

Perioperative management of a patient with myxedema coma and septicemic shock

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Abstract

Myxedema coma is a life-threatening but uncommon complication of long-standing, neglected hypothyroidism. It was first reported by Ord in 1879. Till date only around 200 cases have been reported in literature. The incidence in European countries is 0.22 per million per year. No epidemiological data is available from the Indian subcontinent. We are reporting the case of an elderly lady who went into life-threatening myxedema coma along with septicemic shock, and was successfully treated with oral thyroxine.

Key words: Inotropes, myxedema coma, parenteral elroxine, septicemia, shock, thyroxine

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Introduction

Myxedema coma is a rare life-threatening complication of long-standing, neglected hypothyroidism. Till date only around 200 cases have been reported. We are reporting the case of an elderly lady with life-threatening myxedema coma and septicemic shock who was successfully treated with oral thyroxine.

Case Report

A 61-year-old lady, known case of hypothyroidism for 20 years, was admitted in surgical emergency with complaints of abdominal pain and distension for 3 days. She had no recent thyroid function test (TFT) reports.

On examination, she was found to be drowsy but arousable. Her pulse rate (PR) was 68/min., blood pressure (BP) 140/86 mmHg, temperature 36.5°C, respiratory rate (RR) 10/min. Her investigations were Hb -8.8 gm%, total leucocyte count -12,400/mm³, serum

Na⁺-112 meq/l (normal range 135-145 meq/l), serum K⁺-2.2 meq/l (normal range 3.5-5.5 meq/l). Her liver function tests, kidney function tests and chest X-ray were normal and ECG showed low voltage complexes. A sample for TFT was immediately sent. Abdominal ultrasound revealed a mass in right iliac fossa suggesting appendicular lump along with paralytic ileus. She was started on conservative management along with correction of electrolytes and active rewarming. Meanwhile her TFT showed severe hypothyroidism with T3 being 62 ng/dL (normal range 100-180 ng/dL), T4-2.2 µg/dL (normal range 4-12 µg/dL) and TSH-18 µU/ml (normal range 0.5-6 µU/ml). Her dose of eltroxin was elevated to 100 µg/day.

Her abdominal distension increased over the next 48 hours and a contrast-enhanced CT scan of abdomen revealed intestinal perforation. She was immediately taken up for laparotomy. Preoperatively, the patient was still drowsy but stable hemodynamically.

General anesthesia was induced using rapid sequence induction. Immediately after induction, she had systolic hypotension (85 mmHg), managed with fast crystalloids. There was a further drop in her blood pressure after the abdomen was opened during surgery, necessitating dopamine infusion along with noradrenaline infusion. Her entire colon was found to be gangrenous and

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extended right hemicolectomy was done. Intravenous steroids were given. Surgery lasted for 2 hours and she was shifted to ICU for elective ventilation

When received in ICU, patient was hypothermic (temperature 34.6°C) and hypotensive (systolic BP-68 mmHg). Rewarming was started with of warm fluids, bladder lavage with warm saline and electric blanket. Three litres of lactated ringer's solution was pushed intravenously. Adrenaline infusion was now added. This managed to maintain her systolic BP to around 85 mmHg. Unfortunately parental thyroxine was not available so we decided to give eltroxine tablet (500 µg) once daily via Ryle's tube. This along with aggressive antibiotic coverage, steroid supplementation and inotropic support gradually improved her blood pressure over the next 24 hours. Her TFT also showed marked improvement after 3 days. Her eltroxine was titrated to 150 µg twice a day now.

Over the next 2 days, her inotropic support was gradually tapered off and stopped. Her eltroxin dose was also decreased to 100 µg twice a day. Finally on postoperative day 5, she was extubated and shifted to the postoperative ward.

Discussion

Myxedema coma is an extreme complication of hypothyroidism in which patients exhibit multiple organ abnormalities and progressive mental deterioration. It occurs almost exclusively in age group 60 years and above, with 80% preponderance in females, as seen in our patient.^[1,2]

More than 80% of cases of myxedema coma occur in winters, probably due to age-related loss of ability to sense temperature and lower production secondary to hypothyroidism.^[1] Other events which can precipitate it include infections, cardiovascular accidents, congestive cardiac failure and certain drugs.^[3,4] We presume that in our patient the precipitating factors included winter season, septicemia and stress of surgery and anesthesia.

Myxedema coma causes drastic decrease in metabolic rate, hypoventilation, hypotension, hypothermia, decreased mental state progressing to coma and decreased cardiac output.^[5]

A common misconception is that a patient must be comatose to be diagnosed with myxedema coma. However, most patients exhibit neither edema nor coma. Instead the cardinal manifestation is a deterioration of the patient's mental status.^[6,7]

It is said that long-standing hypothyroid patients have associated hypopituitarism.^[3] There have been reports of myxedema coma with coexisting adrenal crisis.^[8,9] Preoperative cortisol level determination along with perioperative steroid supplementation is advised. Although we could not get cortisol level determined, empirical steroid supplementation aided in the recovery of our patient.

Mortality in untreated or unrecognized myxedema coma reaches nearly 100%. Even with optimum therapy, mortality rate as high as 30-60% has been reported.^[10,11] Dutta *et al.* did a study to determine the various predictors of outcome in myxedema coma and concluded that various factors associated with increased mortality included bradycardia, need for mechanical ventilation, hypothermia, sepsis, hypotension and intake of sedative drugs.^[12] In our patient most of these factors were present but were managed aggressively and judiciously. In literature, intravenous thyroid hormone supplementation has been advised for the treatment of myxedema coma but since it was not available, early supplementation of a higher dose (500 µg/day) of oral thyroxine was started via the Ryle's tube. Suspicion and empirical treatment of adrenocortical suppression prevented further deterioration of our patient. The good outcome of our patient despite severe hypothyroidism and septicemic shock with gangrene occurred most probably due to the fact that she was already receiving eltroxine, though suboptimal dose and our aggressive inotropic therapy, fluid management and multiple antibiotics.

To conclude, the combination of myxedema coma and sepsis in a patient can be lethal. Early institution of thyroxine supplementation, even via oral route, along with inotropic support and steroid supplementation, can improve the prognosis.

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