DETECTION OF ANTI-CCP ANTIBODY IN SERA OF SMOKER PERSONS
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INTRODUCTION
Smoking is a risk factor for several diseases and has been increasing in many developing countries (Ezzi and Lopes, 2003). Smoking cessation lead to an improvement in a range of respiratory symptoms and health-related quality of life (Tillmann and silcock, 1997). Smoker counts of CD3+ T cell fall factor than non-smokers following infection with HIV (Royce and Winkelstein, 1990).

There are some immunological findings in cigarette smoker, tobacco smoke is suppress the hormonal immunity by decreasing the concentration of immunoglobulin and lysozyme, especially in men smoking for more than ten years, decreased CD16+ NK cells absolute number and elevated population of CD8+ T cytotoxic lymphocyte entailing a decrease in CD4+/CD8+ ratio (Moszcynski et al., 2001). Cigarette smoker was shown to augment the production of numerous proinflammatory cytokines such as TNF-α, IL-1, IL-6, IL-8, GM-CSF and decrease the level of anti-inflammatory cytokines such as IL-10 (Arnson et al., 2010).

Tobacco smoking has been linked to development of rheumatic disease, smoking increases the risk of dermatologic features and nephritis in systemic lupus erythematosus and multiple joint involvement in rheumatoid arthritis. Smoking is known to modulate the immune system through many mechanisms, including the induction of the inflammatory response, immune suppression, alteration of cytokine balance, induction apoptosis and DNA damage that results in the formation of anti-DNA antibody (meiret et al., 2007). Our study is to detection of anti-cyclic citrullinated peptide (CCP) antibody as autoantibody in smoker persons.

MATERIALS AND METHODS

Subjects and samples collection
This study was carried out on thirty smoker men as voluntary in Baghdad between march – may 2012, age considered was between thirty to fifty years (mean age: 38.5 years, SD.: 7.2), blood was collected from patients and each sample was used to detect anti-CCP antibody by ELISA technique. Serum was separated by blood centrifugation at 3000 rpm for five minutes, and then stored until use in deep freeze at -20 C .

Anti-CCP antibody
Serum was screened for anti-CCP antibody by using ELISA kit from Euroimmune Company (Germany) according to manufacture's instructions.

Studied groups
1. Group I: smoker persons
   According to cigarette smoking, samples were divided into two subgroups:
   - Group A: heavy smoking who smoking 20 cigarette/day aged between 30-50 years (mean age: 35.9, SD.:6.4).
   - Group B: non heavy smoker who smoke less 20 cigarette/day aged between 30-50 years (mean age: 40.5, SD.: 7.3).
2. Group II: non smoker persons aged between 30-50 (mean age: 40.2, SD.: 6.7).

Statistical analysis
Data were analyzed with student T- test to evaluate the possible differences between the studied groups, P value < 0.05 was considered significant, mean ± SD. was calculated.
for all studied groups. The data was analyzed by using statistical software SPSS version 13.0.

RESULTS AND DISCUSSION

The current study was done to detect the concentration of anti-CCP antibody in sera of smoker persons (group I), the results that compared with healthy men (non smoker persons) (group II) showed that the concentration of anti-CCP antibody in group I was higher than in group II, which was (30.8 ± 19.5 RU/ml) and (2.7 ± 1.3 RU/ml) respectively as shown in figure (1), as well as significant differences were detected between these two groups (P<0.05).

![Figure 1](image1.png)

**Figure 1** Anti-CCP antibody concentration in studied groups

Group I was divided into two subgroups according to cigarette smoking: group A and group B, which the concentration of anti-CCP antibody in sera of group A showed high concentration (48.2 ± 17.6 RU/ml) compared with group B (17.5 ± 4.3 RU/ml) as shown in figure (2), statistical analysis showed that significant differences between these two subgroups (P<0.05).

![Figure 2](image2.png)

**Figure 2** Anti-CCP antibody in Group I

Our results agree with some previous studies which showed that tobacco exposure increases the risk factor for anti-CCP antibody only in shared epitope positive patients with rheumatoid arthritis (Rasker et al., 2006). Anti-CCP antibody is more specific for detecting rheumatoid arthritis and it is presence early in the disease process, and ability to identify patients who are likely to have sever disease and irreversible damage (Niewold et al., 2007). Shared allele-smoking interaction was present in anti-CCP positive and RF positive rheumatoid arthritis (Bang et al., 2010).

Our results also agreed with other previous studies that showed there was evidence of a significant additive interaction between shared epitope status and heavy smoking in rheumatoid arthritis risk (Mikuls et al., 2010). It can be concluded that smoking related to alternate of cytokine balance, the increased risk of infection and modification of auto antigen by citrullination may contribute to development of rheumatoid arthritis, this lead to support that smoking is involved in the pathogenesis of autoimmune disease and we can recommend further studies to concern main facts and hypothesis in the development of RA in connection with smoking.

References


