subject to dysregulation in several human diseases including some with skin manifestations [9].

According to the self-destruction hypothesis initially put forward by Lerner [7], melanocytes in vitiligo have lost an intrinsic protective mechanism that eliminates toxic intermediates or metabolites in the melanogenesis pathway. Several reports provide evidence for increased oxidative stress in the entire epidermis of vitiligo patients [7]. Although greater oxidative stress is observed in the active vitiligo group, this is probably correlated with increased intracellular reactive oxygen species (ROS) production in the tissues of these patients [10]. Excessive free radical generation, leading to lipid peroxidation in vitiligo, may be related to a decrease of superoxide dismutases (SOD) and an increase of xanthine oxidase (XO) activities. In addition, an increased level of malondialdehyde (MDA) can support these findings. Lipid peroxidation in the cellular membrane of melanocytes may play an important role in depigmentation of generalized vitiligo [11], whereas the neural hypothesis [7] was based initially on anecdotal observations suggesting that stress and severe emotional trauma may initiate or precipitate vitiligo.

The conventional treatment for vitiligo include photo chemotherapy (PUVA), phototherapy (UVB), vitamin D3 analogues, topical corticosteroids, topical immunomodulators, excimer laser, and surgery. These treatment options have limited success [12-14], and some present significant risks, including suspected increases in skin cancer risk by PUVA, skin atrophy with corticosteroids, and skin boils with UVB therapy [12-14]; so probable useful therapeutic effects of Co-enzyme Q10 will be evaluated in patients with vitiligo.

Coenzyme Q10 (CoQ10) is a fat-soluble, vitamin-like, ubiquitous compound that functions as an electron carrier in the mitochondrial respiratory chain, as well as serving as an important intracellular antioxidant. CoQ10 protects phospholipids and mitochondrial membrane proteins from peroxidation and protects DNA against the oxidative damage that accompanies lipid peroxidation [15-17]. Coenzyme Q10 is one of the antioxidants found in the skin [18]. Coenzyme Q10 in its reduced form as the hydroquinone (called ubiquinol) is a potent lipophilic antioxidant and is capable of recycling and regenerating other antioxidants such as tocopherol and ascorbate [17,19]. CoQ10 is able to act as an antioxidant against the effects of hydrogen peroxide and UVA in cultured epidermal keratinocytes and UVA in dermal fibroblasts [20].

The aim of this study was to measure the oxidative stress parameters (glutathione and MDA) in patients with vitiligo and to evaluate the effectiveness of systemic Co-enzyme Q10 in them.

Methods

Clinical Study design: This prospective, randomized, single blind and placebo study was done in the Department of Dermatology and Venereology, Al-Kadhimiya Teaching Hospital between November 2011 and March 2012. The total number of patients sharing in this study was 26. Only 24 patients completed the study successfully while 2 patients were unable to do so for unknown reasons. All included subjects have consented to be enrolled in this study and approval of College Council at Al-Nahrain Medical College was taken under order number 1093 in 1/12/2011.

All patients were subjected to detailed examination including the general, physical and mainly the skin examination. The diagnosis was made clinically by dermatologist. For all the patients at the initial visit, baseline characteristics had been made and involve age, sex, medical history, family history and drug allergy.

Participants and Setting: A new onset(less than 2 years), localized small patches vitiligo of both sexes that the affected body surface area of 10-20% and age range 12-58 years were included in this trail.

Along the course of treatment, each patient should satisfy three visits at 0, 4 and 8 weeks. In each visit, the assessment of response of vitiligo