Journal Homepage: -www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI:10.21474/IJAR01/14516
DOI URL: http://dx.doi.org/10.21474/IJAR01/14516

RESEARCH ARTICLE

ACUTE PSYCHOSIS AND RHABDOMYOLYSIS IN THE FACE OF HYPOTHYROIDISM

Bilihi Bouyela N.C, Camara M., Rafi S., El Mghari G. and El Ansari N.
Department of Endocrinology Diabetology and Metabolic Diseases, FIP Laboratory, GFCM, Cadi Ayyad University, CHU Mohamed VI Marrakech.

Manuscript Info

Manuscript History
Received: 05 February 2022
Final Accepted: 11 March 2022
Published: April 2022

Key words:-
Acute Psychosis, Rhabdomyolysis, Hypothyroidism, Creatine Phosphokinase

Abstract

Myxedematous psychosis was first described in 1979 by Pr Asher, it is a rare diagnosis associating an acute psychosis, a rhabdomyolysis whose etiology is hypothyroidism. We report the case of a 32-year-old patient followed for 6 years for schizophrenia, who had not had any treatment for 3 years, and was admitted with an acute psychotic attack consisting of delusions of persecution. The examination revealed an oedematose syndrome, deep hypothyroidism, and high creatine phosphokinase. The treatment was the correction of the hypothyroidism associated with a short psychogenic treatment with a favorable evolution after 1 month of treatment with regression of the psychiatric picture.

Introduction:-

Hypothyroidism is a common condition with an estimated prevalence of 3.1% of the adult population (1). Rhabdomyolysis in hypothyroidism was first described in 1979 and is a rare diagnosis (2). Neuropsychiatric and muscular symptoms can develop in the setting of hypothyroidism but are rarely the first signs of the latter (3). We report a case of acute psychosis and rhabdomyolysis revealing profound hypothyroidism.

Observation:-

32 year old patient was followed in psychiatry for 6 years for schizophrenia under neuroleptics in discontinuation of treatment for 3 years. He was admitted for an acute psychotic attack made of incoherent and obscene remarks, behavioral weirdness (showers several times in the day and walks without clothes), isolation with a social withdrawal associated with hetero-aggressiveness towards his family, the whole evolving in a context of delusions of persecution towards his sister, and a psychosocial agitation. In addition, the patient reports chronic constipation. The clinical examination showed a slowed down patient, bradycardic at 45 beats/min, normotensive at 120/60mmHg, a generalized myxedema: facial puffiness, palpebral oedema with a moderate bilateral ptosis, filling of the supra clavicular hollows, pre-tibial myxedema, dry skin, hair loss, a regular goiter without palpable nodule.

The work-up: total CPK at 5740 U/l i.e. 28 times normal, kalaemia at 4.4 mmol/l, creatinine at 11.3 mg/l, profound peripheral hypothyroidism with TSH:296 µIU/l and T4L:<5.15 pmol/l and T3L:<1.50 pmol/l, thyroid ultrasound reveals a hypoechoic goitre with hyperechoic fibrous cross-sections with a significant vascularisation, and anti-TPO antibodies are positive at 300 U/ml.

Corresponding Author:- Bilihi Bouyela N.C
Address:- Department of Endocrinology Diabetology and Metabolic Diseases, FIP Laboratory, GFCM, Cadi Ayyad University, CHU Mohamed VI Marrakech.

The diagnosis of Hashimoto's thyroiditis associated with rhabdomyolysis revealed by psychiatric disorders was retained. Treatment with l-thyroxine at a dose of 1.7 µg/kg/D associated with an antipsychotic treatment was initiated with a favourable evolution after 1 month of treatment by the regression of the psychiatric picture and myxedema.

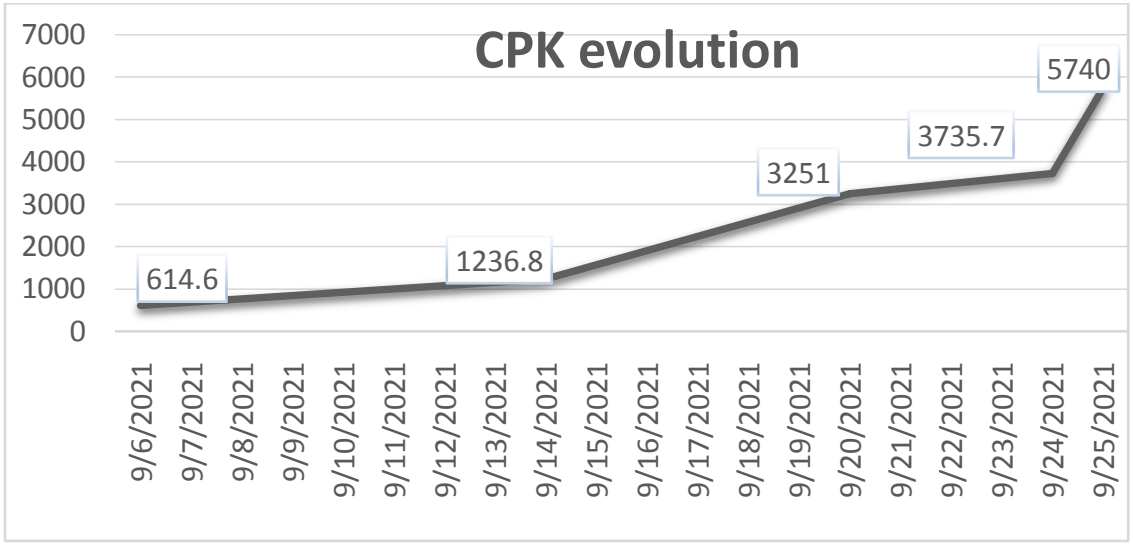

Figure 1:- CPk evolution.

Discussion:-

Hypothyroidism myxedematous psychosis was first described by Prof Asher in 1949 (4). Its prevalence is rare with only a few cases reported in the literature. The neuropsychiatric mechanisms induced by hypothyroidism are not elucidated in humans. Attempts to answer these mechanisms have been provided by data based on animal experiments. Two mechanisms have been described: the presence of thyroid hormone receptors in the limbic structure that controls emotion and behavior, and the anterior imbalance of tyrosine hydroxylase in the locus coeruleus resulting from the dysthyroid state (5). In a cohort of patients studied, there was evidence of reduced glucose perfusion and metabolism (6-7).

Rhabdomyolysis in hypothyroidism is caused by muscle degeneration with atrophy of type II fibres, and compensatory hypertrophy of type I fibres secondary to a defect in mitochondrial metabolism (8). Glycosaminoglycan deposition changes in contractility of actin-myosin units, and low myosin ATPase activity in skeletal muscle are also involved in the pathophysiology (9).

The causes of rhabdomyolysis are numerous, including drugs, toxins, infections, metabolic factors, abnormalities, physical exercise and dehydration as well as neuroleptic malignant syndrome which was eliminated in our patient by interrogation, cessation of all neuroleptic treatment for 3 years (10). Certain favourable factors have been incriminated, in particular lipolipid treatment and physical exercise, but it can occur without favourable factors and be diagnosed concomitantly with hypothyroidism as in our patient (9).

Symptoms of rhabdomyolysis, although not always present, include muscle pain, weakness and myoglobinuria (10). However, the absence of these signs should not exclude hypothyroidism as a cause of rhabdomyolysis (9). Psychiatrically, in our patient, as well as in Mouhand FH Mohamed et al and Professor Asher, delusions of persecution were present (1,4). Signs of hypothyroidism, if present as in our patient, are orientative but their absence does not exclude the diagnosis.

The diagnosis is the demonstration of peripheral hypothyroidism associated with elevated CPK.
The management is made by correcting the hypothyroidism and the evolution is favourable in the majority of cases. Some patients keep deficits probably due to a chronic hypothyroidism having generated irreversible cerebral lesions (5). Our patient in addition to the correction of hypothyroidism benefited from antipsychotic treatment. Many

patients in addition to treatment with pat L thyroxine have benefited from the addition of short term antipsychotics (5).

Conclusion:-

Myxedematous psychosis is a rare condition that combines acute psychosis and rhabdomyolysis with hypothyroidism as the aetiology. This condition may occur with or without contributing factors. The delusion of persecution is in the foreground, the signs of hypothyroidism and rhabdomyolysis, if they are present, are orienting but their absence does not allow to exclude it. The diagnosis is made by demonstrating peripheral hypothyroidism in relation to elevated CPK levels. The management is based on the correction of hypothyroidism sometimes associated with a short term antipsychotic treatment. The evolution is favourable with improvement of psychiatric disorders.

References:-

1. Mouhand FH Mohamed, Ali B Mahgoub, Sundus Sardar, Abdel-Naser Elzouki. Acute psychosis and concurrent rhabdomyolysis unveiling diagnosis of hypothyroidism. BMJ Case Rep 2019;12:e231579.
2. Jobé J, Corman V, Fumal A, Maertens de Noordhout A, Legros JJ. Le cas clinique du mois. Rhabdomyolyse et hypothyroïdie [Rhabdomyolysis and hypothyroidism]. Rev Med Liege. 2007 Jul-Aug;62(7-8):484-6.
3. Aoki Y, Belin RM, Clickner R, Jeffries R, Phillips L, Mahaffey KR. Serum TSH and total T4 in the United States population and their association with participant characteristics: National Health and Nutrition Examination Survey (NHANES 1999-2002). Thyroid. 2007 Dec;17(12):1211-23.
4. Asher, r. "Folie myxœdémateuse." Revue médicale britannique vol. 2,4627 (1949): 555-62.
5. SundusSardar, Mhd-Baraa Habib,AseelSukik, BasharTanous, Sara Mohamed, RaadTahtouh, Abdelrahman Hamad, and Mouhand F. H. Mohamed. Myxedema Psychosis: Neuropsychiatric Manifestations and Rhabdomyolysis Unmasking Hypothyroidism. Hindawi Case Reports in Psychiatry Volume 2020.
6. M. Bauer, D. H. S. Silverman, F. Schlagenhauf et al., "Brain glucose metabolism in hypothyroidism: a positron emission tomography study before and after thyroid hormone replacement therapy," The Journal of Clinical Endocrinology and Metabolism, vol. 94, no. 8, pp. 2922–2929, 2009
7. E. L. Constant, A. G. de Volder, A. Ivanoiu et al., "Cerebral blood flow and glucose metabolism in hypothyroidism: a positron emission tomography study," The Journal of Clinical Endocrinology and Metabolism, vol. 86, no. 8, pp. 3864–3870, 2001
8. Salehi N, Agoston E, Munir I, Thompson GJ. Rhabdomyolysis in a Patient with Severe Hypothyroidism. Am J Case Rep. 2017.
9. Varalakshmi DB, Ram R, Siva Kumar V. Rhabdomyolysis due to hypothyroidism without any precipitating factor. Trop Doct. 2020 Jul;50(3):273-274.
10. Alshamam MS, Gurung DO, Nso N, Saliaj M, Seitaj A. Rhabdomyolysis Secondary to Hypothyroidism: Report of a Case. Cureus . 2021;13(1):e12746.