Letters to the Editor

Frequency of anticardiolipin Ac in patients with hyperprolactinemia

Sir,

Association between hyperprolactinemia and autoimmune diseases has been demonstrated.^[1,2] We assessed the frequency of anticardiolipin (ACL IgG and IgM by ELISA) and thyroperoxidase (ATPO by chemiluminescence) antibodies, in 23 patients with hyperprolactinemia, subdivided into two groups: Group 1, untreated hyperprolactinemic patients (n = 14; 42.5 \pm 14.7 years) and Group 2, treated hyperprolactinemic patients (n = 9, 39.4 ± 12.1 years). Group 3, control, consisted of 20 individuals without thyroid and autoimmune diseases (n = 20; 47.1 \pm 13.6 years). Positivity of ACL was observed in none of the control, in two patients with treated hyperprolactinemia (one IgM and one IgG) and in one with treatment-free hyperprolactinemia (IgM) [Table 1]. ACL IgG levels were significantly higher in the group with hyperprolactinemia $(2.1 \pm 2.5 \text{ GPL})$ compared to the control group $(0.7 \pm$ 0.2 GPL), P = 0.018. ATPO was detected in 11/23 (47%) patients with hyperprolactinemia and in 1/20 (5%) of controls and the mean was significantly higher in the hyperprolactinemic group compared to control (P = 0.004).

The anticardiolipin antibody is organ-nonspecific, occurring in many autoimmune diseases such as rheumatoid arthritis and SLE, and in general population low levels of ACL (IgG < 4.3 μ /ml, IgM < 3.55 μ /ml and polivalent ACA < 4.55 μ /m) are described as well as positivity for IgM and IgG ACL around 4.6%.^[3] In agreement we found no positivity of ACL in control group. Moreover, we detected positivity and mean level of ACL significantly higher in patients with hyperprolactinemia, treated or not, corroborating a previous study that reported positive results of ACL in 27% of women with hyperprolactinemia, with no symptoms of autoimmune diseases.^[4] The result of the ACL presented here, as well as the knowledge that hyperprolactinemic individuals have significantly higher percentage of total lymphocytes and CD2-positive cells^[2] and that levels of PRL in SLE patients are higher than in the general population^[1,2] reinforce the immunomodulating role of prolactin, interacting with environmental and genetic factors for the development of autoimmune diseases.

Increased levels of ATPO were detected in approximately half of patients with treated or untreated hyperprolactinemia. The presence of ATPO has already been linked to both hyperprolactinemia⁴ and to the low reserve of PRL^[5] and hyperprolactinemic women show higher prevalence of thyroid autoimmune disease than the general population.^[4]

Although some studies suggest that the presence of the ACL in thyroid autoimmune disease may be a nonspecific marker for the activation of the immune system, this association is controversial and the influence of these antibodies in the course of thyroidopathy has not been totally explained yet. Data are conflicting regarding this issue, with reports of increased prevalence and no correlation between ACL and autoimmune thyroid disease.^[6]

The present findings of highest prevalence of ACL and ATPO in patients with hyperprolactinemia reinforce the concern over possible development of autoimmune diseases in prolonged hyperprolactinemia. However, the correlation between these factors remains unclear, suggesting the need for further studies.

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Table 1: Positivity and variation of the levels of anticardiolipin and thyroperoxidase in hyperprolactinemic and	
control groups	

Group (n)	ACL IgM +	Variation	ACL lgM +	Variation	ATPO +	Variation
	n (%)	(MPL)	n (%)	(GPL)	n (%)	(IU/mL)
Hyperprolactinemia (23)	2/23 (8.6)	0.39-27.5	1/23 (4.3)	0.31-10.5	11/23 (47)	16-3775
Control (20)	0/20 (0)	0.7-6.9	0/20 (0)	0.45-3.2	1/20 (5)	10-144
	0/20(0)	0.7-0.9	0/20(0)	0.45-5.2	1/20(3)	10-144

ACL: Anticardiolipin, ATPO: Thyroperoxidase

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