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## Anticardiolipin and Antiphospholipid Antibodies in Iraqi Patients with Angina Pectoris

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TO THE EDITOR

Angina is a symptom of coronary heart disease and comprises different types including stable and non stable angina. Many factors play a role in unstable angina. Among which are aCL, aPL, ANCA, and anti-dsDNA, that may act in the induction of immunological response leading to the development of unstable angina. Immuno-inflammatory activity mediated by different antibodies may also have a role in unstable angina.

Anticardiolipin (aCL) antibodies are directed against cardiolipin and are among antiphospholipid groups of antibodies that associate with the antiphospholipid syndrome (1). aCL antibodies are strongly associated with venous and arterial thrombosis, both in patients with systemic lupus erythematosus (SLE) and in patients without any apparent autoimmune diseases (2). Much evidence has been reported that such antibodies are associated with thromboembolic manifestations such as cerebral or myocardial infarctions, pulmonary embolism, deep venous thrombosis, intrauterine fetal death due to placental infarction, neurological defects and thrombocytopenia (3). aCL antibodies may be involved in a number of vascular diseases including coronary artery diseases (CAD) or stroke (4). Recently the presence of antiphospholipid (aPL) antibodies in acute coronary syndrome (ACS) has also been assessed (5).

Sixty patients diagnosed to have unstable angina were admitted to the Iraqi center for heart diseases in the specialized surgery hospital and 30 subjects with stable angina and 20 healthy controls were included in this study. aCL and aPL were detected by commercial ELISA assay. This study showed that there was a higher prevalence of aCL antibody in unstable angina patients group as compared to both stable angina and healthy control groups ( $p=0.026$ ).

The IgG aPL among the three groups were assessed and the means of absorbances were compared. A significant difference was found between IgG aPL in unstable angina patients compared with stable angina patients ( $p< 0.05$ ) Our results showed high prevalence of aCL in angina patients indicating that an inflammatory process might play a role in the pathogenesis of this disease.

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This finding is consistent with that of Levy et al (6) who showed that the prevalence of aCL increases with premature atherosclerosis and reaches up to 11% and that of Taylor et al who demonstrated that about 26% of the general population, admitted for vascular surgery, have positive aCL (7).

We observed that about 10% of healthy control group had positive aCL and this is in agreement with the work of Schved et al (8).

Yet Phadke et al reported that aCL does not have an essential role in ischemic heart disease (9). However an association of aCL antibodies with coronary artery disease has also been shown in several but not all studies.

Antiphospholipid IgG antibody represents a chronic immunity, representing either a reaction to a primary endothelial injury or a primary role in vascular occlusive disease (10). Therefore in patients with unstable angina the possible scenario may involve endothelial and membrane perturbations exposing neoantigens and adhesion molecules and resulting in binding of aPL. Besides, genetic predisposition may be a link between the recurrent endothelial insults and the endogenous immune reactions (11).

It is possible that antiphospholipid antibodies including aCL (anti-beta GPI) antibodies, may have contributed to the formation of atherosclerotic lesions (12).

aPL (IgG,IgM) was found to be more prevalent in males than in females, consistent with findings of Veres et al (13).

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