CASE REPORT

MYXEDEMA COMA

Atif Munir
Diabetes and Endocrinology, University Hospital of Hartlepool-UK

Myxedema may be the first presentation of patients with undiagnosed hypothyroidism. Definitive management is with thyroid hormone but supportive measures, identification and treatment of precipitating factors in an appropriately safe environment are vital. There is no consensus about preferred thyroid hormone regimen. Corticosteroid therapy is given until adrenal insufficiency has been excluded. We present here a case of seventy-four years old woman of myxodema coma
Keywords: Myxedema; Hypothyroidism; Precipitating factors; Adrenal insufficiency.

Citation: Munir A. Myxedema coma. J Ayub Med Coll Abbottabad 2018;30(1):119–20.

INTRODUCTION

Myxedema coma has a mortality rate of about 40 percent.[1] It is now a rare presentation of hypothyroidism due to early diagnosis. Early recognition and therapy are essential. Myxedema coma can occur on a background of long-standing undiagnosed hypothyroidism or be precipitated by an acute event such as infection, myocardial infarction or the administration of sedatives as in our case.

CASE REPORT

A seventy four year old woman presented with acute confusion and visual hallucinations. Clinically she had hypothyroid features. No previous thyroid function tests were available, therefore were checked on admission. A presumptive clinical diagnosis of acute delirium secondary to a probable urinary tract infection was made and she was commenced on antibiotics. She required sedation with midazolam in view of rapid worsening of agitation over the first few hours after admission. Over next few hours she deteriorated becoming hypothermic, bradycardic, hypotensive, developed type 2 respiratory failure and dropped her glassgow coma scale requiring transfer to intensive care unit. Thyroid profile results came back the next day and revealed overt primary hypothyroidism (TSH >100 mU/L (0.2–0.4), Free T4 <4 pmol/L (7.8–14.4), midnight cortisol 654 nmol/L). Possibility of myxedema coma precipitated by urinary tract infection and midazolam was considered.

She required intermittent non invasive ventilation and fluid support. Thyroid hormone replacement was initiated in the form of intravenous liothyronine (T3) with steroid cover. Recovery was gradual over the next few days. Thyroid hormone replacement was switched to levothyroxine through nasogastric tube and then to oral route subsequently. Steroids were gradually weaned off however after initial recovery she subsequently succumbed to sepsis and multiorgan failure secondary to pneumonia.

DISCUSSION

The demographics of patients who develop this condition are those of hypothyroidism in general, with older women being most often affected. Commonest cause is chronic autoimmune thyroiditis. The hallmarks of myxedema coma are decreased mental status and hypothermia, but hypotension, bradycardia, hyponatraemia, hypoglycaemia, and hypoventilation are often present as well. Clinical signs of hypothyroidism are usually present. Despite the name, myxedema coma, patients frequently do not present in coma, but with lesser degrees of altered consciousness. The diagnosis is initially based upon the history, physical examination, and exclusion of other causes of coma and should be considered in any patient with coma or depressed mental status that also has hypothermia, bradycardia and/or hypercapnia.[2,3]

Before specific therapy is initiated blood should be drawn for measurements of serum thyrotropin (TSH), free T4, free T3 and cortisol. Ideally a short synacthen test should be performed to exclude adrenal insufficiency. Most patients with myxedema coma have primary hypothyroidism.

It is an endocrine emergency and should be treated aggressively. Treatment consists of thyroid hormone, supportive measures, and appropriate management of coexisting problems such as infection. Until coexisting adrenal failure has been excluded, the patient must be treated with glucocorticoids as replacing thyroid hormone in a hypo adrenal patient would worsen the clinical state.

There is no consensus about the preferred thyroid hormone regimen. Some experts favour administration of T3, some of T4 while others prefer a combination. Intravenous administration is recommended because gastrointestinal absorption may be impaired.[4,5] Alternate route of administration is through nasogastric tube. Supportive measures are vital for survival of the patient. These include treatment and monitoring in an intensive care unit, mechanical ventilation if

required, judicious maintenance of fluid balance, electrolytes and glucose, correction of hypothermia, avoiding and treating any possible precipitants.

REFERENCES

1. Reinhardt W, Mann K. Incidence, clinical picture, and treatment of hypothyroid coma: results of a survey. Med Klin 1997;92(9):521–4.
2. Zwillich CW, Pierson DJ, Hofeldt FD, Lufkin EG, Weil JV. Ventilatory control in myxedema and hypothyroidism. N Engl J Med 1975;292(13):662–5.
3. Klein I. Thyroid hormone and the cardiovascular system. Am J Med 1990; 88(6):631–7.
4. MacKerrow SD, Osborn LA, Levy H, Eaton RP, Economou P. Myxedema-associated cardiogenic shock treated with intravenous triiodothyronine. Ann Intern Med 1992;117(12):1014–5.
5. Arlot S, Debussche X, Lalau JD, Mesmacque A, Tolani M, Quichaud J, *et al*. Myxoedema coma: response of thyroid hormones with oral and intravenous high-dose L-thyroxine treatment. Intensive Care Med 1991;17(1):16–8.

Received: 11 June, 2016	*Revised: 12 October, 2017*	*Accepted: 23 October, 2017*

Address for Correspondence:
Dr Atif Munir, 7-Burghley drive, Ingleby Barwick, Stockton on Tees-UK. TS175GN
Contact: +47767235182
Email: atif113_2000@yahoo.co.uk