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Endocrinology

Advanced Myopathy at the Stage of Rhabdomyolysis Revealing Hashimoto's Thyroiditis

Sara.IJDDA[1*], Sana.Rafi[1], Ghizlane.Elmghari[1], Nawal El Ansari[1], Layla.Bendaoud[2], Said Amal[2]

[1]Department of Endocrinology, University Hospital Mohamed VI, Marrakesh, Morocco
[2]Department of Dermatology University Hospital Mohamed VI, Marrakesh, Morocco

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***Corresponding author:** Sara Ijdda
Department of Endocrinology, University Hospital Mohamed VI, Marrakesh, Morocco

Abstract **Case Report**

Hypothyroidism is a common endocrine disorder with several complications, including muscle damage, particularly rhabdomyolysis, which is a rare manifestation. Clinically, it is manifested by muscle pain, weakness, and stiffness. Biologically, there is an increase in muscle enzymes. We report a case of 42-year-old man, hospitalized in dermatology departement for subcutaneous nodules, and who presented a deep muscular weakness revealing a hypothyroidism complicated by a rhabdomyolysis.

Key words: Hypothroidism, rhabdomyolysis, Hashimoto's thyroiditis.

INTRODUCTION

Acquired hypothyroidism in adults is one of the most common endocrinopathies. Having multiple causes, it is at the origin of a set of clinical and biological abnormalities which are sometimes not very specific. Muscle damage is often frequent and precocious, simulating pathology of the musculoskeletal system. A moderate increase in creatinine phosphokinase (CPK) during hypothyroidism is found in 80% of cases [1-8]. However, cases of major elevation CPK with or without renal failure during hypothyroid myopathy remain exceptional [1, 4, 6]. We report the case of deep peripheral hypothyroidism initiated by severe rhabdomyolysis.

CASE REPORT

M.B. Salah, 42 years old, known type 1 diabetic under insulin, hospitalized in the dermatology department for etiological assessment of subcutaneous nodules on fingers and upper lip; who presented for several month myalgia, muscle cramps in the lower limbs. Clinical examination noted an asthenic patient, with bradycardia at 60 beats per minute, a normal blood pressure at 120/70 mmHg. Examination of the cervical compartment did not reveal any goitre. The biological assessment found total CPK at 2312 IU / l (normal values: 39 to 308) so 9 times the normal, transaminases: AST at 135 IU / l (normal values: 10 to 50), ALT at 125 IU / l (normal values: 10 to 41), and LDH (lactic dehydrgenases) at 694 (normal values: 0 to 250) so 2.5 times the normal with normal serum potassium and normal renal function. The etiological investigation has not found any classic cause of rhabdomyolysis. Among the metabolic causes, only hypothyroidism could be retained. The thyroid hormone checkup then showed a TSHus of 95 IU / l (normal values: 0.27 to 4.20), an LT4 of 0.3 pmol / l (normal values: 12 to 22). The immunological workup found anti-peroxidase antibodies at 740 IU / ml (normal values: 0 to 31) and antithyroglobulin at 193.3 IU / ml (normal values: 0 to 115). Morning cortisol level was normal. The thyroid ultrasound revealed an atrophic thyroid gland. The electrocardiogram and cardiac ultrasound were without particularities. Replacement opotherapy at a dose of 125 µg / day was started in a slowly progressive way, and allowed a normalization of the muscular, hepatic and thyroid tests as well as the disappearance of the muscular signs (Figure 1, 2).

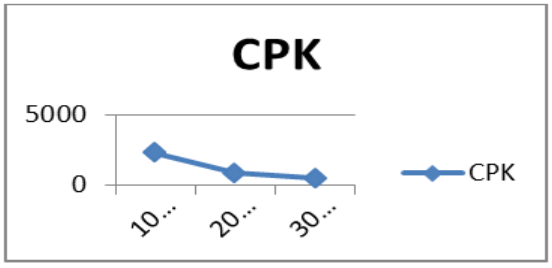

Fig-1: Curve of CPK

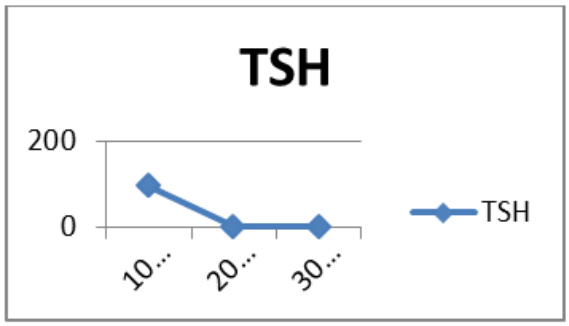

Fig-2: Curve of TSH

For the problem of subcutaneous nodules, the diagnosis of annular granuloma was retained after biopsy and the patient was put on corticoid.

DISCUSSION

Hypothyroid myopathy was first reported by "Ord" in 1884 [7], then thoroughly described by many publications in the years that followed. It usually results in myalgia, painful cramps, and sensations of muscle stiffness predominantly at the roots of the limbs and the shoulder and pelvic girdles, associated with abnormal fatigue and in a third of cases with a moderate rhizomelic muscle deficit [1, 8]. Often precocious, these symptoms can mislead the diagnosis by directing it to other affections, in particular rheumatological [8]. Severe and prolonged forms of hypothyroidism can lead to severe muscle syndromes, as is the case with our patient.

Biological exploration reflects the intensity of the muscle damage. Moderate elevation of CPK, the most sensitive marker, of LDH and transaminases during established hypothyroidism is found in 80% of cases. The origin of this increase is not well known; it could be an increase in muscle catabolism associated with a decrease in enzyme clearance [5].

However, cases of major elevation of CPK with or without renal failure during hypothyroid myopathy are exceptional, as is the case with our patient.

On the other hand, before adopting the diagnosis of hypothyroid myopathy in front of a situtation of rhabdomyolysis, other classic etiologies must be eliminated, in particular:

- Traumatic causes (crush syndrome, direct shock, burns...);
- Intense muscular exercise.
- Muscle compressions (coma, prolonged immobilization...)
- Muscular ischemia (acute arterial occlusion, state of shock, hypovolaemia...);
- Infectious syndromes;
- Toxic causes (heroin, cocaine, alcohol...)
- Iatrogenic causes (statins, colchicine...)
- Other metabolic disorders (hypokalaemia, hypophosphatemia...);
- Malignant hyperthermia (per anesthetic, neuroleptic malignant syndrome, heat stroke..);
- Hereditary enzymopathies;
- Polymyositis and dermatomyositis.

In fact, hypothyroid myopathy is the consequence of a defect in mitochondrial metabolism as well as various metabolic processes in muscle cells with abnormal glycogenolysis, due to the regulatory role of thyroid hormones on the transcription of numerous muscle genes [1, 4, 9].

Biochemical studies also demonstrate changes in myosin and the activity of the Na + / K + –ATPas pump, the presence of glycosaminoglycans deposits and lipid accumulation, as well as a decrease in carnitine [2, 3, 9, 10, 11].

Thyroid myopathy is easily recognized when hypothyroidism is known, but becomes difficult when it is inaugural. Thus, the thyroid myopathy must be evoked in front of any muscular damage.

Hormone replacement therapy allows resolution of thyroid myopathy. This should be slowly progressive, allowing the correction of renal function, the gradual disappearance of the signs of hypothyroidism and the normalization of CPKs and transaminases.

Indeed, Astudillo *et al*. [1] described a case of severe rhabdomyolysis revealing thyroid myopathy, witch hormone replacement therapy normalized CPKs and aldolases with disappearance of muscle signs within four months like the case of our patient.

CONCLUSION

Severe myopathy with rhabdomyolysis should encourage the search for hypothyroidism although this is a rare cause, which justifies monitoring the thyroid exploration during any muscle lysis. Hypothyroidism therefore appears to be an authentic cause of a major rise in muscle enzymes. The clinical and biological outcome is favorable after several months of gradually instituted replacement therapy.

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