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A CASE OF MYXEDEMA SEEN WİTH ACUTE RESPİRATORY FAİLURE

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ABSTRACT

Hypothyroidism is a clinical table that occurs as a result of insufficient effects of thyroid hormones. Myxedema coma is a table of severe hypothyroidism causing a diminished mental status, hypothermia, hypotension, bradycardia, hyponatremia and hypoventilation. Frequently seen symptoms include listlessness, fatigue, sleepiness, constipation, intolerance to cold, dry skin, thickening of the voice, slow movements, weight gain, cramps, carpal tunnel syndrome, and menorrhagia. Atypical presentations are seen more often in elderly patients with hypothyroidism. The case is here presented of a 72-year old patient with myxedema who presented at the Emergency Department with complaints of respiratory problems, sleepiness and inability to speak.

KEYWORDS: Hypothyroidism, Myxedema coma, respiratory failure.

INTRODUCTION

Hypothyroidism is a clinical table that occurs as a result of insufficient effects of thyroid hormones. Myxedema is defined as hypothyroidism characterised by an accumulation of glycosaminoglycan in subcutaneous and interstitial tissues together with severe thyroid insufficency[1]. Myxedema coma is an uncommon but life-threatening complication of hypothyroidism that has been left untreated for a long time. The incidence has been reported as 1.22 per million. In a patient with hypothyroidism, while the body temperature is protected with chronic peripheral vasoconstriction, diastolic hypertension and reduced blood volume, an intervening facilitating condition impairs adaptive mechanisms and a myxedema table develops.

Myxedema is a table of severe hypothyroidism causing a diminished mental status, hypothermia, hypotension, bradycardia, hyponatremia and hypoventilation.[2] Factors disrupting the compensatory response of the body to hypothyroidism lead to myxedema coma. Patients in myxedema coma are seen to have clinically long-term hypothyroidism. Common symptoms include listlessness, fatigue, sleepiness, constipation, intolerance to cold, dry skin, thickening of the voice, slow movements, weight gain, cramps, carpal tunnel syndrome, and menorrhagia. Atypical presentations are seen in elderly patients with hypothyroidism. These findings can be associated with ageing, and can be interpreted as Parkinson's disease, depression or Alzheimer's disease.[1] Myxedema coma occurs in decompensated patients. Despite intense treatment, myxedema coma is a medical emergency with a mortality rate approaching 80%, and it must be treated in the Intensive Care Unit (ICU).[2]

CASE

A 72-year old female was brought by relatives to the ED with complaints of inability to speak for the last 10 days, difficulty walking, sleepiness, confusion, low oral intake and shortness of breath. In the ED, all informed consent forms were obtained from the patient and her relatives for tests and treatment of respiratory problems, and elevated blood pressure. The patient had a low Glasgow Coma Score (GCS) and low urine output, and was admitted to ICU.

On admission to ICU, the patient was confused, sleepy, had dry skin and oedema in the eyelids, and GCS:E3M4V3, TA:170/95, pulse: 92 bpm, O2Sat:88% and body temperature:36.7˚C. There was unsightly bilateral pretibial oedema. An incision line was determined in the lower part of the neck from a previous thyroid operation. According to the information from the patient's relatives, the patient had previously been diagnosed with hypertension and diabetes mellitus and was followed up with medical treatment. When the neck incision was questioned, it was learned that the patient had undergone a thyroidectomy 10 years previously and did not usually take her medication. The results of the laboratory tests made on admission and before discharge are shown in Table 1.

Table 1: The laboratory tests made on admission and before discharge.

Test	Admission	Discharge
Glucose (mg/dL)	230	185
Urea (mg/dL)	52	81
BUN (mg/dL)	25	38
Creatinine (mg/dL)	1.95	1.8
AST (IU/L)	92	17
ALT (IU/L)	67	22
Na (mmol/L)	128	142
K (mmol/L)	3.29	3.95
Ca (mg/dL)	9.04	9.03
Mg (mg/dL)	1.61	2.01
CRP (mg/dL)	1.64	0.60
Ph	7.43	7.48
PaO_2 (mmHg)	72.9	118
$PaCO_2$ (mmHg)	36.8	34.6
Sat	92	98.6
HCO_3	24.2	25
Base deficit	0.2	1.8
TSH (mU/L)	57.6	10.3
Free T3 (ng/L)	0.91	0.93
Free T4 (ng/L)	0.44	0.83
NT PRO-BNP (Pro-Brain Natriuretic Peptide) (ng/L)	2447	1872

BUN: Blood urea nitrogen, AST: Aspartate aminotransferase, ALT: alanine aminotransferase, CRP; C-reactive protein, Sat; saturation, HCO3: Bicarbonate

In the imaging examinations of the patient, on the thoracic CT, there was an appearance of fluid within cystic bronchiectasis in the left lung, bilateral mild pleural effusion, and pericardial effusion. On portable echocardiography, EF:55%, no serious valve pathology, and pericardial effusion 10mm adjacent to the right atrium were observed.

Cultures were taken from the patient and tests were applied. Antibiotics were not started as CRP and procalcitonin were within normal limits. The patient was hypertensivs so an esmolol infusion was started. As the patient was determined with TSH: 57.6, sT4:0.44, sT3:0.91, the Endocrinology and Metabolic Diseases Departments were consulted and treatment was started of levothyroxin sodium at a dose of 1 x 50mg for the first 3 days, and 3 x 75mg thereafter. Methylprednisolone (80mg) was administered to the patient before starting the levothyroxin sodium treatment. The Nephrology Department was consulted as it was thought that the patient could have acute renal failure related to the disrupted oral intake, but no additional recommendations were made. On the 2nd day, due to gr(+) cocci production in the blood culture, the Infectious Diseases Department was consulted and piperacillin+tazobactam was

started at a renal dose. Support was provided by the Physical Therapy Unit in respect of respiration and mobility physiotherapy. On day 3, the esmolol infusion was terminated and oral antihyperintensives were started. The GCS of the patient continued to increase and she started to regain cognitive functions. Oral nutrition was started on the 3rd day. While the treatment of the patient was ongoing, the patient was discharged on the request of the patient's family before treatment was completed.

DISCUSSION

Myxedema coma is the table within endocrine emergencies with the highest mortality rate of 80%[2]. Precipitating factors include burns, carbon dioxide retention, gastrointestinal bleeding, hypothermia, infections, drugs (amiodarone, anaesthetic drugs, beta blockers, diuretics, lithium, fenotiazine, rifampicin, tranquillisers), stroke, surgical interventions and trauma.[3-5] Hypoventilation plays a major role in the pathogenesis by leading to hypoxia and hypercapnea. Hypoglycaemia and dilutional hyponatremia also contribute to the development of myxedema coma.[6] In the current case, the reason for the development of myxedema coma was considered to be the unregulated glycaemic control and irregular use of the thyroid medication of the patient.

Findings which may accompany myxedema coma are typical hypothyroid symptoms, such as macroglossia, dry skin, slow reflexes, and unsightly oedema. In affected cases, hypoxia is seen in 80%, hypercapnea in 5%, and body temperature <34˚C in 88%[7]. Hypoventilation is the result of an insufficient ventilatory response to the suppressed hypoxic respiratory mechanism and hypercapnea. Oedema of the tongue and vocal cords can also be found to contribute to the table. Impaired respiratory muscle strength and obesity can worsen hypoventilation. Concomitant pleural effusion and/or the presence of acid causes a reduction in tidal volume. For all these reasons, the patient may require long-term mechanical ventilation.[8,9] In the currrent patient, there was seen to be dry skin, swollen eyelids, pleural effusion, pretibial oedema, respiratory problems and hypoxia.

In patients in myxedema coma, renal blood flow and the glomerular infiltration rate decrease, and total body volume increases. Bladder atony and urinary retention may be observed. High serum arginine vasopressin levels and disrupted free fluid clearance with reduced fluid flow to the distal nephron, create a predisposition to hyponatremia in the patient. A negative contribution to mental state may be found associated with the depth of hyponatremia. Urine sodium clearnace is normal or high, and urine osmolality is increased compared to plasma.

Creatinine phosphokinase levels may increase associated with rhabdomyolysis and acute renal failure may develop. An increase may be determined in anaemia, hypercholesterolemia and serum lactate dehydrogenase.[8,9] In the current case, hyponatrema was present in the acute renal failure table, together with a tendency to sleepiness, and co-operation was limited. With treatment, the cognitive state and co-operation of the patient returned to the previous levels.

Myxedema coma is a mortal table, and early diagnosis and rapid intervention are life-saving. Providing an open airway is the most important supportive element. Rapid intubation must be applied to cases with hypoxia, hypercapnea and respiratory acidosis problems and, especially to obese patients. The need for mechanical ventilation lasts 36-48 hours on average. Generally mechanical ventilation is required throughout the first 36 – 48 hours, but in some patients the need for ventilation support may continue for up to 2-3 weeks. It is recommended that mechanical ventilation is not terminated until full correction of consciousness. Ventilation sufficiency should be monitored with blood gases.[8,9] In the current case, there was no requirement for mechanical ventilation at any time. Hypoxia in this patient was corrected with oxygen from a nasal tube or mask at 2-4 lt/min. When TSH fell together with treatment, oxygen support via the nasal cannula was reduced and the patient was followed up in room air.

Initial treatment for myxedema coma is formed of high-dose thyroid hormone and glucocorticoid for possible adrenal insufficiency which may be concomitant and not previously diagnosed.[2] In the treatment of myxedema, blood samples are taken to determine cortisol, TSH and thyroid hormone before starting treatment, and if cortisol deficiency is suspected, 50-100 mg hydrocortisone or equivalent should be adminstered every 6-8 hours without waiting for the blood cortisol results. Nutrition is started with T4 300-500 mcg IV or from a nasogastric tube, and T4 50-100 mcg/day should be given by the same route until transfer to oral feeding. Triiodothyronine has a rapid effect, starting to increase the body temperature within a few hours, and can pass the blood brain barrier more quickly than T4.[10] In cases with a predominantly neurological table, it can be added to the treatment or can be given alone (at a dose of T3 10-20 µg IV bolus, followed by 10µg/day).[8] In the current patient, 80 mg methylprednisolone IV was administered for 1 week before thyroid replacement. During follow-up, the methylprednisolone dose was tapered and terminated. Levothyroxin sodium was started at 1 x 50mg PO and after 3 days was continued as 3 x 75 mg PO.

With a detailed anamnesis in ICU and laboratory tests, a diagnosis was made of myxedema coma, which although uncommon, can be fatal. Despite potentially fatal results, the patient was successfully treated with early diagnosis and treatment and adequate basic life support.

Informed consent: Informed consent forms were obtained from the patient and her relatives for tests and treatment of respiratory problems, and elevated blood pressure.

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