SUMMARY AND CONCLUSIONS

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Prolactin is an anterior pituitary hormone has been known as an immunostimulant neuropeptide. It affects almost every component of the immune system and it is also affected by products of immune system and inflammatory mediators, thus altered PRL level could have a role in the development or the flare up of some autoimmune disease ***(Berczi, 1992).***

Systemic lupus erythematosus (SLE) is a connective tissue autoimmune disease characterized by production of multiple autoantibodies to component of cell nucleus in association with multisystem involvement ***(Daniel et al., 1997).*** The initial complaints in early lupus very widely but most frequently include joint pain or swelling (50%) followed by skin rashes (20%) and malaise or fatigue (10-20%) ***(Snothemer and Gilliam, 1992).***

The disease predominantly affect women whose age ranging from 15-45 but the disease also diagnosed in females after menopause ***(Hochberg, 1990).*** Several factors play a role in the pathogenesis of SLE as environmental, genetic subsceptibility and immune cells disturbances. PRL hormone is one of these factors as it plays a role in pathogenesis and consequently clinical expression of the disease ***(Jara et al., 1992).***

This study was carried out to assess the frequency of hyperprolactinemia in SLE. We studied twenty SLE patients and ten healthy subjects as a control group. The studied cases fulfilled at least four of the American Collage of Rheumatology revised criteria for the diagnosis of SLE ***(Tan et al., 1982).*** Patients were assessed by thorough

history and clinical examination guided by SLEDAI for assessment of disease activity. In addition to routine laboratory investigations we assessed serum prolactin level (ng/ml) by immunlite. The prevalence of hyperprolactinemia among SLE patients was 20% as 4 out of 20 SLE patients were hyperprolactinemic. Hyperprolactinemia was reported in SLE patients with sever disease activity status according to SLEDAI with a score ranging from 14-33. These patients showed significant increase in the renal, CNS, respiratory manifestations and vasculities.

Hyperprolactinemic patients showed significantly lower leucocytic count, red blood cells and platelet count as compared to the normoprolactinemic group, insignificant correlation between serum PRL level and these parameters was detected except for platelet count.

In conclusion these findings demonstrate the presence of hyperprolactinemia in some SLE patients. Also, it demonstrate the association between hyperprolactinemia and major disease manifestations (CNS, renal) and disease activity (high activity score, high ESR, leucopenia). Thus suggest the participation of this immunoregulatory hormone in SLE pathogenesis and support a relationship between the immune and neuroendocrine systems in this disease.

Yet, further studies is recommended to detect the exact role of PRL hormone in pathogenesis of autoimmune disease especially SLE.