A short note on how insulin resistance involves in the development of hypersensitivity in diabetics

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Description

Insulin Resistance (IR) is a key factor in the pathogenesis of hypertension in Diabetes Mellitus (DM) patients. IR is the main mechanism involved in the development of HS in DM patients. In fact, it contributes to increased blood pressure in a number of ways, including increased angiotensin and aldosterone activity in tissues, increased sympathetic nervous system activity, and oxidative stress, according to which endothelial dysfunction precedes peripheral IR due to impaired blood flow in peripheral tissues. The association between IR and HS is multifactorial.IR is involved in the development of HS and atherosclerotic cardiovascular disease through 3 main mechanisms, the basic molecular etiology of IR the compensatory hyperinsulinemia that occurs in response to IR and the association between IR and some cardio-metabolic abnormalities.

Insulin acts on its target organs phosphorylating a transmembrane-spanning tyrosine kinase receptor, the insulin receptor. It binds to the subunit of its receptor activating the tyrosine kinase of the subunit of the recep- tor, causing auto-phosphorylation and phosphorylation of several IR substrates, such as IRS-1 and IRS-2. One of the mechanisms through which hyperinsulinemia causes an increase of BP is dysregulation of peripheral vascular resistance, stimulating the sympathetic system and therefore causing vasoconstriction furthermore, hyperinsulinemia contributes to the development of HS-related target organ damage, in particular, impairment of cell membrane ion exchange, enhanced sympathetic nervous and renin-angiotensin systems, suppressed atrial natriuretic peptide activities, sodium retention, and plasma volume expansion lead to chronic kidney disease, left ventricular hypertrophy and carotid atherosclerosis.

IR in obese people is primarily determined by lipotoxicity. Effects of excess lipid accumulation when energy intake exceeds energy expenditure. Elevated free fatty acids in the blood promote lipid deposition in tissues, including blood vessels, and activation of inflammatory pathways. Excess fat in adipocytes increases the secretion of pro-inflammatory/pro-thrombotic cytokines such as TNF, PAI-1 and resistin, promoting atherogenesis. There are some differences in the definitions of elevated blood pressure and HS in children provided by ESH and AAP. First, they suggest two different age categories for her. This makes the ESH-BP percentile considered up to age 15, whereas in the AAP he is only used up to age 12. Children at any stage of HS with DM should be pharmacologically treated, starting with a single drug at the lower end of the dosing range. The dose of the initial drug can be increased every 2 to 4 weeks until the dose is reached or side effects occur. According to the American DM Association (ADA), drug classes with proven cardiovascular benefits, such as renin-angiotensin system inhibitors, should be the first-line therapy of choice, unless there are absolute contraindications. These families of drugs are also useful in micro-albuminuria due to their anti-proteinuria activity. Consistent evidence suggests that IR is involved in the development of HS and atherosclerotic cardiovascular disease. Children with type 1 and type 2 diabetes and HS require special attention in diagnosis and specific treatment. In addition to lifestyle interventions, children at any stage of HS with DM should receive pharmacological treatment starting with ACE inhibitors or ARBs.

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Conflict of Interest

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