blood sugar than normal range and 5 (29.4%) patients were found to have osteoporosis.

Conclusions: Glucocorticoids have substantial side effects in hyperglycemia and osteoporosis in the patient receiving glucocorticoid treatment. More years of taking glucocorticoids could lead to more hyperglycemia and osteoporosis. We should evaluate side effects of glucocorticoids in the patients with AIDs.

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FT3 STRONGLY CORRELATES WITH LIPID PROFILES AND DISEASE ACTIVITY IN SLE PATIENTS

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Background: Dyslipidemia is prevalent in Systemic Lupus Erythematous (SLE) patients and associated with lupus nephritis. Non-thyroidal illness syndrome (NTIS) frequently occurs in some autoimmune diseases. The incidence of dyslipidemia and NTIS in SLE patients vary in different studies and the association of NTIS and dyslipidemia in SLE patients has not yet elucidated.

Objectives: To investigate the frequency of dyslipidemia and NTIS in SLE patients and their association with laboratory parameters and SLE disease activity index (SLEDAI). To further explore the association between FT3 and blood lipid profiles in SLE patients.

Methods: This cross-sectional and prospective study included 271 patients fulfilled the ACR criteria for SLE. Forty-one patients who had a history of thyroid disease and/or familial hyperlipidemia and/or other rheumatologic diseases, and those took lipid-lowering agents or thyroid medications are excluded. Detailed laboratory parameters were collected and SLEDAI were assessed by qualified specialists of Rheumatology.

Results: Frequencies of dyslipidemia and NTIS in SLE patients are 61.8% and 57.2%, respectively. Laboratory indexes such as BUN (p<0.05), urine acid (p<0.01), serum creatinine (p<0.01), uric acid (p<0.05), CRP (p<0.05), ESR (p<0.001) and SLEDAI (p<0.01) are significantly increased in SLE patients with dyslipidemia than non-dyslipidemia. Compared to euthyroid SLE patients, SLE patients with NTIS showed substantially elevated 24 hour urine protein (p<0.001), fasting blood glucose (p<0.001), BUN (p<0.001), serum creatinine (p<0.01), uric acid (p<0.05), CRP (p<0.05), ESR (p<0.001) and SLEDAI (p<0.01). Moreover, triglyceride (p<0.01), total cholesterol (p<0.01), LDL (p<0.01) and ApoB (p<0.001) levels are markedly higher in SLE patients with NTIS than euthyroid ones, while HDL levels obviously decreased in the former group (p<0.01). More notably, the lower FT3 patients showed more severe lipid profiles and significantly higher 24 hour urine protein (p<0.001), BUN (p<0.001), serum creatinine (p<0.001), uric acid (p<0.05), and SLEDAI (p<0.05) than patients with normal FT3. FT3 levels are negatively correlated with triglyceride (r=-0.263, p<0.0001), total cholesterol (r=-0.295, p<0.0001), LDL (r=-0.273, p<0.0001) and positively correlated with HDL (r=0.180, p<0.01).

Conclusions: Dyslipidemia and NTIS are prevalent in SLE patients and strongly correlates with disease activity. SLE patients with NTIS are more likely to combine with dyslipidemia. FT3 levels significantly correlates with lipid profiles and FT3 may plays a protective role in dyslipidemia.