CASE REPORT

Hashimoto's thyroiditis presenting as Hoffman's syndrome, rhabdomyolysis and acute kidney injury

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SUMMARY

An otherwise healthy young man presented with gradual progressive fatigue for the past 12 months disturbing his daily activities. Clinical examination revealed marked generalised muscular hypertrophy including the temporalis muscles bilaterally. Investigation revealed that the patient was grossly hypothyroid due to Hashimoto's thyroiditis with rhabdomyolysis and acute kidney injury. The finding of muscle weakness and pseudohypertrophy in association with hypothyroidism is called Hoffman's syndrome. The patient was hydrated and thyroxine replacement initiated. On follow-up, the patient showed clinical as well as biochemical improvement.

BACKGROUND

Although hypothyroid myopathy is frequently seen, Hoffman's syndrome is rarely described. Electrophysiological studies reveal myopathy, neuropathy or a mixed pattern. Laboratory investigations generally show increased levels of muscle enzymes in association with low serum thyroid hormones and raised thyroid-stimulating hormone (TSH). Hormone replacement is the mainstay of treatment and the prognosis is generally good. We present a case of a young patient with Hashimoto's thyroiditis showing features of Hoffman's syndrome, rhabdomyolysis and acute kidney injury.

CASE PRESENTATION

A 33-year-old man was referred by his general practitioner with gradual onset progressive generalised weakness, muscle stiffness and fatigue for 1 year, worsening over the previous month. The patient reported an increase in the bulk of his muscles especially in the upper body although he denied any excessive exercise, trauma or recent recreational or prescription drug use. The patient worked outdoors in land management and had a history of tick bites. There was no other medical history of note. Alcohol consumption was only moderate. Subsequently, it was noted that there was a strong family history of hypothyroidism. Systems review was negative. On examination, the patient was alert, weighed 93.6 kg and had highly developed upper body musculature including prominence of temporalis muscles bilaterally (figure 1). Physical examination including neurological examination was otherwise normal.

INVESTIGATIONS

Initial blood investigations showed normal full blood count, C reactive protein and electrolytes. Serum creatinine and urea were elevated,

Figure 1 Patient at presentation with temporalis muscle hypertrophy.

164 $\mu\text{mol/L}$ and 8.8 mmol/L, respectively. Creatine kinase was 2333 iu/L (normal range 30–200). Liver biochemistry was as follows: γ -glutamyl transpeptidase 146 iu/L, aspartate aminotransferase 82 iu/L, alanine transaminase 62 iu/L, alkaline phosphatase 63 iu/L and bilirubin 40 $\mu\text{mol/L}$. Fasting cholesterol 8.5 mmol/L. Chest X-ray was normal. ECG showed normal sinus rhythm, rate 78 bpm. Urinalysis was normal. Renal ultrasound scan was normal. Viral screening including cytomegalovirus and HIV and monospot was negative. Autoantibody screen, leptospirosis and Lyme serology were negative. Urine output was 800 mLs in the first 24 h with input 2700 mLs. Urine output increased on the second and third day of admission to 1450 and 3000 mLs, respectively.

TREATMENT, OUTCOME AND FOLLOW-UP

An initial diagnosis of rhabdomyolysis with acute renal injury of unknown cause was carried out. The patient was started on intravenous fluids. Urea and electrolytes were checked daily. Urine myoglobin was not detected. On the third day of hospital admission thyroid function test results became available and showed TSH 209 μL (normal range 0.15–3.2), T4 was <5 pmol/L (normal range 9.9–20.1). Thyroid peroxidase antibody was greater than 1000 iu/mL (normal range <35 iu/mL). A final diagnosis of Hashimoto's thyroiditis presenting as Hoffman's syndrome with rhabdomyolysis and acute renal injury was made. The patient was started on thyroxine 50 μg daily and discharged. At outpatient review 6 weeks later, his symptoms had improved. TSH had fallen to 10.9 mu/L, T4 was 17.8 pmol/L. Eltroxin was increased to 100 μg once daily. He was reviewed in another 6 weeks and he

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Figure 2 Follow-up at 24 weeks showing resolution of temporalis muscle hypertrophy.

reported feeling much better with a TSH level of 9.26 $\mu\text{u/L}$ and T4 of 14.9 pmol/L . By 12 weeks his renal and liver function had returned to normal, cholesterol level fell to 5.6 mmol/L and creatine kinase was 125 iu/L . The previously noted temporalis muscle hypertrophy had fully resolved by 24 weeks (figure 2).

DISCUSSION

Hoffman's syndrome is a specific rare form of hypothyroid myopathy which causes proximal weakness and pseudohypertrophy.

In children with cretinism, a similar presentation called Kocher-Debré-Sémélaigne syndrome exists although the two may overlap.¹ We are reporting this case of Hoffman's syndrome for its rarity. Deficiency of thyroid hormone interferes with metabolism leading to a decreased metabolic rate. These metabolic changes affect a number of organs including skeletal muscle.^{1 2} Patients may have muscle cramping, proximal symmetrical muscle weakness, muscle stiffness and exercise intolerance.^{2 3} Muscular hypertrophy is reported in less than 10% of the patients.^{4 5} Pseudohypertrophy of muscles result from the accumulation of glycosaminoglycans.⁵ Thyroxine replacement therapy is the mainstay of treatment and prognosis is good.

Learning points

- Assessment of the thyroid function is to be considered in any patient presenting with rhabdomyolysis and acute kidney injury.
- Early initiation of treatment will lead to clinical as well as biochemical improvement in the patient's condition.
- After initiation of thyroxine therapy, the patient should be re-evaluated and serum thyroid-stimulating hormone (TSH) should be measured in 6 weeks, and the dose adjusted accordingly. Symptoms may begin to resolve after 2–3 weeks, but normalisation of TSH concentrations may not be achieved for several months.

Contributors GSA is the main author of the case report, HMZ provided the images of the case, MM acted as senior author and editor of the final draft of the submission.

Competing interests None.

Patient consent Obtained.

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