Involvement of genomics and environmental factors in diabetes and hypertension.

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Introduction

Atherosclerosis and its consequences, such as heart attacks and strokes, are exacerbated by hypertension and diabetes. The genesis and disease processes of diabetes and hypertension are very similar. Only 42% of patients with diabetes had normal blood pressure, and 56% of people with hypertension had normal glucose tolerance, according to the Hong Kong Cardiovascular Risk Factor Prevalence Study. Hypertension affects about 30% of type 1 diabetes patients and 50% to 80% of type 2 diabetic patients in the United States.

Genomics

Genome scans comprising hundreds of participants and controls indicated a vast number of genes with small impacts, rather than the limited number of genes with substantial effects that had been expected. Variants in the angiotensinogen, adrenomedullin, apolipoprotein, and alpha-adducin genes have been linked to diabetes, hypertension, dysglycemia, and metabolic syndrome [1].

Aside from genetics, the environment plays an essential role in the development of diabetes and hypertension. The foetal period, as well as lifestyle factors like as diet and physical exercise, are all environmental factors. Three variables may predispose a foetus to cardiometabolic syndrome in adulthood: gestational diabetes, foetal starvation, and high birth weight. An unhealthy lifestyle includes high sodium, alcohol, and unsaturated fat intake, as well as smoking, lack of physical activity, and mental stress.

Inflammation and oxidative stress

Diabetes and hypertension both cause a low-grade inflammatory response. Chronic periodontitis is a hidden risk factor for diabetes, hypertension, cardiovascular disease, and the metabolic syndrome. Diabetes and hypertension might be considered chronic inflammatory disorders in some aspects.

Inflammatory markers (e.g., C-reactive protein (CRP)) are elevated in individuals with diabetes, hypertension, and the metabolic syndrome, and they can also predict disease progression. In vascular pathophysiology, the local Renin-Angiotensin-Aldosterone System (RAAS) is extremely important. In the shoulder of coronary artery plaques, Angiotensin-Converting Enzyme (ACE) is expressed. Angiotensin II (Ang II) is responsible for a major part of vascular inflammation and oxidative stress [2].

It activates Rho/Rho kinase, Protein Kinase C (PKC), and mitogen-activated protein kinase by stimulating NADH/NADPH oxidase (MAPK). Ang II also inhibits proinflammatory transcription factors including nuclear factor-B (NF-B), which leads to the production and release of Reactive Oxygen Species (ROS), inflammatory cytokines (e.g., interleukin-6 [IL-6]), chemokines, and adhesion molecules. Endothelial dysfunction and vascular damage are the results of these processes.

Through antioxidant, anti-inflammatory, antiproliferative, antihypertrophic, and antifibrotic activities, Peroxisome Proliferator-Activated Receptor (PPAR) activators reduce blood pressure, generate beneficial effects on the heart, and ameliorate endothelial dysfunction. The mRNA and protein of PPAR- and PPAR- are downregulated by Ang II, resulting in a loss in PPAR anti-inflammatory capability and inflammation activation. Independent of their metabolic activities, PPAR- and PPAR-activators have been shown to have cardiovascular preventive effects. Recent trials with dual PPAR activators, however, have thrown doubt on their clinical usefulness in cardiovascular protection when compared to presently marketed PPAR activators [3].

Insulin resistance

Insulin is a pleiotropic hormone that plays a key role in hypertension, diabetes, and the metabolic syndrome. Insulin's major metabolic activities are to accelerate glucose uptake in skeletal muscle and the heart, as well as to decrease glucose and Very Low-Density Lipoprotein (VLDL) synthesis in the liver. Insulin secretion is reduced during fasting, resulting in increased glucose synthesis in the liver and kidneys (gluconeogenesis) and increased glycogen to glucose conversion in the liver (glycogenolysis) [4].

Insulin is secreted from pancreatic beta-cells after a meal, which suppresses gluconeogenesis and glycogenolysis. Insulin increases cardiac output and glucose transport and utilisation in peripheral tissues via stimulating the Sympathetic Nervous System (SNS). Insulin also inhibits glucose release from the liver, inhibits the release of Free Fatty Acids (FFAs) from adipose tissue, and stimulates the process of amino acid

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incorporation into protein, among other metabolic functions. Insulin-mediated glucose absorption by muscle varies more than sixfold in apparently healthy people, with half of the variation due to genetics and the other half due to variances in adiposity and physical fitness. Insulin resistance affects the majority of individuals with type 2 diabetes, and roughly half of those with essential hypertension are insulin resistant. Insulin resistance is thus a significant relationship between diabetes and hypertension.

Mental stress and sympathetic nervous system

Stressors are inherent or extrinsic stimuli that disrupt physiology and psychology, posing a health risk. Modern stressors resulting from psychological threat (e.g., work stress, marital abuse, and natural disasters) are more sustained than physical stressors. Chronic mental stress, which is common in today's society, is linked to physiologic and psychological problems, and can lead to diabetes and hypertension indirectly. Chronic stress stimulates the Sympathetic Nervous System (SNS), which raises pulse rate and cardiac minute output while also activating the RAAS, another essential presser mechanism in the human body. The development of poor glucose and lipid metabolism is also linked to increased SNS activity. Understanding the functions of the SNS and RAAS in the development and management of hypertension, metabolic syndrome, and diabetes requires further research, in patients with diabetes and hypertension, there is also a relationship between mental stress and obesity. Psychosocial factors, such as chronic stress, have been linked to a high prevalence of hypertension in obese people. Obesity, hypertension, and chronic stress have all been linked to the hypothalamic–pituitary–adrenal axis [5].

Conclusion

SNS, RAAS, oxidative stress, adipokines, insulin resistance, and PPARs are all prevalent routes in diabetes and hypertension. These routes interact and impact one another, perhaps creating a vicious cycle. The metabolic syndrome occurs in both hypertension and diabetes. As a result, they may appear one after the other in the same person. The metabolic syndrome is caused by central obesity. For the long-term management of obesity, only orlistat is now accessible. As a result, improving one's lifestyle is still the most important factor in preventing and treating diabetes and hypertension.

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