Int J Biol Med Res.2017;8(3):6020-6022

Contents lists available at BioMedSciDirect Publications

International Journal of Biological & Medical Research

Journal homepage: www.biomedscidirect.com

Original Article
The Relationships Between Sorbitol Dehydrogenase (SDH) Level and Diabetic Rethinopathy in Diabetes Melitus Type-2 Patients

Ramzi Amin[a], Reyno Satria Ali[b], Rachmat Hidayat[c], Hikmat Permana[d], Arief S Kartasasmita[e], Dany Hilmanto[f]

[a]*Ophthalmology Department, Medical Faculty, Universitas Sriwijaya, Palembang, Indonesia*
[b]*Ophthalmology Department, Medical Faculty, Universitas Sriwijaya, Palembang, Indonesia*
[c]*Pharmacology Department, Medical Faculty, Universitas Sriwijaya, Palembang, Indonesia*
[d]*Ophthalmology Department, Medical Faculty, Universitas Padjadjaran, Bandung, Indonesia*
[e]*Internal Medicine Department, Medical Faculty, Universitas Padjadjaran, Bandung, Indonesia*
[f]*Pediatric Department, Medical Faculty, Universitas Padjadjaran, Bandung, Indonesia*

ARTICLE INFO

Keywords:
Sorbitol dehydrogenase
Diabetic Rethinopathy
Diabetes Melitus

ABSTRACT

Background: The conversion of sorbitol accumulation to fructose by SDH triggers the osmotic damage in retinal endothelial cells and pericyte through the activation of advanced glycation end products (AGEs), oxidative-nitrosative stress, Protein Kinase C (PKC) pathway activation, inflammation, and the imbalance of growth factor. The osmotic damage eventually leads to DR (Diabetic Rethinopathy). Methods: This is a case-control study. DM type 2 patients who had the direct ophthalmoscopy and fundus imaging in dr. Mohammad Hoesin Central General Hospital opthalmology outpatient in Palembang was selected as a subject in this study.Patient's blood sample was collected from a mediancubital vein for 3 mL and stored in ethylene diaminetetraacetic acid (EDTA) coated tube for SDH assay by ELISA. Results: The average level of SDH in DR subjects was 13,3 ±7,8 ng/mLand the average level of SDH in Non DR was 10,7±2,3 ng/mL, p=0,044.There was no significant difference in subjects with DR and subjects in control group in level of SDH by chi square-test, which the level of SDH was devided into two groups, higher than 11,18 ng/mL and lower than 11,18 ng/mL by ROC curve. Conclusion: There was significant difference in subjects with DR and subjects in control group in level of SDH. The average of SDH level in DR subjects were more higher than Non DR subjects.

Introduction

Diabetic retinopathy (DR) has the potential to cause blindness in diabetes mellitus (DM) patients. The probability of this complication is escalating along with the duration of DM[1,2,3]. This complication prevalence in DM patients is 28,5 % with a threat to eye vision capability for about 30%, and 15% of them are blind2,3. The Diab Care Asia report in 2008, involving 1758 DM patients in 18 primary and secondary health service in Indonesia, revealed that 42% of DM patients will get retinopathy complication, in which about 6,4% is proliferative DR[4].

Blindness caused by DR is related to the obstruction and damage in micro blood vessels in the retina. Chronic hyperglycemia condition triggers the cascade of physiology and biochemical alteration which leads to microvascular damage and retina disfunction5. The Diabetes Control and Complications Trial (DCCT) and the United Kingdom Prospective Diabetes Study (UKPDS) stated that there is strong relevance between chronic hyperglycemia with DR, but the mechanism is not yet clear5,6. Some of the biochemical pathways regarding the potential links between hyperglycemia and DR were already investigated. Under the hyperglycemic condition, glucose flux will increase via polyol pathway, where the aldose reductase (AR) enzyme will deplete the glucose supply as they are converted to sorbitol, and finally, are converted to fructose by sorbitol dehydrogenase (SDH)[5].

Quick conversion of sorbitol accumulation to fructose by SDH triggers the osmotic damage in retinal endothelial cells and pericyte through the activation of advanced glycation end products (AGEs), oxidative-nitrosative stress, Protein Kinase C (PKC) pathway activation, inflammation, and the imbalance of growth factor. The osmotic damage eventually leads to DR[5,6]. The experimental study revealed some important connection between DR and SDH, which have an important role in the second part of polyol pathway[5,7]. Amano et al (2007) showed that SDHwas overexpressed in mammalian pericyte culture after exposed to high dose of glucose and eventually stimulated reactive oxygen. AR inhibitor and antioxidant significantly block the bad effects of excess SDH by

* Corresponding Author : **Ramzi Amin**
Ophthalmology Department, Medical Faculty, Universitas Sriwijaya, Palembang, Indonesia
Email: ramzi.amin@rocketmail.com