Co-existence of Systemic Lupus Erythematosus and Autoimmune Hypothyroidism

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Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease characterized by the production of autoantibodies and chronic inflammation of numerous organs and tissues, including the thyroid gland. It is a common autoimmune disease throughout the world with a prevalence varies from 40 to 100 per 1,00,000. The course of SLE is variable, with periods of exacerbation (called flares) alternating with periods of remission. SLE occurs nine times more often in the female gender than in male and it is more frequent in people of non-European descent.

The difference of clinical features of SLE in different patients is due to the complexity of the risk factors (genetic, hormonal and environmental) and the variety of circulating auto antibodies present. Further, SLE is associated with considerable morbidity and a fivefold increase in mortality compared to age and gender matched control, mainly because of an increased risk of premature cardiovascular disease.

It is suggested that patients with SLE may develop autoimmune thyroid disorders or vice-versa and its association has been observed in numerous studies. Symptoms of thyroid disease and lupus can be confused given that they both have nonspecific features, including fatigue, weight change, dry hair and skin manifestations. In 1961, the first associations between thyroid abnormalities and lupus were described. The first prospective study of thyroid disorders in patients with SLE was performed in 1987 and it was concluded that abnormal thyroid function test results are frequently found in patients with SLE. Since then, studies have repeatedly observed that thyroid dysfunction is more frequent in patients with lupus compared with the general population.

It is unclear how the pro-inflammatory immune state caused by SLE impacts thyroid function. A strong commonality seen between thyroid disease and SLE appears to be the immune predominance of Th helper 1 (Th1) cells. Both SLE and AIID share elevations in interferon gamma and its associated chemokines. Interferon gamma is one of the main cytokines produced by Th1 cells. The mechanism for autoimmune destruction of the thyroid probably involves both cellular immunity and humoral immunity. Lymphocytic infiltration of the thyroid gland by B cells and cytotoxic T cells is a common histologic feature of all forms of autoimmune thyroiditis. Autoimmune thyroiditis is linked to HLA-DR3, which are also linked to SLE.

Despite the theoretical and plausible immunological association seen between SLE and thyroid disease, the clinical correlations vary according to the context of the thyroid disease. The patients with SLE may be euthyroid, subclinical hypothyroid, overt hypothyroid or hyperthyroid. The most common thyroid disease in patients with lupus is hypothyroidism. Primary hypothyroidism occurs in 15% to 19% of patients with lupus.

Dong et al observed 363 patients with SLE and subclinical hypothyroidism (elevated TSH levels in the setting of a normal serum free T4 level) for six months and showed that a delay in treatment of subclinical hypothyroidism delays remission of SLE. In addition, the course of SLE can affect thyroid disease. Gao et al performed a case-control study of 1006 patients with SLE and showed that patients with lupus nephritis had persistent subclinical hypothyroidism.

In this edition of JAFMC, an original article on “Association of SLE and Autoimmune Hypothyroidism have published” which find the most common coexistence of SLE with Autoimmune Hypothyroidism (8%). Other coexisting autoimmune diseases like autoimmune hemolytic anemia (7%), ITP (6%), APS (5%), Type IDM (3%), MCTD (3%), RA (2%), Dermatomyositis (1%), Polymyositis (1%), Grave’s Disease (1%) were also found in their study. Therefore, careful Thyroid monitoring should be done in every case of SLE for early detection of thyroid dysfunction for effective management of both the diseases.

References


