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PROPYLTHIOURACIL AND PERIPHERAL NEUROPATHY

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SUMMARY — Peripheral neuropathy is a rare manifestation in hyperthyroidism. We describe the neurological manifestations of a 38 year old female with Graves' disease who developed peripheral neuropathy in the course of her treatment with propylthiouracil. After the drug was tapered off, the neurological signs disappeared. Therefore, we call attention for a possible toxic effect on peripheral nervous system caused by this drug.

KEY WORDS: hyperthyroidism, peripheral neuropathy, propylthiouracil.

Neuropatia periférica induzida por propiltiouracil.

RESUMO — Os autores descrevem o caso de uma paciente de 38 anos de idade, com doença de Graves, que desenvolveu sinais e sintomas compatíveis a polineuropatia sensitivo-motora durante o tratamento com propiltiouracil. Após a retirada gradual da referida droga, o quadro neurológico desapareceu por completo. Sendo extremamente incomum o surgimento de neuropatia periférica secundária ao hipertiroidismo e em vista do uso corriqueiro do propiltiouracil no tratamento dessa enfermidade endócrina, os autores chamam a atenção para a possível participação do fármaco como agente etiológico de neuropatia periférica em enfêrmos usuários do propiltiouracil.

PALAVRAS-CHAVE: hipertiroidismo, neuropatia periférica, propiltiouracil.

Hyperthyoroidism is a common disease in medical practice and is frequently associated to various neurological and neuromuscular disorders, the commonest of them is proximal myopathy [1]. Thyrotoxic neuropathy is extremely rare and this association is questioned by several authors. Some publications describe this entity clinically and electrophysiologically [1,2,5]. It was first described as Basedow's paraplegia by Charcot in 1889, and Joffroy in 1894 [1]. Neurological disturbances associated to propylthiouracil (PTU) are rare. Vertigo, paresthesia, dysesthesia are described but there is no known direct neurotoxic effect of the drug capable of determining neuropathy [1]. Meyer-Gessner and colleagues [4], in a series of 1256 patients during antithyroid treatment, reported peripheral neuropathy in 0.7%, of cases, not mentioning which drug was implicated in this disorder.

In this brief report we show one case of peripheral neuropathy induced by PTU

CASE REPORT

INM, a 38 year old woman presented with hyperthyroidism at age of 37 with emotional lability, heat intolerance, bilateral hand tremulousness, palpitations, fatigue, hyporexia, loss of 20 Kg in four months, hair loss, ungueal fragility, maculo-papular pruriginous rash in trunk and lower limbs (with no drug use) and difficulty to maintain the arms over her head (legs' muscular strength was normal). Two sisters and one brother had hyperthyroidism. She denied smoking, alcohol use nor any other drug. At admission the examination showed:

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emotional lability; smooth and scanty hair; moisty and warm skin; maculo-papular rash distributed in trunk and thighs; bilateral palmar erythema; bright eyes; diffuse enlargement of thyroid gland (three times the normal size) of elastic consistency, and smooth surface; sinus tachycardia at rest; Plummer's nails on both haluxes; muscular strength reduction on scapular girdle; fine tremor of hands; hyperactivity of muscle stretch reflexes in all limbs. Ultrasonography and scan of thyroid gland suggested the diagnosis of difuse toxic goiter. Laboratory results are shown in Table 1.

Table 1. Laboratory Findings.

TSH	0.5 μIU/mL
T4	15.0 μg/dL
T3	240.0 μg/dL
Thyroid microsomal antibody	1/25600
Thyroglobulin antibodies	Negative
ALT	32.0 U/L
AST	26.0 U/L
CK	10.0 U/L
LDH	74.0 U/L
Aldolase	6.0 U/L
Alkaline phosphatase	211.0 U/L

We initiated PTU 600mg/day. Beta-adrenergic-blockers were not utilized since there was previous history of bronchial asthma. Two weeks after the dose was augmented to 800mg/day because of no clinical response. Two days later arthralgias, cervical nodes and peripheral neuropathy were detected. Neurological examination revealed: ataxic gait; loss of strength on scapular girdle, tibial anterior and gastrocnemius muscles in both sides; bilateral hand tremor; loss of ankle jerk reflexes; hypalgesia, hypesthesia and pallanesthesia of stocking type. We were in doubt if peripheral neuropathy was part of the syndrome of hyperthyroidism or secondary to PTU's use. Anyway it was a rare manifestation. Electroneuromyography confirmed the diagnosis of sensorimotor peripheral neuropathy.

The drug was then tapered off gradually with concomitant use of dexamethasone 8mg/day. Five days later, the patient was able to walk, and within 8 days there was no clinical signs of peripheral neuropathy, although she continued with clinical signs of hyperthyroidism.

COMMENTS

Through this concise report we can deduce that PTU was responsible for the patient's neurological disorder since dose reduction, till the suspension of the drug, resulted in disappearence of peripheral neuropathy. Leger and colleagues [3] reported four cases of peripheral neuropathy and one of bilateral retrobulbar optical neuritis, related to the use of carbimazole. Electrophysiological findings and neuromuscular biopsy suggested axonal and demyelinating lesion associated to microvasculitis.

Is the mechanism of PTU's neurotoxicity similar to carbimazole?

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