensorium rather than classic hypothyroid symptoms.
- 3) Hyponatremia and hypoventilation contribute to encephalopathy.
- 4) Autoimmune thyroiditis is a common cause of severe hypothyroidism.
- 5) Early diagnosis and prompt treatment significantly improve patient outcomes.

References

- [1] Jameson JL, Mandel SJ, Weetman AP. Disorders of the Thyroid Gland. Harrison's Principles of Internal Medicine. 21st Edition.
- [2] Wartofsky L, Burman KD. Altered mental status in hypothyroidism: Myxedema coma.
- [3] Mathew V et al. Myxedema coma: A new look into an old crisis.
- [4] Rodriguez I et al. Factors associated with mortality of patients with myxedema coma.