

Myxoedema Coma Masquerading as Acute Stroke

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Doi: 10.12890/2020_001563 - European Journal of Case Reports in Internal Medicine - © EFIM 2020

Received: 15/03/2020

Accepted: 19/03/2020

Published: 22/04/2020

How to cite this article: Butter C, Rashid N, Banatwalla R, FitzGerald T. Myxoedema coma masquerading as acute stroke. *EJCRIM* 2020;7: doi:10.12890/2020_001563.

Conflicts of Interests: The Authors declare that there are no competing interests.

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ABSTRACT

This report describes the management of a patient with myxoedema coma masquerading as an acute stroke (with or without 'cold sepsis'). Myxoedema coma is an endocrine emergency occurring when physiological adaptations to untreated hypothyroidism are overwhelmed by an acute precipitant. Even promptly treated, it has an associated mortality of up to 50%.

LEARNING POINTS

- Early recognition of myxoedema coma is essential, however the chance of misdiagnosis remains high.
- Key management consists of rapid thyroid hormone replacement (intravenous T4 at 300–500 µg over 24 hours, followed by 50–100 µg daily), supportive measures and the concomitant management of triggers such as infection.

KEYWORDS

Myxoedema coma, thyroid emergencies, hypothyroidism

CASE PRESENTATION

A 79-year-old woman presented to the Emergency Department with slurred speech and arm weakness in the context of deteriorating mobility, cognition and oral intake for 3 months.

She was not taking regular medications and her only medical history consisted of radioactive iodine treatment for hyperthyroidism in 1990 and subsequent iatrogenic hypothyroidism. The patient had chosen to stop taking levothyroxine in 2010.

On arrival she had a temperature of 33.3°C, a heart rate of 45 bpm and a blood pressure of 170/99 mmHg. Her oxygen saturations were 90% on room air. Her GCS was 9/15. She had dry flaky skin, coarse hair, frontal balding, globally depressed reflexes, a hoarse voice and peripheral oedema. There was no palpable goitre. A CT scan of the head demonstrated focal right cerebellar calcification of questionable clinical significance, but otherwise no acute pathology to explain her stupor.

Blood tests demonstrated Na⁺ 127 mmol/l (133–146), CRP 19 mg/l (<10), creatine kinase (CK) 586 IU/l (25–200), TSH 51 mU/l (0.35–5.0), T4 2.6 pmol/l (9–24), T3 <0.8 pmol/l (3.5–6.5) and cortisol 1,001 nmol/l (140–690).

Re-warming was initiated alongside the administration of broad-spectrum intravenous antibiotics for possible aspiration pneumonia. The patient was later commenced on combination treatment of T4 25 µg via the nasogastric (NG) route and 10 µg IV T3 replacement three times a day (due to concerns about poor absorption). This was initially combined with regular hydrocortisone as adrenal insufficiency often coexists.

Although no organ support was required, the patient was taken to the critical care unit for close monitoring as treatment risks arrhythmia and myocardial infarction.

Unfortunately, the patient passed away after a 28-day admission, which was complicated by recurrent hospital-acquired pneumonias, non-ST-elevation myocardial infarction (NSTEMI), a subsequent per rectum bleed, and raised CK secondary to thyroid myositis.

DISCUSSION

Myxoedema coma is an endocrine emergency which presents when a patient with longstanding untreated hypothyroidism is no longer able to maintain physiological adaptations to profound hypothyroidism due to an acute precipitant.

The term is somewhat of a misnomer as patients may be more obtunded than comatose. Myxoedema coma occurs more commonly in females (80%), almost exclusively in the over 60s, and 90% of cases occur in winter^[1]. This is because common causes of decompensation are hypothermia and infection^[2]. Other precipitants are medications, surgery, GI haemorrhage and myocardial infarction^[3].

Given the rare nature of this presentation, the chance of misdiagnosis is high. This case was a prime example, with the differential diagnosis before medical admission being stroke and 'cold' sepsis with no consideration of thyroid dysfunction.

Presentation

Dula et al. studied 23 patients with myxoedema coma and noted that 61% were known to have hypothyroidism at initial presentation, with sepsis the most common precipitating factor for myxoedema coma^[4].

Diagnosis is clinic and biochemical. In addition to low T4, T3 and elevated TSH levels, other abnormalities commonly include renal impairment, hyponatremia, hypoglycaemia and raised CK^[5-7].

The three main presenting features are altered mental state, hypothermia and the presence of a precipitating event^[2]. Other signs may include coarse dry hair/alopecia, dry skin, generalised oedema, macroglossia, bradycardia and delayed reflexes^[6,7].

Management

Despite the first recorded case of myxoedema coma being in 1879, there are still different schools of thought regarding the best method of thyroid hormone replacement^[7]. The majority of evidence supports T4 replacement as monotherapy, with relatively few adverse events when compared with T3 replacement^[8]. Recent data support thyroid hormone replacement with IV T4 at 200–400 µg, followed by 100 µg IV daily. Oral thyroxine can be given safely in similar doses, however absorption may be impaired due to complications such as constipation/paralytic ileus, although this has been disputed^[4].

In this case study, combination treatment with intravenous T3 (instead of T4) was used due to stock availability, with T4 given via the NG route. Evidence supports intravenous T3 due to the theoretical impairment of T4-to-T3 conversion in severe illness^[3,9,10]. However, T3 therapy is more expensive, with evidence that excess T3 is associated with cardiac side effects^[3,6,10]. Suggested advantages of T3 include faster onset with early improvement in neuropsychiatric symptoms^[8].

Intravenous T3 may be given at doses of 10–20 µg 8-hourly for the first 24 hours, followed by 2.5–10 µg 8-hourly until the patient is alert enough for it to be continued via the oral route. Lower doses should be used in the elderly and those with ischaemic heart disease^[12].

Hydrocortisone (50–100 mg IV three times a day) should be given until coexistent adrenal insufficiency is excluded, and because cortisol clearance may be increased in severe hypothyroidism^[7,11].

Due to multi-organ involvement and the potential for arrhythmia, admission to critical care is recommended, where at a minimum the patient can be closely monitored^[3,7,12].

Prognosis

Evidence shows that advanced age and reduced consciousness level are associated with worse outcomes^[8,13]. Other poor prognostic factors include persistent hypothermia (unresponsive to 72 hours of therapy) as well as significant underlying precipitants such as myocardial infarction^[4,14].

CONCLUSIONS

Myxoedema coma can be difficult to recognise, but early treatment is imperative. Management is three-fold and consists of rapid thyroid hormone replacement, supportive measures and the treatment of coexisting problems, most importantly infection^[7]. This is most effectively done in a critical care environment, at least in the acute phase.

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