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Diagnostic Challenges of Myxoedema Coma in Geriatric Patients: A Case Report

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ABSTRACT

Hypothyroidism is a commonly diagnosed endocrine disorder in medicine. Myxoedema coma is a rare manifestation of hypothyroidism, and it can be lethal if it goes undiagnosed and untreated. Our patient presented with an acute manifestation of probable long-standing but undiagnosed hypothyroidism. He was symptomatic but did not complain by him. Stressful situations such as Infection are the usual precipitating factors for myxoedema coma. The patient responded well to treatment with levothyroxine, empirical antibiotic, and other general supportive measures.

Keywords: geriatric, myxoedema coma, thyroid hormone.

BACKGROUND

Myxoedema coma is the presentation of severe long-term hypothyroidism. The body can no longer compensate for the neurovascular system's homeostasis in this state. Based on data from the American College of Physicians in 1998, the total number of patients with severe hypothyroidism over 60 years in America is 2 percent of the entire elderly population. Moreover, patients over 60 with a history of polypharmacy make the interpretation of the thyroid physiology examination unclear. [1,2]

Myxoedema coma is a life-threatening emergency and challenging to recognize. Some patients with myxoedema coma come with many serious conditions, such as severe lung infections, sepsis, respiratory failure, and digestive disorders, so it becomes challenging to recognize and confuse other causes of coma. Until now, there are no criteria to determine when a patient with hypothyroidism will develop myxoedema. [1,2]

CASE ILLUSTRATION

An 85-year-old man complained of nausea and vomiting four days ago. One month earlier, the abdomen was enlarged and difficult to defecate. The patient also complained of coughing and shortness of breath, making it difficult to wake him up. According to the patient's family, the patient's hair has been falling for the last three weeks. The patient had a history of total thyroidectomy 12 years ago and routinely used levothyroxine daily. However, it has been discontinued for six months because there are no more complaints.

On examination, the patient's GCS (Glasgow Coma Scale) was E2V3M3. Vital signs, blood pressure 110/70 mmHg, pulse 90 bpm, axillary temperature 37.1'C, oxygen saturation with 12-liter non-rebreathing mask 94%. Physical examination of the eyes showed edema on both lids with good pupil reflexes and isochor; the face looks swollen, the skin is dry, and alopecia, redness, and dryness of the patient's oral mucosa. The apex of the heart is not visible; heart sounds are still normal. There are coarse rhonchi in the right lung. On abdominal examination,

the abdomen was distended, and no bowel sounds were heard. There is non-pitting edema on the hands. The patient has grade 1 four-extremity weakness, hypotonia, and hyporeflexia.

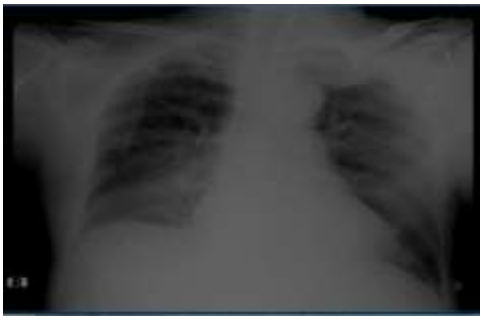

Figure 1. Thorax suggests pulmonary oedema, and pneumonia.

A full blood count suggests leucocytosis - patient with mild hypoalbumin. Blood gas analysis shows metabolic acidosis. ECG shows sinus bradycardia, low voltage QRS complex, and prolonged QT interval. Chest radiograph suggests pulmonary edema and pneumonia (Fig. 1.) Abdominal radiograph reveals a sentinel loop suggesting a paralytic ileus. Moreover, on the CT scan of the abdomen, colon dilatation and thickening of the connective tissue around the abdominal organs and mesentery were found (Figure 2).

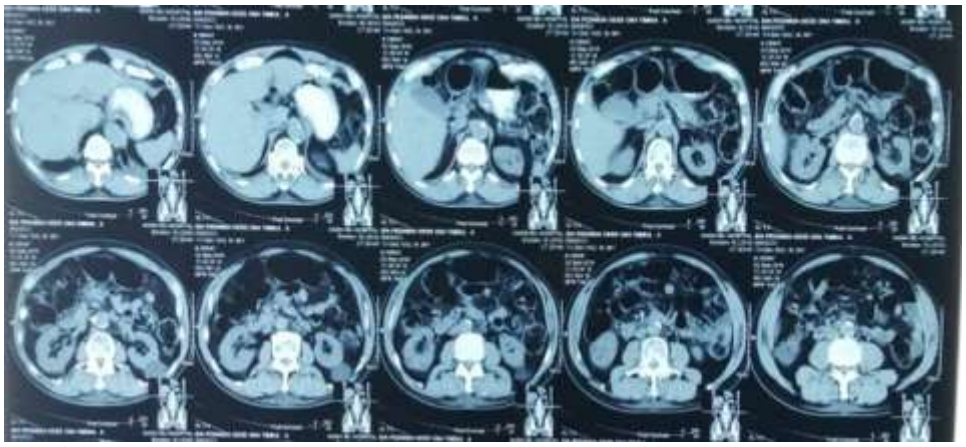

Figure 2. CT scan of the abdomen showed a dilatation of the colon and thickening of the connective tissue around the abdominal organs and mesentery

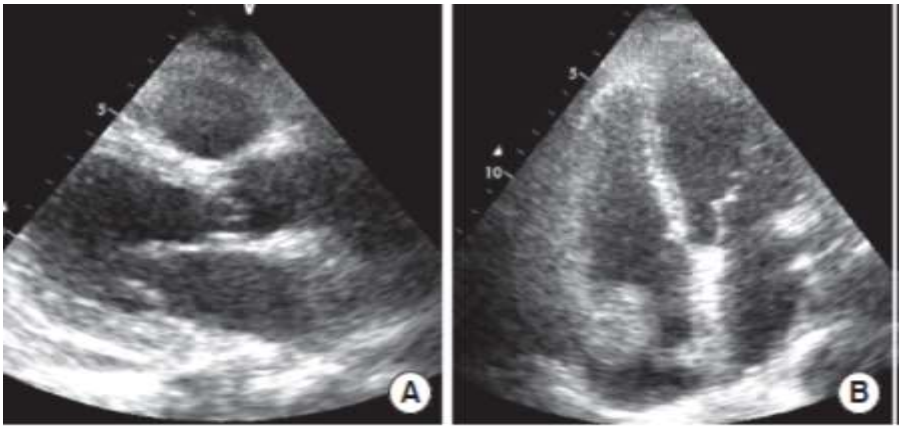

Figure 3. Echocardiography of the patient with parasternal long axis view (A) and para apical four chamber view. Demonstrates presence of pericardial effusion without hemodynamic compromise.

The patient was diagnosed with a myxoedema coma. The patient was complicated by aspiration pneumonia which caused sepsis shock, primary hypothyroidism, and related hypothyroid paralytic ileus. The patient was treated in the intensive care unit and placed on a ventilator. The patient was then given oral levothyroxine because intravenous levothyroxine is unavailable in Indonesia. With a loading dose of levothyroxine 400 micrograms, then continued with 100

micrograms every 24 hours. Aspiration pneumonia was given empirical antibiotics: Levofloxacin, Cefoperazone, and intravenous Metronidazole. Paralytic ileus is treated by placing a decompressed nasogastric tube. In its development, the patient worsened, experienced septic shock, and died.

DISCUSSION

Manifestations of hypothyroidism can vary from mild subclinical to severe degrees in the form of myxoedema coma. Myxoedema coma in the elderly is relatively high, around 0.22 per one million population. The high mortality rate is caused by old age, bradycardia, hypothermia, and other complications such as sepsis, respiratory infections, hypotension, and left heart failure.[6]

The risk factor for myxoedema is iodine deficiency. The patient had a history of total thyroidectomy ten years ago, but he was told not to take his medicine for the last two years. Guidelines for the 2017 ATA in patients over 60 with complaints of weakness and other nonspecific complaints should be screened for hypothyroidism by checking TSH and FT4 hormones. [5,6] Infection and sepsis are the most common precipitating factors for hypothyroidism, and the most common cause of infections are pneumonia, urinary tract infections, and cellulitis.[7]

The diagnosis of myxoedema coma is made on clinical grounds only and is supported by a thyroid hormone examination (Table 1). If a patient is in a stupor or coma, with dry and cold skin, hair falling out quickly, hoarseness and macroglossia, decreased tendon reflexes, and supported by hypothyroid hormone examination. Myxoedema coma can be suspected (Table 1).[7] Other important clinical can be hypoventilation, bradycardia, decreased cardiac contractility, decreased intestinal motility to paralytic ileus, and megacolon. On electrocardiographic examination, low voltage complexes, branch blocks, complete heart blocks, and nonspecific ST changes, bradycardia with prolonged QT will usually be found; in some cases, pericardial effusion can be found. [8,9] In this patient, we find almost all clinical signs.

Table 1. Scoring system for myxoedema coma diagnosis

Thermoregulatory dysfunction (temperature, °C)			**Cardiovascular dysfunction**	
>35		0	Bradycardia	
32-35		10	Absent	0
<32		20	50-59	10
Central nervous system effects			40-49	20
Absent		0	<40	30
Somnolent/lethargic		10	Other EKG changes[b]	10
Obtunded		15	Pericardial/pleural effusions	10
Stupor		20	Pulmonary edema	15
Coma/seizures		30	Cardiomegaly	15
Gastrointestinal findings			Hypotension	20
Anorexia/abdominal pain/constipation		5	Metabolic disturbances	
Decreased intestinal motility		15	Hyponatremia	10
Paralytic ileus		20	Hypoglycemia	10
Precipitating event			Hypoxemia	10
Absent		0	Hypercarbia	10
Present		10	Decrease in GFR	10

Abbreviations: EKG = electrocardiogram; GFR = glomerular filtration rate.
A score of 60 or higher is highly suggestive/diagnostic of myxoedema coma; a score of 25 to 59 is suggestive of risk for myxoedema coma, and a score below 25 is unlikely to indicate myxoedema coma.
Other EKG changes: QT prolongation, or low voltage complexes, or bundle branch blocks, or nonspecific ST-T changes, or heart blocks.

Hypothyroidism will get worse if there is an infection in the patient. There will be an overlap between a very severe infection, respiratory failure, and myxoedema, so it becomes more difficult to confirm the diagnosis.[5] Severe electrolyte disturbances and severe sepsis in patients with symptoms overlap and are difficult to distinguish. Nephrotic syndrome and heart failure are other conditions that can be confused with myxoedema.[6.7] However, the difference in edema is very noticeable. Edema in

hypothyroidism is non-pitting, and the main constituent of the edema is deposits of hyaluronic acid. Meanwhile, in heart failure and nephrotic syndrome, the main constituents of edema are water and sodium.[8,9]

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Thyroid hormones have systemic effects on almost all organs. Myxoedema on the central nervous system makes the patient go from coma, lethargy, and somnolence to cognitive and behavioral disorders and depression. The central nervous system disorders will usually be exacerbated by hyponatremia, which can cause seizures. CO2 narcotics will also further exacerbate disorders of the central nervous system. In addition, hypothyroidism also causes a decrease in metabolism and impaired glucose metabolism in nerve cells. [7,9]

Based on the ATA guidelines, the administration of levothyroxine to patients with myxoedema coma is given intravenously, and then thyroid hormone measurements are taken every two days.[6] With an initial dose of 200-400 micrograms or 1.6 micrograms/kg/day, then reduced by 75% the following day as a maintenance dose. Decreased motility causing levothyroxine absorption cannot occur properly, especially with hemodynamic disorders. There will be a decrease in blood flow to the digestive tract, further worsening the absorption of levothyroxine.[11]

Ventilation and cardiovascular support in patients treated in intensive care units with ventilators and vasopressors. Meanwhile, for paralytic ileus, the patient fasted, and a nasogastric tube was placed with the aim of decompression. Meanwhile, for the precipitating factors of Infection, patients are given empirical antibiotic therapy according to the pattern of germs at that time.[2]

CONCLUSION

Myxoedema coma is a rare event and has a high mortality, and it generally occurs in parents with a history of hypothyroidism before. The condition of myxoedema coma is very complex and requires comprehensive therapy. Thyroid hormone should be given immediately with a loading dose and maintenance dose, and Intravenous dosing is recommended. Cardiorespiratory support is essential. Moreover, handling precipitating factors becomes an integral part that must be considered in treating myxoedema coma.

Declaration by Authors

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