Introduction

Systemic lupus erythematosus (SLE) is a heterogeneous, multi-aetiological systemic autoimmune disease with various organ involvements, encompassing mild to moderate forms, and also severe progressive variants (**Mok et al., 2010**).

Although patients with SLE exhibit diverse clinical and serologic manifestation they share immunologic abnormalities resulting in loss of tolerance, auto reactivity, inflammatory sequel and organ dysfunction (**Ben-Zvi et al., 2010**).

Vitamin D is synthesized in human skin exposed to ultraviolet radiation (UVR) (**Holic , 2007**). A lesser amount is obtained from food, although dietary sources supply less than 20% of body requirement (**Cutolo and Otsa, 2008**).

Vitamin D has progressively become recognized as a pluripotent regulator of biological functions beyond its classical effects on bone and calcium homoeostasis. Most tissues and cells in the body have vitamin D receptor and enzymatic machinery to activate 25 (OH) D **(Holick , 2007**).

Low vitamin D levels have been found in patients with autoimmune diseases (**Marco et al., 2010**).

Patients affected by SLE have multiple risk factors for vitamin D deficiency (**Barnes and Bucknall , 2004**).

They have renal involvement and 1- hydroxylation is essential to make 25-OH vitamin D active and this can be disrupted in significant renal disease (**Kamen et al., 2006**).

Furthermore, photosensitivity is a key feature of SLE and the resultant avoidance of sun exposure also may result in impaired vitamin D metabolism (**Cutolo and Otsa., 2008**).