

Summary

Prolactin (PRL) is a polypeptide hormone of 23 kDa molecular weight made up of 199 amino acids, and produced by lactotropes, acidophilic cells of the anterior lobe of the pituitary. PRL is also synthesized in some other parts of the brain and in certain peripheral blood elements. Whether this extra-pituitary PRL, also known as PRL-like hormone, interferes with serum PRL radioimmunoassay (RIA) and whether it also has a feedback effect on PRL secretion in the pituitary, has yet to be elucidated. There is, however, proof of its apocrine and paracrine function of cellular growth factor, a function enhancing mitogenesis and lymphocyte differentiation at the site of inflammation and thereby their own production of yet other mediators and immunomodulators - including interleukines (IL) and growth factors. PRL also directly interferes with the synthesis of some acute-phase proteins in the liver (stimulating, e.g., alpha-2-macroglobulin synthesis). As a result of these discoveries, PRL was classed among immunomodulators, and the hypothesis was advanced of its part in the pathogenesis of autoimmune diseases.

The aim of the thesis was to verify the presence of hyperprolactinemia (hyper-PRL) in SLE patients compare to patients with other auto-immune diseases and to healthy controls and to find its association with high disease activity, specific organ involvement or presence of anti-dsDNA antibodies.

We found a significantly higher rate of elevated PRL levels in SLE patients (40.0%) compared with the healthy controls (14.8%, $p < 0.017$). No proof was found of association with disease activity or with specific organ involvement or the presence of anti-dsDNA. Similarly, elevated PRL levels were found in RA patients (39.3%). The PRL elevation tended to decline from the 1st to the 3rd sample in the group of patients with SLE and patients with other auto-immune diseases but not in healthy controls.

In conclusion, as follows from our measurements of prolactin serum values in SLE patients they are variable by definition. Summarizing in our study, we abided by certain physiological, pathophysiological and endocrinological rules in studying the PRL serum levels, hormones and a potential agent interconnecting the neurohumoral and immune systems. The blood samples were taken repeatedly after a period of time

following the introduction of the cannula, while the subjects were in a perfectly relaxed condition, and in the morning hours. We observed different physiological norms for men, for women in the child-bearing age, and for menopausal women. As positive results were regarded solely increased values in all three samples. However, even these aspects taken into account in our study are far from all the factors that call for closer examination if the PRL-SLE interconnection is to be fully elucidated. Our study has added to the number of potential factors likely to help expose the PRL relative to the immune system and, consequently, to autoimmune processes and SLE as such but further investigations are needed.