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ORIGINAL ARTICLE

Glucagon-Like Peptide-1 Receptor Agonists and Brain Vascular Function

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Prevention of cardiovascular events and regression of atherosclerotic changes are the primary aims of preventive cardiovascular medicine. Arterial thrombosis is caused by endothelial dysfunction, which disrupts vascular haemostasis. Glucagon-like peptide 1 (GLP-1) receptor agonists have been initially used as glucose lowering agents, but over time have been used for other indications due to their cardiorenal benefit, as well as their benefit in the regression of atherosclerosis process. The aim of this paper is to present the benefits of GLP-1 receptor agonists in the prevention of atherosclerotic changes, in the preservation of brain vascular function, and to show the possible role in the treatment of neurodegenerative diseases.

Keywords Alzheimer's disease • Atherosclerosis • Glucagon-like peptide 1 • Neurodegenerative diseases • Therapeutics

Introduction

The function of the circulation is to satisfy tissue needs, to maintain homeostasis which is necessary for survival and cell function [1]. The magnitude of blood flow through each tissue is consistent with tissue needs, cardiac output is generally determined by total local tissue flow, and blood pressure is monitored independently of local blood flow and cardiac output [1]. Normal blood flow through the adult brain per minute averages from 50 to 65 mL per 100 grams of brain mass, and or the whole brain that is 750 to 900 mL per minute, or 15% of total cardiac output [2].

Regulation of cerebral flow depends on the concentration of carbon dioxide, hydrogen ions and oxygen concentration [3]. Cerebral microcirculation is very important. An important structural property of cerebral capillaries is that they are less permeable than capillaries in other tissues in the body [3]. The walls of the small arterioles that lead to the brain capillaries are significantly contracted in people with arterial hypertension, and remain permanently contracted to prevent the transmission of high blood pressure to the capillaries [3]. In the elderly, some small arteries are already clogged, and in 10% of people the clogging is so extensive that the symptoms of a stroke are present clinically [3]. All changes are associated with the process of atherosclerosis, which, together with aortic stiffness, are associated with numerous pathological conditions of the central nervous system [3,4]. Interventions that would reduce aortic stiffness may delay memory decline among the older population [4].

Atherosclerosis forms the basis of about 80% of cardiovascular disease, and is the result of hyperlipidaemia and lipid oxidation [5,6]. Given the high percentage of potentially fatal complications of arterial thrombosis (the process progresses through life before finally manifesting as an acute ischaemic event), a correct understanding of the pathogenesis of this disease is of great importance for determining the optimal modality of prophylaxis and therapy.

Atherosclerosis is a chronic inflammatory process in the intima of the middle arteries [7]. It is characterised by thinning of the intima of the blood vessel, with the accumulation of fat and macrophages (essentially it consists of two parts, and after the accumulation of fat and macrophages, there is a process of sclerosis, when there is fibrous thickening of muscle cells, leukocytes and connective tissue) [6–8]. The mechanism of arterial thrombosis is conditioned by endothelial damage leading to platelet adhesion and aggregation

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