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A Rare Case of Myxoedema Coma Presenting With Seizures

Preethi CD*, Nethaji Sree Vaishnavi, Konda Sanjith, Anuradha, Viknesh Prabhu, KM Vignesh, Patel Harshvardhan Anilbhai

Department of General Medicine, Sree Balaji Medical College and Hospital
Bharat Institute of Higher Education and Research, Tamil Nadu, India

ABSTRACT

An elderly woman in her 60s presented with characteristic clinical features of myxoedema coma, which is a rare medical emergency case. Myxoedema crisis is a critical form of severe hypothyroidism which has a high mortality rate. The reason behind the condition is because of low intracellular T3, the patient may present with hypothermia or coma with cardiogenic shock or respiratory depression. These types of patients may also experience organ specific symptoms. If there is involvement of nervous system, seizure is the commonest symptom but a very rare manifestation of myxoedema and also carries a very high mortality rate. To intervene the progress of the disease an immediate diagnosis is required. This can be life saving for the patient and with appropriate management; the prognosis of the patient can also improve. Seizures may develop in a hypothyroid patient as a result of hyponatremia and during this presentation, if the patient is found to have hyponatremia it indicates that the patient has moderate to severe form of myxoedema. Initial treatment goal is to correct the patient's sodium level and followed by thyroid replacement therapy in intensive care unit along with steroids and fluid replacements.

Key words: Myxoedema coma, Hypothyroidism, Hyponatremia

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Corresponding author: Preethi CD

e-mail✉: preethicd007@gmail.com

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CASE PRESENTATION

A 67year old female had presented to the OPD with history of hypothyroidism and first episode of generalized seizure which lasted for few minutes. Patient had history of decreased food intake for the past 1 month. Patient has altered mental status, drowsiness, lethargy and irrelevant speech for the past 2 weeks and had no history of headache, neck rigidity or any injury to the head. There was no history of fever, no history of previous seizure disorder. Patient is a known hypothyroid for 38 years and is on levothyroxine 75mcg daily.

Examination

Blood pressure: 90/60 mmHg, pulse irregular, temperature: 35C. GCS 10/15 . Facial puffiness with peri-orbital swelling and macroglossia is present. Patient had dry skin. Examination of the neck showed no lymph-node enlargement or goiter or dysphagia. Cardiovascular system, respiratory system was normal. Plantar reflex was flexor. No history of fever, headache with vomiting or any neurological deficit. Patient consumes alcohol occasionally and is a non-smoker. No history of external injuries or trauma. No family history of seizures. Family history of cardiovascular disease is present.

Investigations

Blood reports: TSH-275.6 mU/L, T3 – 46.7, serum sodium 127, haemoglobin 10.7 g/dL, with macrocytosis, normal white cell count, and normal inflammatory markers. Anti TPO antibodies were negative. Patient's ACTH and Cortisol levels were measured to assess the functioning of adrenals and pituitary gland. ECG- low amplitude QRS, Prolonged QT, T wave inversion. Non-contrast CT was normal. EEG findings are nonspecific with slowing and decreased amplitude. CSF analysis was normal.

Hyponatremia and/or myxedema were taken into consideration as potential explanations for the patient's presentation, which included fits and drowsiness and lethargy. The level of hyponatremia in our patient was moderate (125–129 mmol/L). Though neurological symptoms are more common in severe hyponatremia, particularly when the Na+ is below 115 mmol/L, the patient was initially started with Na+ correction, by giving 3% NaCl in 100ml NS bolus. Patient's GCS improved. She was started on intravenous glucocorticoids

(hydrocortisone 100mg IV, followed by 50mg IV every 8h), loading dose of intravenous levothyroxine(400mcg IV) was given followed by daily IV doses of 50-100 mcg until the patient can take T4 orally ,which was after 48 hours. She improved symptomatically, her cognition and mental status improved. There was no episode of seizure again. Continuous monitoring of T3,T4, TSH was done. After 2 days TSH was 90 mU/L, sodium levels improved.

DISCUSSION

Myxoedema coma is a rare complication yet one of the severe manifestations of hypothyroidism. Women are more likely to develop myxoedema coma. Myxoedema coma can result from severe chronic hypothyroidism or it can be due to cold exposure, sedative use, infection, or stopping thyroid medications. This patient's precipitating factor may be due to irregular thyroid medications. The hallmark of myxoedema coma is decreased mental status, hypothermia, hypotension, bradycardia, hyponatremia, hypoglycemia and hypoventilation. In addition to these symptoms there might be multiple organ dysfunctions. Patients usually presents with the characteristic hypothyroidism signs and symptoms. These patients should be started immediately on thyroid replacements along with steroids and fluid replacement. Thyroid hormone replacement through ryle's tube is proven to be more effective in certain studies. Thyroid hormone usually works by lowering mean arterial pressure and decreasing peripheral artery resistance by directly affecting the smooth muscles of the arteries.

Seizures in myxedema can occur for a variety of reasons, but myxedema itself can trigger seizure activity. It is uncertain what triggers epileptic seizure activity in hypothyroidism. It might be brought on by cerebral edema or an increase in the volume of extracellular fluid. This may be connected to hyponatremia, hypoventilation, and post anoxic encephalopathy, which can all contribute to improper antidiuretic hormone (ADH) secretion and further trigger seizure activity. In this seizures may be due to concomitant hyponatremia [1-10].

SUMMARY AND CONCLUSION

Myxedema coma is an endocrine emergency which should be managed aggressively and immediately, since the mortality rate is very high. Advanced age, decreased GCS, need for mechanical ventilation, persistent hypothermia, sepsis are all predictors for mortality. So myxedema coma, though it is rare it should be considered while managing unconscious patients. Early recognition of hypothyroidism and appropriate medical management will reduce the risk of developing serious illness like sepsis and mental alteration and to advice continuation of thyroid medications may prevent significant morbidity and mortality.

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