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Overview on Epidemiology and Management of Myxedema Coma or Crisis

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Abstract

The most severe type of hypothyroidism is known as myxedema coma, which can quickly lead to death if not identified and treated aggressively. The condition known as hypothyroidism is easily detected and treated. But if ignored, it might eventually progress to myxedema coma, the most serious form of hypothyroidism. Since the majority of patients do not initially appear in a coma, the term "myxedema coma" is usually regarded as misleading. Lethargy

usually progresses to stupor, which then becomes a coma with hypothermia and respiratory failure. only clinical criteria are used for diagnosis because thyroid hormone assays are unable to distinguish between simple hyperthyroidism and thyroid storm. Apart from essential medical interventions, the treatment focuses on preventing thyroid hormone production and secretion as antithyroid medications, and preventing the peripheral effects of thyroid hormone as β-blocker, glucocorticoids. The diagnosis of thyroid-stimulating hormone (TSH) in the blood is the same as that of simple hypothyroidism. As soon as a diagnosis is obtained, treatment should start right away. The majority of hospital and commercial laboratories can turnaround a TSH test in a matter of hours.

Keywords: hypothyroidism, myxedema, TSH, coma, thyroid hormone.

Introduction:

A decompensated state of severe untreated hypothyroidism is represented by myxedema coma. The body is unable to sustain homeostasis in this condition through neurovascular adaptations, which are necessary for hemodynamic stability. Myxedema coma may resemble other severe life-threatening illnesses in many ways, including altered mental status, decompensated heart failure, renal failure, respiratory failure, and hypothermia and cold exposure. The majority of symptoms may be nonspecific, which makes diagnosis challenging. It is possible that thyroid dysfunction will be overlooked in the differential diagnosis of these illnesses unless the doctor is obviously suspicious and mentions it, this might have fatal consequences [1]. The majority of individuals who may present with myxedema coma are female since hypothyroidism affects women about eight times more frequently than it does males. Since hypothyroidism often manifests in later life stages, the majority of these women are elderly. Maintaining a high degree of suspicion is crucial, particularly when dealing with an older female patient whose signs and symptoms are consistent with hypothyroidism, hypothermia, hyponatremia, and hypercarbia [2].

Uncommon and severe side effect of hypothyroidism, myxedema coma can be deadly and is characterized by various organ problems linked to impaired sensorium. Patients with hypothyroidism display a variety of physiological changes to make up for their thyroid hormone deficit. Myxedema coma can result from factors like infection that overpower these homeostatic processes. Myxedema coma may be the earliest symptom of any type of hypothyroidism, regardless of the underlying reason. The death rate from myxedema coma varies; some studies put it as high as 60%, while others put it as low as 20 to 25%, even with early diagnosis and treatment [3].

A study done in 2011 to assess the typical clinical presentation, course of therapy, mortality predictors, and issues that cast doubt on the wisdom of the existing ideas in myxedema crisis management, and found that myxedema crises are linked to symptoms of hypothyroidism, hypothermia, hyponatremia, hypercarbia, and hypoxemia. The prognosis might be negatively impacted by a considerable delay in diagnosis. Intensive care units should be made available to patients, along with timely therapy for hypotension, hypothermia, steroid replacement, and thyroid hormone supplementation. Sepsis-Related-Organ Failure Assessment grading system could assist us in early patient identification of those who are at danger of death. Receiving medical treatment as soon as a hypothyroid patient develops a serious disease, particularly sepsis, and making sure thyroid supplements are continued can help significantly reduce morbidity and mortality [4].

Another study done in 2014 to assess myxedema coma (MC) diagnostic criteria, produced that, multisystemic hypothyroidism symptoms in MC patients and the high death rate linked to treatment delays, so a diagnostic score system for MC based on information from both specific case reports taken from the literature and data from retrospective cases identified at our facilities will be a useful manual for early diagnosis. This scoring method evaluated a variety of the diagnostic characteristics linked to MC and discovered that our patient cohort's frequency of observations was comparable to that of those evaluated based on published works [5].

A study in 2019 in Riyadh in Saudi Arabia to evaluate the hypothyroidism patients' knowledge, attitudes, and behaviors in Riyadh, Saudi Arabia. produced that among the responders, 45% were men and 55% were women. Consistent with other research, the study shows that females in the 45+ age group have a greater prevalence of hypothyroidism. Similarly, 24% of respondents strongly disagree that women should be tested for hypothyroidism on a regular basis and that they are more likely to develop the condition than men; on the other hand, 19% of respondents concur that women should be tested for hypothyroidism on a regular basis, so

in order to raise the general population's understanding of hypothyroidism in Saudi Arabia, more public education and awareness campaigns are required [6].

Epidemiology of myxedema coma:

The incidence of myxedema crises is around 0.22 million per year, according to case series and case reports from the western world. However, there is a dearth of comparable epidemiological data from nations around the equator [4] The majority of patients are older women who frequently have long-standing, undetected hypothyroidism. Since winter months account for over 90% of occurrences, it is possible that cold temperatures have a role. Age-related loss of temperature control may also be a factor, as may a reduction in heat output brought on by hypothyroidism. There was a time when estimates of mortality ranged from 50% to 60%. However, death has dropped to between 20% and 25% due to a high degree of clinical suspicion with early diagnosis, improvements in intensive care, and general treatment [7].

Diagnosis of myxedema coma:

Clinical signs and a history of moderate to severe hypothyroidism are typically used to make the diagnosis of MC. Laboratory testing also confirms the diagnosis, showing high serum thyroid-stimulating hormone (TSH) and reduced total thyroid function.as well as triiodothyronine (T3) and free thyroxine (T4). It has been demonstrated that therapy with intravenous T4, supportive care, and early diagnosis enhance results [8] in MC noticed electrolyte abnormalities, reduced free water clearance is the cause of the hyponatremia observed in myxedema coma as free water retention is thought to be caused by increased antidiuretic hormone levels or decreased blood supply to the kidneys. Low serum osmolality is typically linked to hyponatremia. Serum creatinine levels are often raised, and although calcium levels are normally low, they might be higher. Hypoglycemia might be a sign of adrenal insufficiency or the down-regulation of metabolism associated with hypothyroidism. Also hypoxia, hypercapnia, and respiratory acidosis are frequently detected by arterial blood gases. Additionally common are normocytic anemia and mild leukopenia help in MC diagnosis [9].

Causes of myxedema coma or crisis:

Homeostatic systems are disrupted in people with hypothyroidism, resulting in the occurrence of myxedema coma. There are numerous precipitating factors. The following factors are of utmost significance:
Infections: urinary tract infection, pneumonia, viral infections, influenza, and so forth. Causes include burns, the accumulation of carbon dioxide, and physical injury. Conditions of abnormally low body temperature and low blood sugar levels. Low oxygen levels in the blood and strokes. Medications: amiodarone, lithium, sedatives, tranquilizers, anesthetics, opioids, phenytoin, rifampin, diuretics, beta-blockers (due to reduced drug metabolism in individuals with hypothyroidism, there is an increased risk of an overdose of anesthetics and tranquilizers in these patients). Recent case reports in the literature have documented the occurrence of myxedema as a result of anti-TNF medication. Cardiac decompensation, Hemorrhage occurring in the digestive tract, Surgery can lead to a decrease in the release of thyroid hormones due to the impact on the pituitary-thyroid axis in reaction to stress [10,11]. In 2019, a case report demonstrated that a patient had myxedema coma due to diabetic ketoacidosis following a complete thyroidectomy [12].

Clinical presentation of myxedema coma or crisis:

80% of the patients are hypothermic, and all have abnormal mental states. Patients may present with atypical features such as heart blocks, arrhythmias and prolonged QT interval, myocardial infarction, pericardial/pleural effusions, respiratory depression, hypercapnia, bleeding manifestations with prolonged active partial thromboplastin time APTT, acquired von Willebrand factor defects, and psychosis, in addition to the typical features of hypothyroidism. Myopathy, neuropathy, reflex alterations, ataxia, psychotic episodes, headaches, altered mental state with slowness, poor focus and lethargy, hoarseness, and fits are among the neurological signs associated with myxedema. The end outcome would be a condition of coma, and it is important to investigate the roles that hypothermia, CO2 narcosis, cerebral edema, and other metabolic disruptions play in the development of coma [13]. epileptic seizures in myxedema may occur for a variety of reasons, a shift in mental function is brought about by the brain using less glucose, receiving less oxygen, and having less cerebral blood flow. Furthermore, chronic unmanaged hypothyroidism can result in the improper release of antidiuretic hormone, which can lead to cerebral edema and hyponatremia as a result

of the extracellular fluid volume growth. If not identified right once, these neurological alterations may trigger seizures, which might result in irreparable hypoxic damage [14].

Management of myxedema coma or crisis:

Appropriate therapy of the underlying thyroid hormone deficit is crucial in addition to intense supportive care. Unfortunately, many nations do not currently have evidence-based treatment for myxedema since the condition is uncommon and undiagnosed [15]. Levothyroxine replacement treatment raises thyroid-stimulating hormone levels; the recommended daily dosage is 1.5 to 1.8 mcg per kilogram. Even in individuals with prolonged symptoms and appropriate thyroid-stimulating hormone levels, adding triiodothyronine is not advised. Levothyroxine dosages should be started lower for patients who are over 60 or who have ischemic heart disease, whether confirmed or suspected (12.5 to 50 mcg per day). When a woman with hypothyroidism gets pregnant, her weekly dosage should be increased by 30% up to nine doses (taking one extra dose twice a week), with a monthly examination and care scheduled afterward [16].
Initial corticosteroid therapy is advised by the American Thyroid Association recommendations since concurrent adrenal insufficiency affects 5–10% of individuals with myxedema coma. 100 mg of intravenous hydrocortisone should be administered every eight hours until adrenal insufficiency is ruled out before starting thyroid hormone therapy. Prior to starting medication, a random cortisol level should be taken to evaluate adrenal function. A corticotropin stimulation test might be carried out at a later time if required. Following the ruling out of adrenal insufficiency, hydrocortisone may be stopped [17].
It is clear that these patients require thorough monitoring of their cardiovascular condition while receiving treatment in an intensive care unit. Ventilatory assistance is frequently required due to reduced awareness, drug-induced respiratory depression, underlying pneumonia, or, in rare cases, macroglossia or laryngeal myxoedema that obstructs the airway. In addition to the standard care of warm blankets and water, in hypothermia [18].
In myxedema coma emergency, patient with. Immediately receiving intensive supportive care and a combination therapy of 200 μg levothyroxine and 50 μg liothyronine until the fifth hospital day. Subsequently, monotherapy with levothyroxine was continued at a dose of 150 μg daily. The thyroid hormone level reached the normal range a few days later [15].

Conclusion:

If started as soon as possible, thyroxine replacement is quite beneficial in treating hypothyroidism-related myxedema. the hardest part of caring for these individuals is figuring out the diagnosis quickly enough to start the right treatment.
Provide women's awareness and let them know that they are the most vulnerable to myxedema coma especially elder women with hypothyroidism.
In an intensive care unit, treatment needs to start right away. Thyroid hormone treatment is essential for life, however it's not clear if it should be given as T4, T3, or both. Adjunctive treatments such pressors, fluids, breathing, warmth, and corticosteroids may be necessary for survival.

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