CASE REPORT

Postoperative myxoedema coma

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SUMMARY

Hypothyroidism is a commonly diagnosed endocrine disorder in medicine. Hyponatraemia is reported in up to 10% of hypothyroid patients, although it is usually mild and rarely causes symptoms. 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. She was asymptomatic but when subjected to surgery (identifiable stress factor) it precipitated as myxoedema coma. Stressful situations such as surgery or infection are the usual precipitating factors for myxoedema coma. The patient responded well to treatment with levothyroxin, 0.9% saline infusion and other general supportive measures. Judicious use of intravenous saline to correct hyponatraemia is important. Correction with 3% normal saline should be carried out with great caution, as rapid correction with 3% normal saline can lead to central pontine demyelination.

BACKGROUND

This case highlights the importance of taking a complete history and performing a thorough physical examination of the patient.¹

A thorough history and examination prior to the elective surgery could have revealed the hypothyroidism and avoided the resulting complications.

CASE PRESENTATION

The patient was a 73-year-old woman presented to surgical outpatient with pain in the right hypochondrium, low-grade fever, bloating and nausea intermittently for 5–6 months.

She correlated these symptoms especially on eating fatty, fried or greasy foods.

A general physical examination revealed an elderly obese woman.

The patient had a pulse rate of 60/min, which was regular and of normal volume. Her blood pressure was 100/62 mm Hg in a supine position, there was no postural fall in blood pressure and respiratory rate was 18–20/min and her temperature was 37.5°C.

Systemic examination: abdominal examination revealed a soft abdomen. Bowel sounds were present. There was no area of tenderness, guarding or rigidity. There was no palpable organomegaly.

Baseline preoperative tests were conducted which were as follows:

Full blood count and renal functions were normal. ECG showed sinus bradycardia and low-voltage complexes. There was no ST segment deviation.

Chest X-ray was within normal limits.

Abdominal ultrasound revealed hyperechoic shadows suggestive of cholelithiasis with multiple

gallstones of varying sizes. The patient was diagnosed as having cholelithiasis and advised to undergo a laparoscopic cholecystectomy.

The patient underwent the elective cholecystectomy under general anaesthesia. Propofol was used as the anaesthetic medication, along with injection pentazocine 15 mg intramuscularly and injection promethazine 25 mg intramuscularly as preanaesthetic medication.

Laparoscopic cholecystectomy was performed. Throughout the surgery, the patient remained haemodynamically stable, but after the surgery the patient did not regain consciousness.

The patient was extubated after she was evaluated by an anaesthetist and found fit to be extubated, and she was given oxygen supplementation via the venturi mask at 1 L/min and was shifted to the recovery ward.

Postoperative blood tests were conducted and the reports were as below.

Postoperative laboratory tests revealed severe hyponatraemia.

- Full blood count and renal functions were normal except for the sodium levels which were low.
- Her serum sodium was 120 mmol/L (normal 135–145 mmol/L; [tables 1 and 2](#)).
- Blood culture and urine culture were sent.
- Arterial blood gas revealed a pH of 7.33 (normal 7.35–7.45), pO₂ 86 mm Hg (normal 80–100 mm Hg) and hypercarbia with pCO₂ 60.2 mm Hg (normal 35–45 mm Hg).
- ECG showed sinus bradycardia, low-voltage complexes and T wave inversion in all leads. However, there was no ST segment deviation.
- Brain MRI and EEG were normal.

The patient was started on intravenous 0.9% saline infusion at 100 mL/h flow rate to correct hyponatraemia, but despite the saline infusion, hyponatraemia worsened and serum sodium dropped to 101 mmol/L in the next 24 h.

It was then that a medical consult was sought.

A thorough history was taken again from the patient's daughter with whom the patient lived, and bedside examination was performed. The patient's daughter did reveal that historically her mother had gained weight gradually, felt lethargic and sad quite often and she also had started developing pedal oedema in both feet recently.

There was, however, no history of craving for salt or chronic, steadily worsening fatigue, loss of appetite, diarrhoea or weight loss. There was no history of progressive darkening of skin or syncope. Historically, the patient did not take any medications like diuretics, serrtraline or lithium which could cause hyponatraemia.

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To cite: James R, James J, Vij AS, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-010066

Table 1 Patient's reports before medical consult was sought

22.12.2011, time	Blood urea,mg/dL (20–40)	Creatinine, mg/dL (0.5–1.4)	Sodium, mmol/L (135–145)	Potassium, mmol/L (3.5–5.5)	Chloride, mmol/L (95–107)
Preoperative 9:00	35	0.9	135	4.70	102
Postoperative 10:00	36	0.8	120	4.45	103
14:00	32	0.8	112	4.76	101
18:00	35	0.8	101	4.48	102

A general physical examination in recovery room revealed an elderly woman in coma. She was obese and had dry coarse skin and pretibial non-pitting pedal oedema. However, there were no signs of chronic Addison's disease. There was no pallor, cyanosis, icterus, clubbing and koilonychia. There was no palpable lymphadenopathy.

Examination of the neck revealed a small goitre 4×5 cm size, which moved with spontaneous swallowing movements which the patient experienced occasionally. Pemberton's sign was negative. The Pemberton manoeuvre is a very useful part of bedside examination, especially when one is evaluating neck swellings like retrosternal goitre or large mediastinal mass or tumour.²

Postoperative vital signs were as follows: She had a pulse rate of 58/min, which was feeble, of low volume and regular, and blood pressure was 98/60 mm Hg in a supine position; postural fall in blood pressure was not checked as the patient was in coma and in hypotension. Her respiratory rate was 16–18/min and temperature was 35°C (core rectal).

Respiratory, abdominal and cardiovascular examinations were normal except for the bradycardia and hypotension.

Central nervous system examination: the patient was unresponsive; limited cranial nerve assessment did not show any obvious abnormality in this clinical situation. Sensory and motor system examination was not performed as the patient was non-responsive. Reflexes revealed a sluggish knee jerk. Planters were bilaterally flexors. There were no signs of meningeal irritation.

In view of the above clinical findings, the possible diagnoses of hypothyroidism and adrenal insufficiency were considered. When subjected to stress, both these conditions can cause rapid deterioration in the clinical state of the patient as there was a poor response to stressful situations.

Table 2 Patient's reports after levothyroxine and hydrocortisone were started

Date, time	Sodium, mmol/L (135–145)	Potassium, mmol/L (3.5–5.5)
Day 1, postoperative 18:00	101	4.48
Day 1, postoperative 12:00	103	4.80
Day 1, postoperative 18:00	105	4.78
Day 1, postoperative 00:00	107	4.02
Day 2, postoperative 6:00	111	4.28
Day 2, postoperative 00:00	113	4.48
Day 2, postoperative 18:00	115	4.31
Day 2, postoperative 12:00	119	4.44
Day 3, postoperative 6:00	123	4.38
Day 3, postoperative 00:00	128	4.18
Day 3, postoperative 00:00	135	4.41
Day 3, postoperative 00:00	140	4.40

INVESTIGATIONS

Laboratory findings (postoperative) were as follows:

Full blood count and renal functions were normal except for the sodium levels (tables 1 and 2).

Serum osmolality was 216 mOsm/kg (280–300 mOsm/kg) and urine osmolality 812 mOsm/kg (50–1200 mOsm/kg) and urine sodium level 95.02 mmol/L (25–220 mmol/L).

A repeated ECG showed sinus bradycardia, low-voltage complexes and T wave inversion. There was no ST segment deviation.

Cardiac enzymes including Trop T spot and CPK-MB (2 sets at 6 h interval) were analysed and they were within the normal range.

Brain MRI and EEG were normal.

Short synacthen test (SST) was performed and it did not reveal any abnormality. The basal value was 278 nmol/L and the post-SST was 499 nmol/L; since the increment was more than 200 it was considered normal.

The presence of a Coexisting adrenal insufficiency was ruled out by a repeat SST performed 12 weeks after stopping the steroids, and it did not reveal hypocortisolism.

DIFFERENTIAL DIAGNOSIS

In view of the clinical and laboratory findings, the diagnosis of myxoedema coma was considered.

Acute adrenal crisis was also considered initially, but SST was performed and it did not reveal hypocortisolism.

TREATMENT

Diagnosis of myxoedema coma was made and medical treatment was started. The patient was warmed gradually and oxygen was supplied via a venturi mask.

Injection levothyroxine 600 µg intravenously was given as a start dose followed by tab levothyroxine 125 µg once daily via a nasogastric tube.

Injection hydrocortisone 200 mg bolus intravenously was given followed by 100 mg Q6H intravenously for 2 days and then decreased to 50 mg Q6H intravenously for the third day. (Injection hydrocortisone was given as part of treatment of myxoedema coma.) It was stopped on the fourth day as the patient improved.

Also 0.9% saline infusion at 100 mL/h was continued; the infusion rate was adjusted as per central venous pressure.

Injection cefuroxime+sulbactam 3 g Q12H intravenously was started empirically as subtle occult infections can also precipitate myxoedema coma and/or acute adrenal crisis in susceptible patients.

The patient's clinical condition started improving soon after starting the treatment with levothyroxine and 0.9% saline infusion (table 3).

Hypothermia improved gradually; heart rate increased to 88–90 bpm and her blood pressure increased to 130/80 mm Hg.

Table 3 Serial thyroid function tests

Date, time	ft4 (ng/L) (0.8–1.8)	TSH (μIU/mL) (0.5–4.70)
Day 1: 08:00, postoperative	0.1	145
Day 4: 08:00, postoperative	0.2	124
Day 6: 08:00, postoperative	0.2	79
Follow-up in medical outpatient 12 weeks after discharge	0.3	3

TSH, thyroid stimulating hormone.

Over the next 4 days, as hyponatraemia was corrected, the patient's sensorium improved gradually. On the fifth day of therapy, the patient was fully conscious and her serum sodium was within the normal range. The patient was then started on liquid oral feeds and in 1 week time she was discharged in a satisfactory condition.

OUTCOME AND FOLLOW-UP

The patient was diagnosed with myxoedema coma and treated appropriately. She was discharged without any residual deficit and is now being managed successfully for hypothyroidism in medical outpatient unit.

On follow-up, a repeat SST was performed 12 weeks after stopping the steroids and it did not reveal any evidence to suggest hypocortisolism.

DISCUSSION

Hypothyroidism is one of the common endocrine disorders encountered in general medical outpatient unit. Patients with hypothyroidism may be asymptomatic or they may present with symptoms such as fatigue, lethargy, hypotension, excessive sleepiness, depression, anxiety, hair loss, cardiac failure, hyponatraemia, obesity, sexual dysfunction, infertility, or, as in the present case, they may present with myxoedema coma.¹

Myxoedema coma is an extreme manifestation of hypothyroidism and can be potentially lethal if it goes undiagnosed and untreated. Patients with myxoedema coma usually have long-standing hypothyroidism, though it may not have been previously diagnosed, as in our patient's case where surgery led to decompensation. Myxoedema coma occurs almost exclusively in persons aged 60 years and older with female preponderance.³

Hyponatraemia is not an infrequent occurrence in hypothyroidism.^{4–7} Thyroid function should be assessed in all patients presenting with unexplained hyponatraemia. If hyponatraemia is due to hypothyroidism, then addition of levothyroxin to sodium replacement therapy will correct the hyponatraemia rapidly.⁸

From the clinical profile of this patient, it was clear that she had a long-standing hypothyroidism but it was noticed when the patient was subjected to a stressful situation (surgery in this case), which led to severe hyponatraemia, which further led to myxoedema coma. The patient fully recovered on successful diagnosis and treatment with levothyroxin, 0.9% saline infusion and other general supportive measures. Hyponatraemia is frequently observed in hospitalised patients. Stressful situations such as surgery or infection are the usual precipitating factors. In case of our patient, it became evident that the coma was because of severe hyponatraemia, which in turn was due to hypothyroidism. The mechanism by which hypothyroidism induces hyponatraemia, although it was suggested to be its inability to suppress anti-diuretic hormone in normovolemic patients, is not fully clear.⁷ It may be due to reduced cardiac output, which can lead to the

release of ADH via the carotid sinus baroreceptors.^{4 5 9} Further, the glomerular filtration rate is also low in hypothyroidism. This can directly diminish free water excretion by diminishing water delivery to the diluting segments.^{6 7} This reduced delivery of water may be particularly important in those cases in which hyponatraemia develops in spite of appropriate suppression of ADH release.^{10 11} Irrespective of the mechanism, impairment in water excretion causes water retention which causes hyponatraemia by dilution. Restoration of normal water balance and sodium levels can be achieved by administering the thyroid hormone.⁸

Judicious use of intravenous saline to correct hyponatraemia is extremely important. This will also help prevent iatrogenic complications. Rapid overcorrection of hyponatraemia with 3% saline should be avoided as it can cause central pontine myelinolysis. It occurs inconsistently as a complication of severe and prolonged hyponatraemia, particularly when corrected too rapidly.¹²

To avoid such fatal iatrogenic complications, the correction rate should be 6–8 mmol/L in 24 h, 12–14 mmol/L in 48 h and 14–16 mmol/L in 72 h.¹³

Learning points

- ▶ More emphasis should be given to history taking and physical examination of patients. They give clues as to what abnormality one may find later in laboratory examination; thus, these practices may aid in preventing unforeseen complications.
- ▶ Given the original symptoms as stated in the case presentation, it was appropriate to arrange an abdominal ultrasound but should not have detracted from considering a list of differential diagnoses.
- ▶ It would be worth to consider hypothyroidism in patients with unexplained hyponatraemia. If left untreated, hyponatraemia can be life threatening and it should be corrected carefully.
- ▶ Possibility of myxoedema coma should be considered in a setting of hyponatraemia with coma.
- ▶ Supportive treatments, such as oxygen supplementation and warming the patient, are important contributors to a successful positive outcome.

Acknowledgements We thank the patient and all fellow doctors who contributed to this case report. We also thank Wilhelmina Masih, Monika and Andrew Dering for their help in giving this manuscript the final shape.

Contributors All the authors contributed to the submission in the form of editing, guidance and also approved the submission.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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