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General

Intraoperative Recognition and Anesthetic Management of Myxedema Coma During Emergent Intertrochanteric Femur Fracture Repair

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Myxedema coma is a rare, life-threatening complication of severe, long-standing hypothyroidism, often precipitated by physiological stress such as infection, trauma, or surgery. Perioperative presentations of myxedema coma are especially uncommon. This case report describes a 71-year-old male with a history of poorly controlled hypothyroidism who underwent emergent surgical fixation of a traumatic left intertrochanteric femur fracture who developed intraoperative myxedema coma. Timely diagnosis and intervention—including intravenous thyroid hormone replacement, corticosteroids, hemodynamic support, and close anesthetic management were crucial to optimizing the patient's outcome. This report highlights the critical role of anesthetic management in myxedema coma cases, emphasizing the importance of endocrine and hemodynamic support. Furthermore, it promotes multidisciplinary coordination when managing endocrinological emergencies in high-risk surgical patients.

INTRODUCTION

Myxedema coma is a rare and life-threatening complication of severe thyroid hormone deficiency.[1-4] The clinical syndrome develops out of a long-standing history of undiagnosed or untreated hypothyroidism and an acute stress-associated precipitating event such as infection, trauma, or surgery. Myxedema coma is characterized by multi-organ dysfunction presenting as a myriad of clinical manifestations including neurological symptoms, hypothermia, bradycardia, respiratory depression, and renal impairment.[1,2] If left untreated, the syndrome has a reported mortality rate of as high as 60%.[1] While surgery has been described as a possible trigger for myxedema coma, the condition is particularly rare in the perioperative setting, with only a few cases reported. Rapid response from all members of the perioperative team is essential for positive outcomes. Here, we present a case of long-standing hypothyroidism with concern for acute onset myxedema coma intraoperatively and its anesthetic management.

CASE PRESENTATION

A 71-year-old male with a medical history of hypothyroidism, hypogonadism, hyperlipidemia, chronic kidney disease, tobacco use disorder, and anemia presented to the hospital overnight as a code 2 trauma after a mechanical fall. Our patient was 6', 57 kg, and had a body mass index of 17. The patient had a history of poorly controlled hypothyroidism since 2020 due to medication non-adherence. His preoperative heart rate, blood pressure, and O2 saturation were all within normal limits. He was found to have an intertrochanteric fracture of the left femur and was posted for emergent surgical fixation.

The patient underwent general anesthesia with endotracheal intubation in combination with a preinduction pericapsular nerve group (PENG) block and fascia iliaca compartment block. Following induction, the patient experienced hypotension, which was managed with and responded well to intravenous fluids, phenylephrine, and ephedrine boluses.

Intraoperatively, critical laboratory testing resulted in a thyroid-stimulating hormone (TSH) level of 66 uIU/mL (reference range 0.45 - 5.33 uIU/mL) and a free T4 (FT4) level of less than 0.25 ng/dl (reference range 0.64 – 1.42 ng/dl), consistent with primary hypothyroidism. At the time, the patient was hypotensive but responding appropriately with the measures above, so the decision was made to not abort the procedure. Per the recommendations of our medicine colleagues, a single dose of hydrocortisone 150 mg IV was administered for possible concomitant adrenal insufficiency.

As the case proceeded, the patient developed worsening hypothermia despite multiple warming measures, including nasopharyngeal and esophageal temperature monitoring, convection warming, warm blankets, an elevated ambient room temperature, and the use of multiple fluid warmers; the lowest recorded temperature was 93.5°F. In response to hypothermia and concern for myxedema coma, a 50 mcg

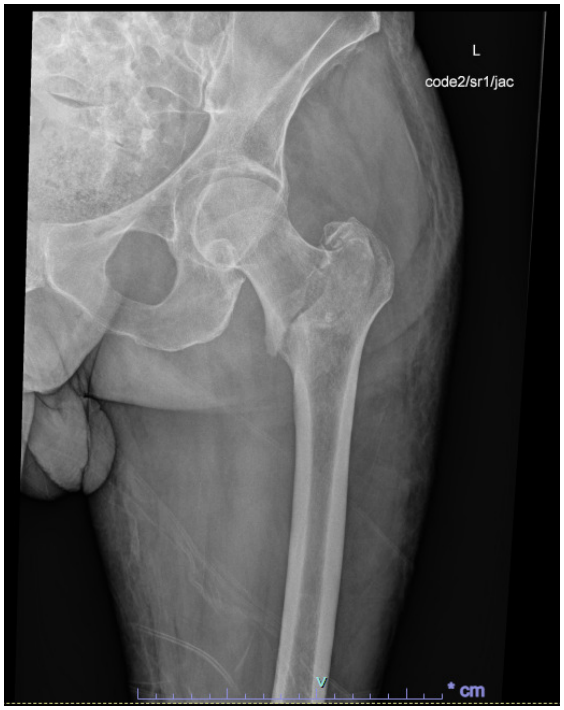

Figure 1. Displaced left femoral comminuted intertrochanteric fracture

of intravenous levothyroxine was administered intraoperatively.

Given ongoing blood loss and a starting hemoglobin of 8.0, the decision was made to transfuse one unit of packed red blood cells (pRBCs). Towards the end of the procedure, after multiple interventions, the patient stabilized and normothermia was achieved. The decision was made to attempt extubation given improved hemodynamics, no EKG changes, and stable temperature. The patient emerged from anesthesia and was extubated without complications. Pain was adequately controlled with pre-incision regional techniques. We limited narcotics as to not mask signs and symptoms of myxedema coma.

Postoperatively, he was continued on intravenous levothyroxine 50 mcg daily and hydrocortisone 50 mg IV every 6 hours for 24 hours, given the high risk for myxedema coma, with a calculated myxedema coma score of 40. Unfortunately, the patient was lost to follow-up after discharge on postoperative day 9.

DISCUSSION

Myxedema coma is a rare but life-threatening endocrine emergency resulting from undiagnosed, long-standing, and/or untreated hypothyroidism. It is characterized by multi-organ dysfunction, altered mentation, and hypothermia – often precipitated by stressors such as infection, trauma, cold exposure, or surgery.[1,2] In this case, myxedema coma was identified during the intraoperative period during emergent surgical fixation of an intertrochanteric femur fracture.

Fractures in elderly patients are associated with significant physiological stress and often require emergent surgical intervention. In hypothyroid patients, this stress can disrupt compensatory mechanisms and unmask or exacerbate latent metabolic and endocrine disturbances.[1,3] The patient's perioperative deterioration manifested as hypotension and hypothermia, along with their laboratory findings of an elevated TSH and critically low T4, raised suspicion for myxedema coma. The high mortality rate associated with this condition (up to 30–60%) mandates prompt recognition and intervention.[2]

Anesthetic management in myxedema coma presents several challenges. Low intracellular T3 leads to depressed inotropy and chronotropy with resultant vasoconstriction, often observed as hypotension in patients with myxedema coma. Use of anesthetic agents can further exacerbate hypotension through their vasodilatory effects and reduced myocardial contractility, leading to a decompensated state where low cardiac output and hypotension may result in cardiogenic shock.[4]

Additionally, reduced central nervous system sensitivity to hypoxia and hypercapnia can lead to decreased respiratory drive and respiratory failure. Additionally, the swelling of the nasopharynx and larynx can be observed as well and contribute to respiratory failure.[5]

Patients with severe hypothyroidism or myxedema coma may also exhibit heightened sensitivity to sedatives and opioids due to reduced hepatic metabolism and decreased cardiac output, increasing the risk of prolonged drug effects and respiratory failure.[6] Careful selection of anesthetic agents and close hemodynamic monitoring are essential.

In our case, hypotension was managed with vasopressor support, while hypothermia was corrected using active warming measures. Thyroid hormone replacement was initiated intraoperatively with intravenous levothyroxine, and corticosteroid therapy was added for suspicion of adrenal insufficiency, which may be unmasked when treatment with levothyroxine is initiated.[7] Endocrinology societies recommend quick replacement of thyroid hormone in suspected myxedema coma. The recommended intravenous dose is half the oral dose. Furthermore, thyroid replacement therapy should be carefully started in the elderly and those with cardiac disease due to risk for precipitating myocardial infraction and arrythmias.[8]

Post-operatively, the patient was able to be extubated and emerged from anesthesia without complications. However, in cases with continued organ dysfunction requiring prolonged mechanical ventilation or vasopressor support, intensive care management focused on continued hormonal replacement, supportive measures, and monitoring for complications such as arrhythmias and electrolyte imbalances is recommended.[9] As previously mentioned, careful titration of opioids is required as these patients are exquisitely sensitive to opioids.

The timely recognition of myxedema coma in the perioperative setting allowed for immediate intervention, which likely contributed to the favorable outcome in this case. This underscores the importance of maintaining a high index of suspicion in elderly patients with a history of hypothyroidism who develop hypotension, hypothermia, and/or bradycardia intraoperatively, especially when unrespon-

sive to conventional treatment. Early diagnosis, thyroid hormone replacement, supportive care, and close anesthetic monitoring are key to optimizing outcomes. Additionally, multidisciplinary coordination between anesthesiology, endocrinology, and surgery is critical in managing such complex cases effectively.

CONCLUSION

Myxedema coma is a rare, life-threatening endocrine emergency that can be precipitated by surgical stress in patients with undiagnosed, untreated, or poorly controlled hypothyroidism. Intraoperative myxedema coma is especially uncommon, and this case highlights prompt recognition and successful management of myxedema coma. Timely initiation of thyroid hormone replacement is essential to optimizing patient outcomes, as demonstrated in the present case. A multidisciplinary approach involving anesthesiology, surgery, and endocrinology are vital to recognizing and treating endocrine emergencies in high-risk surgical patients.

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