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PENTRAXIN-3 LEVELS IN TYPE 2 DM WITH CAD AND TYPE 2 DM WITHOUT CAD

Asep Sulaiman[*1], Zulfikar Lubis[1] & Santi Syafril[2]
[1]Department of Clinical Pathology, Faculty of Medicine, Universitas Sumatera Utara / Haji Adam Malik General Hospital, Medan, North Sumatera
[2]Department of Internal Medicine, Faculty of Medicine, Universitas Sumatera Utara / Haji Adam Malik General Hospital, Medan, North Sumatera

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Abstract

Background

Coronary artery disease (CAD) is the main cause of death and illness in type 2 DM patients. This is closely related to endothelial damage caused by inflammation, platelet hyperactivity and activation of coagulation factors that trigger an increase in various chemical mediators in the body. Pentraxin-3 (PTX3) is produced by a variety of cells in atherosclerotic lesions, including monocytes and macrophages, endothelial cells, vascular smooth muscle cells, fibroblasts, dendritic cells, and adipocytes.

Aim

To see the relationship between pentraxin-3 level with type 2 DM and coronary arterial disease.

Method

An analytical observational research with cross-sectional measurement method. A total of 38 subjects with sepsis, were tested for pentraxin 3 serum level then analyzed statistically.

Result

Of the 38 patients who were the study sample, 19 type 2 DM patients without CAD had a median pentraxin-3 level of 2.45 ng / mL while 19 other patients who had type 2 DM with CAD median pentraxin-3 levels were 8.68 ng / mL. By using statistical analysis to see the difference between the two it was found p = 0.005.

Conclusion

There was a significant difference between the pentraxin-3 levels of type 2 DM patients with CAD and without CAD. Type 2 DM patients with CAD had higher pentraxin-3 levels.

Introduction

Diabetes Mellitus (DM) is a disease that is a public health problem, both globally, regionally, nationally and locally. One type of metabolic disease that is always experiencing an increase in sufferers every year in countries around the world.[1]

Diabetes that is not managed properly will result in vascular complications which are divided into macrovascular complications such as coronary heart disease, peripheral vascular disease and stroke, microvascular disease such as retinopathy, nephropathy and neuropathy. The mechanism of coronary heart disease in diabetes is very complex and the risk of atherosclerosis is influenced by many factors, including hypertension, hyperglycemia, total cholesterol levels, LDL cholesterol levels (low density lipoprotein), HDL cholesterol (high density lipoprotein) levels, triglyceride levels, smoking, lack of physical exercise, male gender, age (aging), family history of disease, and obesity.[2]

In patients with diabetes mellitus, it is closely related to endothelial damage, this is because type 2 diabetes is closely related to inflammation, damage to vessel endothelium, platelet hyperactivity and activation of coagulation factors, causing increased interleukin-1 beta (IL-1β), tumor necrosis factor- alpha (TNF-α), VEGF, IL-8, IL-6, IL-8, IL-10, IL-13, VEGF and chemoattractant protein-1 (MCP-1) monocytes, also induced by LDL oxidation, cells Smooth muscle cells, platelet hyperactivity, growth factors that eventually lead to atherosclerosis, which is

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one of the macrovascular complications that often occurs in type 2 DM patients, namely coronary arterial disease (CAD).[3]

Pentraxin-3 (PTX 3) is an evolutionarily preserved acute-phase inflammatory glycoprotein in the same family of cardiovascular biomarkers C-reactive protein (CRP). PTX 3 is an acute phase protein superfamily that induces short pentraxin such as CRP or long pentraxin such as pentraxin 3. PTX 3 is produced by a variety of cells in atherosclerotic lesions, including monocytes and macrophages, endothelial cells, vascular smooth muscle cells, fibroblasts, dendritic cells, and adipocytes. Meanwhile, CRP is mainly produced in the liver. These findings suggest that plasma PTX 3 concentrations reflect local inflammation at the site of atherosclerotic lesions. Thus, PTX 3 is considered a candidate for a marker of vascular inflammation for evaluating vascular complications.[4]

This study aims to assess pentraxin-3 levels in type 2 diabetes mellitus with CAD and type 2 diabetes mellitus without CAD

Methods

This research was using cross sectional analytic research design. The study was conducted from March to Oktober 2020. The population was patients who were admitted to Adam Malik General Hospital Medan and diagnosed with type 2 DM and CAD. The sample in this study amounted to 38 people. Pentraxin-3 levels were measured using the ChemWell tool using the ELISA method. Data were analyzed using statistical computer applications, with a confidence level of 95% ($\alpha = 0.05$).

Result

In this study, the characteristics of patients based on gender were obtained as many as 26 men (68.4%), and 12 women (31.6%). From this study also obtained the mean age value of all study patients was 57.34 years

Table 1. Characteristics of patient

Variable		n	%	Mean±SD
Age (Year)				57,50±8,71
Sex				
	Men	26	31,6	
	Women	12	68,4	

From all study samples, it was found that the mean arterial lactic acid level in septic patients who entered the ICU was 3.45 mmol / L with an average SOFA score of 6 (Table 2).

Table 2. The results of measurement of Pentraxin-3 levels and Glucose Metabolism Profile of Type 2 DM

Variable		Median(Min-Max)
Pentraxin-3 Level (ng/mL)		5,74(0,43-22,13)
Glucose Metabolism		
	HbA1C(%)	7,55(5,00-11,50)
	Fasting Glucose (mg/dL)	124(68-231)
	2 Hour post prandial glucose (mg/dL)	200(102-381)

By using the Mann Whitney test to assess the comparison of Pentraxin-3 levels in type 2 DM patients with CAD with type 2 DM patients without CHD, the value of $p = 0.005$ was obtained. This shows that there is a significant difference between Pentraxin-3 levels in type 2 DM patients with CAD and type 2 DM patients without CAD.

Table 3. Comparison of Pentraxin-3 levels in type 2 DM patients with CAD with type 2 DM patients without CAD

Variable	Patient Categorize		p
	Type 2 DM with CAD	Type 2 DM without CAD	
PTX 3 level (ng/mL)	8,68(1,50-21,93)	2,45(0,43-22,13)	0.005*

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*Mann Whitney test

Discussion

Based on the results of statistical tests with the Mann Whitney test, there was a significant difference between Pentraxin-3 levels in type 2 DM patients with CHD and type 2 DM patients without CHD (p = 0.005). In the group of type 2 DM patients with CHD, the median value of Pentraxin-3 was 8.68 ng / mL, while in the type 2 DM group without CHD, it was found that the level was lower with a median value of 2.45 ng / mL.

This could be because PTX3 in a proatherogenic event could be associated with possibly an increase in the inflammatory status in the vessel walls, which could contribute to the atherogenic process. [5]In contrast, overexpression of GMO PTX3 has been found to result in greater resistance to lipopolysaccharide toxicity and abrasions and prickle secretions. There is also evidence that PTX3 can modulate inflammation-related tissue damage. [6]

Other evidence suggests that PTX3 provides an important role in modulating the cardiovascular system in humans and in experimental models. [7] In particular, there are conflicting opinions regarding the effect of PTX3 on cardiovascular disease (CVD) as several observations point to the cardiovascular protective effects of PTX3. [8]

PTX3 has also been found to provide a protective effect against atherosclerosis. As a link between PTX3 and the cell adhesion molecule P-selectin in atherosclerotic lesions. It is possible that PTX3 exerts some of these effects through protein association. For example, neutrophils on P-selectin in venules at the site of infection or injury receive signals for PTX3 release and stimulate PTX3 production then selectively bind to Pselectin expressed locally by paracrine, whereas separation of these complexes is slowed by increased binding avidity due to the multimeric nature of the PTX3. As more neutrophils are produced, they release more PTX3, which then binds to more P-selectin molecules. It is a local negative feedback system that reduces neutrophil tethering, accelerates rolling, and increases immune system detachment. Indeed, PTX3 expression has been found to reduce neutrophil counts in P-selectin in vitro in a concentration-dependent manner, whereas in vivo injection of PTX3 has been shown to reduce neutrophil counts in mouse thrombin-stimulated mesenteric venules due to PTX3 being a competitive inhibitor between P-selectin and P-selectin glycoprotein 1 (PSGL-1) bonds. [6]

Conclusion

From this study it can be concluded that There was a significant difference between the pentraxin-3 levels of type 2 DM patients with CAD and without CAD. Type 2 DM patients with CAD had higher pentraxin-3 levels.

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