Review article

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Hyperparathyroidism and Connectivities: Study of 04 Observations

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Abstract

Primary hyperparathyroidism (HPTp) as an appropriate secretion of parathyroid hormone (PTH) by one or more parathyroid glands [1]. We thus report 4 observations of patients exhibiting connectivitis with HPTp, describe the therapeutic and evolutionary aspects.

Keywords: Hyperparathyroidism, connectivitis, hypercalcemia.

Introduction

Primary hyperparathyroidism (HPTp) is defined as an appropriate secretion of parathyroid hormone (PTH) by one or more parathyroid glands [1]. It can be sporadic or familial or occur in the context of multiple endocrine neoplasia (MEN). The association of HPTp and connectivitis is rare, even exceptional. It has never been reported in Africa, to our knowledge. We thus report 4 observations of patients exhibiting connectivitis with HPTp.

Patients and Methods

We carried out a retrospective study in the Rheumatology department of the Aristide Le Dantec University Hospital where we collected all the observations of connectivitis associated with hyperparathyroidism. The diagnosis of connectivitis was based on epidemiological, clinical and paraclinical arguments in accordance with the usual international classification criteria. For rheumatoid arthritis (RA), we used the American College of Rheumatology (ACR) / European League Against Rheumatism (EULAR 2010) criteria; for Sjogren's syndrome (SS), the European-American consensus criteria of 2002. The diagnosis of polymyositis (PM) was based on the criteria of Bohan and Peter (1975). The diagnosis of pHTp was made on the basis of permanent hypercalcaemia associated with an increase in PTH as measured by immunoradiometry. The diagnosis of localization of parathyroid lesions was based on ultrasound and computed tomography.

Results

We have collected four (04) observations. They were three (03) women aged 63, 59 and 46 and a man of 60 years. The diagnosis of connectivitis was concomitant with the diagnosis of pHTp in all cases. HPTp was discovered incidentally in the presence of a serum calcium demanded systematically and high income. Autoimmune diseases were represented by RA (01 case) in a 63-year-old patient, primary gougerot-sjogren syndrome (02 cases) in 59 and 46-year-old patients. Polymyositis was diagnosed in the 60-year-old patient. There was no urolithiasis or osteoporosis, nor kidney involvement. The lesion causing pHTp was parathyroid adenoma in all cases. No patient had an MRI (magnetic resonance imaging) or MIBI scintigraphy.

The diagnosis of RA was made due to the association of a predominantly distal polysynovial arthritis, a biological inflammatory syndrome, a positivity of rheumatoid factors (latex at 82 IU / l, Waaler-Rose positive at 115 IU / l) and anti-CCP at 169 U / ml.

Gougerot-sjogren ssyndrome manifested by dry oculo-oral syndrome, positivity of anti-SSA antibodies and the presence on biopsy of salivary glands accessory to grade IV sialadenitis of Chisholm and Masson.

PM was retained due to the association of myogenic syndrome, increased muscle enzymes, a myogenic trace on EMG and positivity of antinuclear autoantibodies.

HPTp was revealed by incidentally discovered hypercalcemia, with an average level of 126.5 mg / l (range: 115 - 132). PTH was

increased with an average rate of 109 ng / l (range: 67 - 192).

Therapeutically, the patients received treatment with corticosteroids, hydroxychloroquine and methotrexate. Surgery was performed in all patients. The evolution has been favorable in all cases.

Discussion

Hypercalcemia has been reported in connectivitis, particularly in systemic lupus and RA [2,3]. Several hypotheses have been put forward concerning their etiology. Indeed, some authors incriminate the presence of a PTHrp, others, the presence of pro-inflammatory cytokines [4]. The first hypotheses concerning their etiology believed that they were due to an overactivity of the parathyroid glands, this hyperactivity being considered as a clinical sign of RA [3]. However, a systematic dosage of PTH in a first originator study found low values of this hormone, thus leading to the hypothesis of a non-PTH factor responsible for a systemic release of calcium [3]. However, primary hyperparathyroidism, which is a common cause of hypercalcaemia (if not the primary cause) has only rarely been described in connectivitis. These are mainly lupus, antiphospholipid syndrome and RA [3, 5-8]. This association is rare. Indeed, Ralston and al., in 3250 patients followed for RA, found only 24 cases of pHPT (1/135) [3]. This prevalence is however higher than that found in the general population (1/5000) [3]. Thus, there is probably a connection between HPTp and connectivities, in particular RA. Canas has put forward two hypotheses to explain this association [9]. The 1st hypothesis is that the PTH receptors are present on the T and B lymphocytes. Thus, the PTH causes a direct stimulation of these immune cells via its receptor. The second hypothesis is indirect. It assumes that B cells are stimulated through the production of interleukin-6 by stromal cells, osteoblasts and parathyroid glands. This stimulation of the B lymphocytes leads to their maturation and their differentiation into plasma cells, thus resulting in the production of antibodies.

PHPT in our patients was moderate, asymptomatic and incidentally discovered, which is consistent with the data in the literature [3]. All our patients underwent parathyroidectomy without major complications. Surgery remains the treatment of choice for pHTp, whether isolated or associated with another pathology [1, 10].

Conclusion

PHTp is a disease caused by the inappropriate secretion of parathyroid hormone, resulting in hypercalcemia. Its association with connectivitis is rarely reported and may be related to stimulation of B lymphocytes by hypersecretion of PTH.

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