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Myxedema coma: A Case Report

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ABSTRACT:

Myxedema coma is a rare, life-threatening complication of prolonged, severe hypothyroidism. It most commonly affects elderly individuals and often presents with decreased consciousness, hypothermia, and metabolic dysfunction. Prompt recognition and treatment are essential for survival. This case study explores an 88-year-old male patient who presented with progressive bilateral lower limb weakness and altered sensorium, later diagnosed with Myxedema coma. The study emphasizes clinical presentation, diagnostic workup, and therapeutic strategies employed.

Keywords: myxoedema coma, hypothyroidism, altered sensorium, geriatric emergency, endocrine crisis

1. INTRODUCTION

Myxedema coma represents the most severe and life-threatening manifestation of hypothyroidism. It is characterized by progressive deterioration of mental status, hypothermia, bradycardia, and multisystem organ involvement. This condition is often precipitated by external stressors like infection, trauma, or cold exposure in a person with undiagnosed or untreated hypothyroidism.

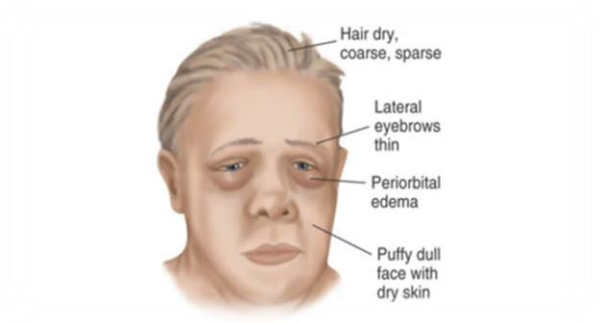

Fig-1: Myxedema Face

Epidemiology

Global Prevalence (WHO Estimates):

- Total Affected Population (Hypothyroidism): ~5% of global population
- Adults: 4–10%
- Older Adults (60+ years): Higher prevalence in females; rare in elderly males
- Children and Adolescents: Less than 2%

Prevalence of Myxedema Coma

In India, the estimated incidence of myxedema coma is approximately 1.08 cases per million population per year, based on tertiary hospital data. This rate is higher than in many Western countries and reflects the widespread prevalence of untreated hypothyroidism. Limited awareness and late diagnosis contribute to underreporting and increased morbidity.

Etiology & Risk Factors

- Longstanding untreated primary hypothyroidism
- Autoimmune thyroiditis (e.g., Hashimoto's disease)
- Thyroid surgery or radioiodine therapy
- Infection, trauma, or use of CNS depressants
- Male sex (rare), age >80 years

2. CASE PRESENTATION

Present Medical History:

Mr XYZ 88-year-old male was admitted in IGMC, SHIMLA (H.P.) ON 5/02/2025 to the emergency department with progressive bilateral lower limb weakness for 15 days, initially affecting gait and posture, eventually leading to inability to walk without assistance. Five days prior to admission, he developed altered sensorium characterized by confusion, disorientation, and a marked reduction in speech output. The family also noted increased sleepiness, lethargy, slurred speech, dry skin, and cold intolerance. He had reduced appetite, constipation, and decreased fluid intake over the previous week. Over the last 2 days, the patient became completely unresponsive. There was also a history of hoarseness of voice and facial puffiness. No fever, trauma, seizures, or signs of infection were observed. Urine output had significantly declined over the last 3 days, raising suspicion of underlying metabolic derangement.

Chief Complaints:

- Weakness in bilateral lower limbs for 15 days
- Altered sensorium for 5 days
- Increased sleepiness and lethargy
- Slurred speech
- Cold intolerance
- Reduced appetite and constipation

Present/Past Surgical History:

No history of past or present surgeries.

PAST HEALTH HISTORY:

- **Past Medical History:** No known chronic illnesses previously diagnosed.
- **Childhood Illness:** Normal childhood development.
- **Other Illnesses:** None reported.

FAMILY HEALTH HISTORY:

- **Type of family:** Nuclear
- **No. of family members:** 5
- **Illnesses:** No known hereditary or chronic illnesses in the family

Family Chart:

Name	Relation to Patient	Age	Sex	Education	Occupation
Mr. X (Patient)	Self	88	M	5th Pass	Retired Farmer
Mrs. X	Wife	82	F	Illiterate	Homemaker
Mr. Y	Son	55	M	Graduate	Government Clerk
Mrs. Y	Daughter-in-law	50	F	12th Pass	Homemaker
Master Z	Grandson	22	M	College Student	Student

PERSONAL HISTORY:

The patient is a retired farmer who resides with his wife, son, daughter-in-law, and grandson in a rural village. His lifestyle has been sedentary in recent years due to age-related physical decline. He followed a mixed diet consisting of cereals, pulses, dairy, and occasional meat; however, his dietary intake had significantly decreased over the past 10 days, with very little solid food consumed and minimal water intake. The patient has experienced persistent constipation, and his urine output progressively reduced to less than 300 mL/day by the third day of admission. He reports no history of alcohol use, smoking, or recreational drug use. He was mostly housebound and depended on his family for mobility and personal care. He had limited education (5th pass) and minimal exposure to modern healthcare facilities throughout his life. His sleep pattern was disrupted, with increased daytime drowsiness and night-time restlessness.

PHYSICAL EXAMINATION

General examination:

- Elderly male, unconscious, pallor present, no cyanosis, edema or icterus
- Height: 162 cm
- Weight: 70 kg
- BMI: 26.7 kg/m²

Vital signs table:

S. No	Parameter	Normal Value	Day 1	Day 2	Day 3	Remarks
1	**Temperature**	97.8°F - 99.1°F	95.2°F	96.0°F	97.0°F	Hypothermic
2	**Pulse**	60-100 bpm	48 bpm	52 bpm	58 bpm	Bradycardia
3	**Respiratory Rate**	12-20 /min	10 /min	12 /min	14 /min	Decreased, improving
4	**Blood Pressure**	100-120/70-80 mmHg	90/60 mmHg	100/70 mmHg	110/72 mmHg	Hypotensive, improving

SYSTEM ASSESSMENT:

- **CNS:** Glasgow Coma Scale: 8/15, delayed response to stimuli
- **CVS:** Distant heart sounds, bradycardia
- **Respiratory:** Shallow respiration
- **Abdomen:** Soft, non-tender
- **Skin:** Dry, coarse, pale

INVESTIGATIONS:

Test	Normal Range	Patient Value	Interpretation
TSH	0.4-4.5 µIU/mL	>100 µIU/mL	Severe hypothyroidism
Free T4	0.8-2.0 ng/dL	0.2 ng/dL	Markedly decreased
MPV	7.5-11 fL	12.5 fL	Increased
PCT	0.2-0.4%	0.6%	Increased
RDW-CV	11.5-14.5%	16.2%	Elevated
Eosinophils	1-3%	6%	Elevated
Chloride	96-106 mmol/L	89 mmol/L	Low
Sodium	135-145 mmol/L	128 mmol/L	Hyponatremia
Uric Acid	3.4-7.0 mg/dL	8.9 mg/dL	Elevated

TREATMENT:

Medication	Dose	Route	Frequency	Purpose
Levothyroxine	300 mcg stat, then 100 mcg/day	IV	Once Daily	Thyroid hormone replacement
Hydrocortisone	100 mg	IV	TID	Prevent adrenal crisis
Normal Saline	500 mL	IV	As needed	Volume resuscitation
Oxygen	2-4 L/min	Nasal cannula	Continuous	Improve oxygenation
Paracetamol	500 mg	Oral	PRN	Symptomatic treatment for fever

3. DISCUSSION

ANATOMY OF THE THYROID GLAND

- **Location:** Anterior neck, just below the larynx (voice box), in front of the trachea.
- **Structure:**
    - Lobes: Two lateral lobes (right and left) connected by a narrow band called the isthmus.
    - Sometimes a pyramidal lobe extends upward from the isthmus.

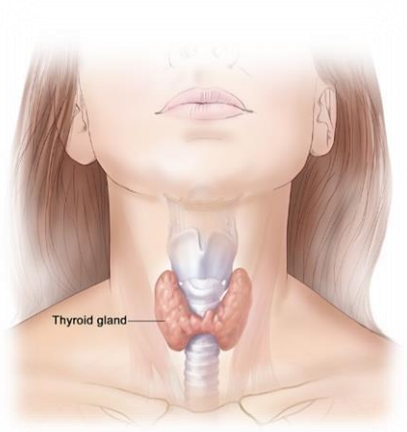

Fig-2: Thyroid Gland

- **Size:** About 4–6 cm long; weighs 15–25 grams in adults.
- **Blood Supply:**
    - Arteries: Superior thyroid artery (from external carotid) and inferior thyroid artery (from thyrocervical trunk).
    - Veins: Superior, middle, and inferior thyroid veins.
- Innervation: Autonomic (sympathetic and parasympathetic) nerves from cervical ganglia.

PHYSIOLOGY OF THE THYROID GLAND

- **Hormones Produced**:
    - Thyroxine (T_4) and Triiodothyronine (T_3): Regulate metabolism, growth, and development.
    - Calcitonin: Lowers blood calcium levels by inhibiting osteoclast activity.
- **Hormone Synthesis**:

1. Iodide uptake from blood into thyroid follicular cells.
2. Oxidation and organification: Iodide is converted to iodine and attached to tyrosine residues on thyroglobulin → forms MIT and DIT.
3. Coupling: MIT + DIT → T_3; DIT + DIT → T_4.
4. Stored in colloid in the follicles and released when needed.

- **Regulation:**
    - Controlled by TSH (thyroid-stimulating hormone) from the anterior pituitary.
    - TSH is regulated by TRH (thyrotropin-releasing hormone) from the hypothalamus.
    - Negative feedback from circulating T_3 and T_4 levels.

INTRODUCTION:

Myxedema coma occurs due to prolonged deficiency of thyroid hormones, leading to decreased cellular metabolism and impaired thermoregulation, cardiovascular, respiratory, and neurological function. The condition is precipitated by external stressors such as infections, trauma, exposure to cold, and certain medications in patients with preexisting hypothyroidism. The central features of the condition include impaired thermogenesis, decreased myocardial contractility, hypoventilation, electrolyte imbalances, and CNS depression. Decreased T4 and T3 levels result in the inability to maintain homeostasis, thus leading to coma-like symptoms. Patients may have a history of untreated thyroid disease or inadequate thyroid hormone replacement.

SYMPTOMS & CLINICAL PRESENTATION:

Hallmarks include altered mental status, hypothermia, bradycardia, hypotension, hypoventilation, and non-pitting edema. In elderly patients, the symptoms may be subtle or nonspecific, which delays diagnosis. Myxedema coma often mimics other disorders like stroke or sepsis. Hence, clinical suspicion supported by laboratory confirmation is critical.

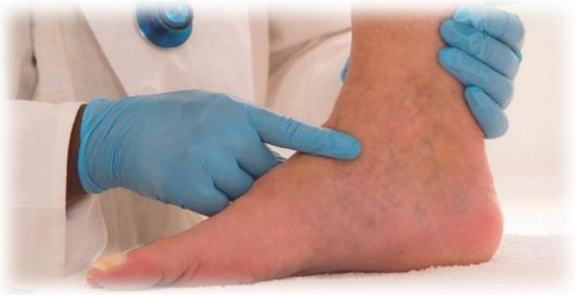

Fig-3: Non pitting edema

Fig-4: Altered Mental Status

DURATION AND DIAGNOSIS CRITERIA (DSM-5):

DSM-5 does not classify myxedema coma as a psychiatric disorder; however, hypothyroidism may contribute to depressive features and altered mental status. Diagnostic confirmation is based on clinical presentation, hormone levels (elevated TSH and low T4), and exclusion of other metabolic or neurologic conditions.

TREATMENT APPROACHES:

Treatment of myxedema coma is multifaceted and requires immediate intensive care. The cornerstone of therapy is intravenous thyroid hormone replacement, typically with high-dose **levothyroxine** and sometimes with **liothyronine (T3)** if available. Hydrocortisone is administered to manage possible coexisting adrenal insufficiency and prevent adrenal crisis. **Supportive care** includes correction of hyponatremia, hypoglycemia, hypoventilation, and hypothermia. Careful fluid management, electrolyte monitoring, passive rewarming techniques, and mechanical ventilation may be necessary. Infections should be ruled out or treated empirically with antibiotics**. Cardiac function and urine output** must be continuously assessed. Early diagnosis and a multidisciplinary approach improve prognosis significantly.

CONCLUSION

This case highlights the diagnostic challenge and clinical importance of recognizing myxedema coma in elderly patients. The patient's gradual neurological decline, coupled with metabolic abnormalities and thyroid dysfunction, emphasized the need for timely intervention. Despite aggressive thyroid hormone therapy and intensive supportive management, the patient’s condition deteriorated. He remained comatose and developed multi-organ dysfunction. Unfortunately, he succumbed to the illness on the sixth day of hospitalization. This case demonstrates the grave prognosis associated with delayed recognition and treatment of myxedema coma. It underscores the importance of public awareness, early detection, and geriatric healthcare accessibility, particularly in rural regions.

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