CME/CPD Certificate

This is to certify that

Eka Fithra Elfi

(first, last name, degree)

participated in the

10th International Congress on Autoimmunity (Autoimmunity 2016)

Leipzig, Germany
April 6-10, 2016

As speaker in Short Oral Presentation

Y. Shoenfeld

Yehuda Shoenfeld, MD, FRCP, MaACR
Congress President

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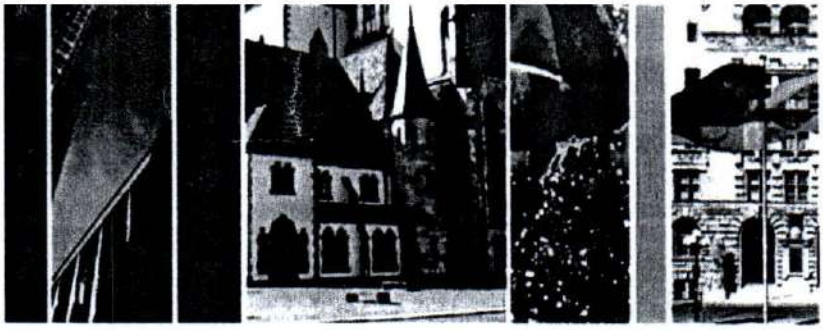
10TH INTERNATIONAL CONGRESS ON AUTOIMMUNITY

APRIL 6-10, 2016, LEIPZIG, GERMANY



December 30, 2015

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Dear Eka Fithra Elfi,

As Congress President, I extend to you a cordial invitation to participate in the forthcoming 10th International Congress Autoimmunity which is scheduled to take place April 6-10, 2016 in Leipzig, Germany.

Your contribution to this Congress will not only mutually benefit all participants but also help make this event a success.

The official Organizing Secretariat for this meeting is Kenes International - the company details can be found below.

This meeting will take place at the following venue:

Congress Center Leipzig
Messe - Allee 1
04356 Leipzig
Germany

Please feel free to use this letter for visa purposes. This letter does not constitute any financial obligation on behalf of the organizers.

We look forward to greeting you in Leipzig.

Yours Sincerely,

Y. Shoenfeld

Yehuda Shoenfeld, MD, FRCP
Congress President

Effect of hyperlipidemia to the level of IL-17, IL-6 and TGF-β

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Departement of Cardiology * and Departement of Histology ** Faculty of Medicine, Andalas University Padang-Indonesia

Abstract

Hyperlipidemia plays an important role in endothelial dysfunction and vascular inflammation which leads to atherosclerosis initiation and progression. Lipid, particularly oxidized LDL, potentially trigger an attenuating effect on the numbers and suppression properties of T-regulator by several mechanisms including decreased anti-inflammatory cytokine production. TGF-β induced T-regulator, and in the presence of IL-6 drives the differentiation of Th17 cells that release IL-17, which can promote further inflammation. To determine the effect of hyperlipidemia to endothelial, we studied the levels of IL-6, IL-17 and TGFβ -in hyperlipidemic rat. After treated with different duration of high-fat diet, serum LDL of hyperlipidemic rat groups were significantly higher than control group. Level of IL-6 were higher in the treatment groups compared to the control group and were significantly associated with increasingly level of LDL. TGF-β levels were lower in the treatment group, and associated with high levels of LDL, whereas IL-17 levels seen tend to be lower in the treatment group, although it's not significantly different from the control group. This study shows that high LDL levels in hyperlipidemia, associated with low TGF-β level as a regulator as well as high IL-6 and IL-17 as pro-inflammatory cytokines.

Introduction

The high-fat diet may cause hypercholesterolemia, that has been associated with the generation of oxidized low density lipoprotein (LDL) and reactive oxygen species in the endothelial cells of vascular wall and circulating blood. Endothelial cells modulate the permeability of the vascular membrane to the flow of LDL. Through the intervention of endothelial cells, smooth muscle cells and macrophages, LDLs can become oxidized LDLs molecules of greater atherogenicity. The process of endothelial dysfunction induced by oxidized LDL begins with a reduction in NO production and an increase in the expression of adhesion molecules, which help the monocytes adhere to the damaged endothelium and penetrate the vessel wall, to become foam cells.

Smooth muscle cells are activated by cytokines (TNF α,IL-1, IL-6, IL-8) and growth factor, and these migrate from the medial layer to the intima of the vascular endothelium, contributing to the development of atherosclerosis. Atherosclerosis is a complex inflammatory disease characterized by derangements in the vascular, metabolic, and immune systems a novel T helper subset that produces the unique cytokine, IL-17. Interleukin-17 is an inflammatory cytokine mainly secreted by Th17 cells. Transforming growth factor-β (TGF-β) induced T-regulator, and in the presence of IL-6 drives the differentiation of Th17 cells that release IL-17, which can promote further inflammation.

Material and Methods

Thirty two of male rattus norvegicus rat at eight weeks of age, were obtained from Farmacology Animal Laboratory. They were divided into four groups, the three groups were treated with high-fat feed for 10, 20 and 30 days, and the other one group as a control. The high-fat feed composed of normal rat feed added with cholesterol from 10% goat fat and 5% yellow egg. After treatment they were sacrificed for analysis. Serum LDL were measured by homogeneous enzymatic colorimetri methods, and the level of IL-6, IL-17 and TGF-β which measured by ELISA. Data were analyse as mean±standard error of the mean. This experiments were approved by the Andalas University board for Animal Ethics.

Results

Serum LDL level were significantly higher in hyperlipidemic rat groups than control group and associated with duration of high-fat diet. Level of IL-6 were higher in the treatment groups compared to the control group and were significantly associated with increasingly level of LDL.

Level of IL-17 levels seen tend to be higher in the treatment group, although it's not significantly different from the control group, whereas TGF-β levels were lower in the treatment group, and associated with high levels of LDL

Table: The level of LDL, IL-6, Il-17 and TGF-β serum control and treated group of rat with hyperlipidemia

Group	LDL (mg/dl)	IL-6 (pg/dl)	IL-17 (pg/dl)	TGF-β (pg/dl)
Control	11,15 ± 3,18	27,60±18,88	19,04±6,94	18,06±10,97
P1	28,62 ± 7,20	35,20±13,59	20,60±18,88	15,49±4,92
P2	31,60 ± 4,29	38,81±14,95	20,04±14,95	13,27±5,96
P3	33,04 ± 2,30	37,40±21,18	21,20±13,59	13,39±4,52

Discusion

In the present study, we found that LDL modulates vascular inflammation by stimulating IL-6 production that increasing vascular reactive oxygen species and vascular leukocyte infiltration, and it contributes to weight gain in response to high-fat diet. Immune responses to potential auto-antigens, such as oxidized LDL have also been shown to promote a Th17 pathway. The role of IL-17 that produced by Th17 in atherosclerosis has been a matter of debate. Whereas some investigators report that IL-17 accelerates lesion size in mouse models of human disease others have observed protective effects. In this study, the low level of IL-17 showed as protective effect. In addition, immune activity in atherosclerosis varies with gender, the phase of disease, and the level of hyperlipidemia.

TGF-β induces the differentiation of naïve T cells into the T_{reg} phenotype. When it operates in combination with IL-6, it promotes the differentiation pathway leading to T_H17 cells. This factor is likely to be IL-6 because this cytokine is produced in significant amounts by vascular as well as inflammatory cells in atherosclerotic plaques. TGF-β is a potent regulatory cytokine with diverse effects on hemopoietic cells. The pivotal function of TGF-β in the immune system is to maintain tolerance via the regulation of lymphocyte proliferation, differentiation, and survival. In addition, TGF-β controls the initiation and resolution of inflammatory responses

Conclusion

High LDL levels in hyperlipidemia, associated with high IL-6 level as pro-inflammatory cytokines as well as IL-17. While TGF-β level as a regulator lower in hyperlipidemia and associated with duration of high-fat feed. These data confirmed that hyperlipidemia can promotes inflammation process and associate with control mechanism

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