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PATHOPHYSIOLOGICAL BASIS OF ERECTILE DYSFUNCTION IN DIABETES MELLITUS: A REVIEW

[1]Abhijit De*, [2]Mamta Farswan Singh, [3]Vinod Singh, [2]Veerma Ram, [2]Shradha Bisht

[1]Department of Pharmaceutical Technology, Bengal School of Technology, Sugandha, West Bengal, INDIA

[2]Department of Pharmacy, SBS PG Institute of Biomedical Sciences, Balawala, Dehradun, INDIA

[3]Faculty of Pharmacy, Gurukul Kangri University, Haridwar, INDIA

Abstract

Erectile dysfunction is a common complication of diabetes mellitus and also is the first symptom of as yet undiagnosed diabetes. The Massachusetts Male Aging Study (MMAS) was the first major epidemiological investigation to study the prevalence of ED in diabetes. According to MMAC the incidence of ED was correlated with glycemic control and increases with increasing age, duration of diabetes and deteriorating metabolic control, and was higher in individuals with type 2 diabetes than those with type 1 diabetes. The pathophysiology of ED in diabetes is multifactorial including vascular and neural factors being equally implicated. In diabetic men, peripheral vasculopathy and neuropathy are intimately involved in the development of ED. Diabetic patients associated with insufficient control of glycemic level extremely suffer from disruption of endothelial functions, generation of increased level of free radicals, loss of control in the parasympathetic and non adrenergenic non cholinergic nerves (NANC). In diabetic patients hypogonadism, autonomic neuropathy, arterial insufficiency, low testosterone, changes in expression of protein kinase C, RhoA-Rho kinase Ca^{2+}-sensitization pathway results in vascular damages of penile smooth muscle which are more or less related to erectile dysfunction. Penile tissue from diabetic men with ED demonstrates impaired neurogenic and endothelium-mediated relaxation of smooth muscle, increased accumulation of advanced glycation end products (AGEs) and upregulation of arginase which lead to decrease in the level of NO in corpora cavernosa. Still there is a need to understand the pathophysiology of ED in diabetic patients and to make an effort to diagnose and treat ED for improving the quality of life of the patients of diabetes. This review aimed to provide an update of the normal physiology of penile erection and the pathophysiological mechanisms of erectile dysfunction (ED) in diabetes patients.

Keywords: Diabetes Mellitus, Erectile dysfunction, Oxidative stress, Advanced glycation end products, Arginase, Rho Kinase, Tumor Necrosis Factor.

Corresponding Author

Abhijit De
Assistant Professor, Dept. of Pharmaceutical Technology
Bengal School of Technology, Sugandha, West Bengal, INDIA
Email: abhi8981@gmail.com
Phone: *+91 8274847571*