Diabetes Complications in Hypertension

email:Rgronaldgreat@gmail.com

Many forms of diabetes complications such as diabetic nephropathy, diabetic retinopathy, stroke, and DM-related cardiovascular disorders such as atherosclerosis are closely associated with hemodynamic variations and hypertension.

Emerging studies have well incontestible that the cooccurring presence of high blood pressure may be a main upstream event and potent risk issue which might induce or exacerbate the progression of polygenic disorder complications. according that in depth uncontrolled hemodynamic aberrations accompany diabetic kidney disease because of the morphological and useful alteration within the kidneys and general and intraglomerular high blood pressure in patients with polygenic disorder. However, capillary vessel hyperfiltration that is taken into account a hemodynamic abnormality in kidneys conjointly acts as associate degree freelance risk issue for diabetic kidney disease in patients with polygenic disorder.

Moreover, hypertension (HTN) in patients with diabetes is associated with cardiovascular complications and atherosclerosis, which markedly increase the risk of stroke and myocardial infarction.

Likewise, other prevalent forms of diabetes complications such as diabetic neuropathy and diabetic retinopathy are potentially influenced by molecular pathways induced by hemodynamic changes and systemic hypertension.

Therefore, the management of hypertension in patients with diabetes is of crucial importance for researchers and physicians.

There is significant evidence that GLP-1RAs and DPP-4i enhance the insulin activity and the glucose uptake in animal and human muscle. It has been proposed that GLP-1 enhances glucose disposal in an insulin-independent mechanism. The GLP-1 receptors are expressed in the brain and β-cells of the pancreas where GLP-1 exerts multiple actions. In the pancreas, it stimulates insulin secretion by many molecular pathways including the release of cyclic adenosine monophosphate (cAMP) by activating β-arrestin-1 (βARR1), activates the voltage Ca^{2+} channels, and induces the Ca^{2+} influx which raises the intracellular Ca^{2+} and stimulates the insulin release. GLP-1 also stimulates β-cell proliferation by downregulating PI3-K, mitogen-activated protein kinase (MAPK), and p38. There is evidence suggesting that GLP-1 possesses anti-inflammatory properties.

It suppresses inflammation by reducing secretion of inflammatory cytokines such as interleukin-1 β (IL-1β), tumour necrosis factor-β (TNF-β), and interleukin (IL). Moreover, GLP-1RAs reduce the stress in the endoplasmic reticulum (ER) by modulating the protein kinase R-like endoplasmic reticulum (PERK) pathway and activate the transcription factor 4 (ATF4) and CHOP (C/EBP homologous protein).

There is growing evidence that GLP-1 A reverses the vascular remodelling by downregulating the matrix metalloproteinase 1 (MMP1), extracellular-regulated protein kinase 1/2 (ERK1/2), and nuclear factor kappa-β (NF-Kβ). Therefore, via this mechanism, GLP-1RAs are reducing cardiac and vascular inflammation.

Some evidence suggested that GLP-1 agonists and DPP-4i can influence the hemodynamic state and modify BP. We have reviewed all possible mechanisms associated with this class of agents and hemodynamics. Since these two antidiabetic agents have the same basis of molecular effects, we have reviewed them together.

Vascular endothelial cells have a significant role in the homeostasis of cardiovascular function and BP homeostasis. GLP-1 agonists may improve vascular endothelial function in the diabetic milieu. It was found that GLP-1 stimulated acetylcholine-induced vasodilatation, improved vascular relaxation, reduced diastolic BP, and has direct beneficial effects on endothelial function in patients with T1DM. Similarly, reported that GLP-1 agonists improve endothelial cell function and regulate vascular contractions by promoting nitric oxide (NO) release and suppressing oxidative stress.