

An uncommon case of rhabdomyolysis in severe hypothyroidism

Sujatha Kamalaksha, Nicole McGrath, Chuen Siang Low, Sanjib Ghosh

Rhabdomyolysis is a triad of myalgia, myopathy and pigmenturia. Clinical presentation is usually triggered by dehydration, drugs and or intense exercise. Hypothyroidism-induced rhabdomyolysis is rarely described in literature.¹⁻³ It is often reported as a risk factor.^{4,6}

Case report

A 77-year-old Caucasian woman was admitted to our hospital with gradual onset of progressive lower limb proximal weakness over that past year, which grew worse in last 2 weeks, associated with brain fog, constipation, cold sensitivity and unintentional weight loss of 4kg. She was an avid ocean swimmer in the past, now only able to paddle around the boat. Her mobility gradually deteriorated from having been independent to requiring support to mobilise. She was a teetotaler with no recent change of medications. Her past medical history included ischemic heart disease, hypothyroidism and cholecystitis, which was managed conservatively with antibiotics 1 year prior to this admission. Medications included bisoprolol, aspirin, losartan, levothyroxine as synthroid and atorvastatin. She had been on statin for 8 years, and the dose was doubled 3 years ago for acute coronary syndrome. She was on 50mcg synthroid that could not be optimised due to side effects of feeling generally unwell. Although consistent euthyroid

status was not achieved, in the past a synthroid dose of 150mcg correlated with brief normalisation of thyroid functions (Table 1).

On examination, she was normotensive, afebrile and jaundiced. There was 3/5 proximal muscle group weakness in the lower limbs, with normal power in other muscle groups. Examination of deep tendon reflexes revealed a positive Woltman sign. There were no fasciculations or sensory impairment. Review of other systems was normal.

Initial investigations (Table 2) revealed significantly elevated creatinine kinase (CK) (39,600U/L) and abnormal liver functions with alkaline phosphatase (ALP) and gamma glutaryl transferase (GGT) of 1,050U/L and 3,240U/L respectively. She had acute kidney injury with serum creatinine of 131umol/L. Thyroid stimulating hormone (TSH) was 59mIU/L with free T4 9.5pmol/L. Serum aldolase of 1,25U/L and positive urine haemoglobin pigments supported rhabdomyolysis. Myositis antibodies were negative. Magnetic resonance cholangiopancreatography (MRCP) and computed tomography (CT) revealed metastatic cholangiocarcinoma.

Initial treatment consisted of intravenous fluids and discontinuation of atorvastatin. Due to synthroid intolerability, she was commenced on eltroxin 50mcg daily. The dose was escalated quickly to 150mcg within a week as she was an inpatient. She was not considered for surgery or chemotherapy.

Table 1: Historical thyroid function tests.

	2017 (synthroid 150 mcg)	2018 (synthroid 50 mcg)	2019 (synthroid 50 mcg)	2020 (synthroid 50 mcg)	2021 (synthroid 50 mcg)
TSH (0.27–4.2mU/L)	2.5	28	35	34	59
Free T4 (12–22pmol/L)	17	13	12	13	9.5

TSH = thyroid stimulating hormone

Table 2: Results at the time of admission.

Urea (3.2–7.7mmol/L)	8.1	Bilirubin (<25 umol/L)	34
Creatinine (45–90umol/L)	131	GGT (<50U/L)	3,240
CK (30–180U/L)	39,600	ALP (40–130U/L)	1,050
Serum aldolase (1–10U/L)	>125	ALT (<45u/L)	566
Urine myoglobin	Positive	CRP (0–5mg/L)	43
Myositis antibodies	Negative		

Table 3: Effect of eltroxin treatment.

	Day 1	Day 5	Day 8	Day 22	Day 26
Free T4 (12–22pmol/L)	9.5		13	28	
TSH (0.27–4.2mU/L)	59		51	2	
CK (30–180 U/L)		21,400	7,700		138

TSH = thyroid stimulating hormone
CK = creatinine kinase

There was clinical and biochemical improvement during her hospital stay and the patient was able to mobilise independently on day 11. There were no side effects from eltroxin. Serum TSH normalised on day 22 and serum CK normalised on day 26 of the treatment (Table 3).

Discussion

Hypothyroidism affects 2–5% of the general population; however, 30–50% patients are inadequately treated.⁷ Rhabdomyolysis rarely occurs in patients with poorly controlled hypothyroidism and often in combination with provoking events like exercise, illness and or drugs.⁸ Statin-induced rhabdomyolysis with hypothyroid state has been reported to occur within weeks of commencing the drug.^{6,9} Our patient had been on statin for 8 years without an adverse event despite having hypothy-

roidism. Hence, it was thought unlikely related to the current presentation. Intolerance to synthroid in our patient could be due to gluten content, although she was unaware of gluten sensitivity in her regular diet.¹⁰ Also, different levothyroxine preparations may exhibit differences in the bioequivalence.⁷ As the concern was of intolerability rather than the bioavailability, the decision was made to equate the dose of eltroxin early. Clinical and biochemical recovery after commencing eltroxin, despite terminal malignancy, concluded hypothyroidism-induced rhabdomyolysis. Patient died within 3 months due to malignancy and hence a sustained effect of eltroxin at that dose cannot be commented on. Thyroid function tests should be checked for rhabdomyolysis presentations and changing the levothyroxine preparation should be considered, if necessary.

COMPETING INTERESTS

Nil.

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