The Egyptian Journal of Critical Care Medicine 5 (2017) 17–22

Contents lists available at ScienceDirect

The Egyptian Journal of Critical Care Medicine

journal homepage: www.sciencedirect.com

Original article

Correlation of anti-cardiolipin antibodies with right ventricular systolic strain in systemic lupus erythematosus patients

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ARTICLE INFO

Article history:
Received 9 October 2016
Revised 9 January 2017
Accepted 9 January 2017
Available online 18 April 2017

Keywords:
Anti-cardiolipin antibodies
Right ventricular strain
Systemic lupus erythematosus

ABSTRACT

Introduction: The association between anticardiolipin antibodies (aCL) and cardiac disease in the presence of systemic lupus erythematosus (SLE) has been reported in various clinical trials. However, the correlation between these auto-antibodies and right ventricular (RV) function has been inadequately investigated.
Objective: The present study investigated the possible correlation of the plasma anticardiolipin antibodies, as a marker of autoimmune phenomenon, with RV functions, assessed by right ventricular speckle tracking, in patients with systemic lupus erythematosus independent of significant pulmonary hypertension, systolic dysfunction or valvular disease.
Methods: Forty-six SLE patients and 20 healthy controls were enrolled in our study and submitted thorough history, complete clinical examination then clinical scoring according to SLEDAI-2K score and then laboratory investigations particularly plasma anticardiolipin Ig_G or Ig_M antibodies. Then echocardiography was done to assess cardiac dimensions, left ventricular systolic functions, right ventricular functions and lastly speckle tracking for assessment of the right ventricular systolic strain.
Results: Most of the study patients were young adult females with long-standing SLE (mean = 26 ± 3.1). All study patients had a high clinical SLE score (>6). All patients were normotensives and non-diabetics. No significant correlation was found between anticardiolipin titre and left ventricular dimensions or systolic functions. Significant negative correlation was found between RV strain and plasma level of both anticardiolipin Ig_M and Ig_G.
Conclusion: The present study identified that with the use of 2D speckle tracking in patients with SLE, right ventricular systolic function was significantly diminished with rising plasma titre of autoimmune (Ig_G or Ig_M) antibodies independent of cardiovascular risk factors.

Abbreviations: aCL, anticardiolipin antibodies (aCL); Apl, anti-phospholipid antibodies; EDD, end-diastolic diameter; EF, ejection fraction; ESD, end-systolic diameter; FAC, Fractional Area shortening; FS, Fractional shortening; Hb, hemoglobin; IVS, interventricular septum; MA e′/a′, medial annulus e′/a′; MAS, medial annular systolic velocity; PASP, pulmonary artery systolic pressure; PWD, posterior wall diameter; RIMP, right ventricle index of myocardial performance; RV GLSS, RV global longitudinal systolic strain; RV, right ventricle; SLE, systemic lupus erythematosus; TA e′/a′, tricuspid annulus e′/a′; TAPSE, tricuspid annular plane systolic excursion; TAS, tricuspid annular systolic velocity; WBCs, white blood cells.

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Peer review under responsibility of The Egyptian College of Critical Care Physicians.

Production and hosting by Elsevier

http://dx.doi.org/10.1016/j.ejccm.2017.01.001
2090-7303/

1. Introduction

Systemic lupus erythematosus (SLE) is an autoimmune disease characteristically affects both innate and adaptive immune mechanisms. Multiple systems and organs may be involved in the primary autoimmune processes [1]).

Although SLE frequently and classically affects the skin, joints, kidneys, and blood elements, early lung and cardiac involvement, although not rare, is mostly subclinical and thus usually escapes detection [2].

Several autoantibodies such as antiDNA, anti-phospholipid antibodies (apl), antiSSA (Ro antibodies) and anti-endothelial cell antibodies present in patients with SLE can mediate cardiac damage. These autoantibodies have either a direct effect on the heart tissue or, trigger mechanisms causing heart damage. For example; apl can contribute to cardiac damage through enhancing atherosclerosis phenomena, causing thrombosis of coronary arteries or starting an immune-complex mediated reaction with deposition at the valve level [3].

The association between anticardiolipin antibodies (aCL) and cardiac disease in patients with systemic lupus erythematosus (SLE) has been reported in various clinical trials [4–6], reported early as valvular affection, including verrucous valvular thickening, global valvular thickening, and mitral or aortic regurgitation.

There is also some evidence that aCL may also be accompanied by a decrease in the left ventricular systolic and diastolic function [7,8]. Probably, some of these effects may be independent of valvular affection. Furthermore, there is a rare knowledge about the right ventricular function detected by Speckle tracking in patients with SLE.

The aim of the present study was to evaluate the right ventricular systolic function using 2D speckle tracking in patients with SLE and its correlation with aCL titre in the absence of systolic dysfunction by standard echocardiography, significant valvular disease or significant pulmonary hypertension.

2. Methods

2.1. Study patients

Forty-six patients with SLE and 20 healthy control subjects were involved in our study. Patients were consecutively enrolled between May 2015 and December 2015 from the rheumatology unit, internal medicine department, Assiut University hospitals. 6 patients were excluded from the study due to insufficient clinical data; other exclusion criteria were clinical manifestations of right or left ventricular failure, arrhythmias, severe valvular disease, significant pulmonary hypertension, liver disease, significant renal impairment or renal failure, history of rheumatic fever, possible coronary artery disease, diabetes, hypertension and whom the 2D STE image quality was inadequate. Written and informed consent was obtained from all of the study participants. The study was approved by the local ethical committee of the Assiut university Hospital.

All patients were subjected to thorough history including components of SLEDAI-2K score as history of seizures, psychosis, visual disturbances etc. Complete physical examination was done particularly cardiovascular system examination.

2.2. Echocardiography

All of the echocardiographic studies were done using a commercial scanner (iE33; Philips Medical System, USA). Real-time 2D echocardiographic imaging was performed by a S4 transducer. Five consecutive cardiac cycles were recorded. Then, an off-line analysis of the recorded images was done using (CMQ, Q-Lab 9, Philips Medical System, USA). An experienced echocardiographer obtained the images using a standard protocol, and another researcher who was blinded to the clinical data of the study subjects, analyzed the data. The LV dimensions (IVS, PWD, EDD, ESD, FS and EF) were then measured according to European society of echocardiography guidelines [9]. The peak systolic S wave, the early (e′) and late (a′) diastolic peak velocities of the septal and lateral mitral annuli as well as lateral tricuspid annulus were measured by tissue Doppler imaging. Subsequently, the E′/A′ ratio was calculated to evaluate the LV and RV diastolic function. RV systolic excursion, Tricuspid annular plane systolic excursion (TAPSE), Fractional Area shortening (FAC), Right ventricle index of myocardial performance (RIMP) were used as an estimate of RV systolic function [10].

PASP was measured using tricuspid regurgitation velocity gradient, and RA pressure was estimated depending on IVC diameter and distensibility. Significant pulmonary hypertension was defined by PASP > 50 mmHg [11].

2.2.1. Assessment of global ventricular longitudinal strain

The global longitudinal systolic strain (GLSS) was assessed via the automated functional imaging method. Three apical views were recorded in each patient (apical long-axis and four- and two-chamber views) in gray scale with a frame rate of at least 50 per second. The mitral annulus and the LV apex were defined in each view. Using modified apical 4-chamber view, RV was traced to obtain RV global longitudinal systolic strain. The strain analyses were done on an offline basis (Fig. 1). The echocardiographic data were gathered and interpreted blindly to the subjects' clinical status.

2.3. Intra- and interobserver variability

For calculation of intra-observer variability 20 single measurements were repeated by the same operator one month later. To examine inter-observer variability, the same sample was re-assessed by another operator blinded with the study data.

2.4. Laboratory testing

Venous blood samples were collected from patients under standardized conditions. Whole blood was used for blood picture and ESR. Serum and plasma samples were prepared by centrifugation (3000*g* for 10 min), divided and stored in aliquots at −20 °C until analysis. Routine laboratory investigations including serum urea, creatinine, liver function tests, electrolytes, ESR and C-reactive protein were measured by standard laboratory methods. Complete blood count was done using Coulter Hmx, USA. Plasma Anticardiolipin Ig G and I gM were measured by ELISA Kit based on indirect enzyme immune reaction, from Orgentec Diagnostika GmbH Germany Ref ORG515, Lot 51544849, according to manufacturer's instruction.

3. Statistical analysis

Data were verified, coded by the researcher and analyzed using SPSS version 21*. Correlation analysis was used to test the association between cardiac strain parameters and other echocardiographic and laboratory variables (Spearman's rank correlation). A significant p value was considered when it is less than or equal 0.05. Unpaired *t*-test and Chi-square test was used to compare between patients and controls.

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