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Journal of Applied Pharmacy

Short communication

The Effect of Insulin and Hypertensive Medications in Cerebral Glucose

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DESCRIPTION

Previously, impaired glucose tolerance was thought to be a risk factor for the development of diabetes. Only recently has there been a lot of focus on the fact that poor glucose tolerance is a considerably bigger risk factor for cardiovascular disease [1]. Hypertension is common among diabetics, especially when nephropathy is present, which exacerbates all vascular problems. It should be noted that diabetic nephropathy is the second leading cause of mortality after cardiovascular disease. Thus, hypertensive medication prescription is critical for people with diabetes and cardiovascular disease [2].

However, little has been conducted on the impact of these medicines on the glucose level in the brain. The major fuel for energy creation in the brain is glucose. Insulin injection that causes blood glucose levels to fall below 3 mm for many minutes' results in mild brain impairment [3]. As a result, the brain must rely on a constant supply of glucose to sustain proper metabolic activity. However, the effect of insulin and hypertensive medications in cerebral glucose control is yet unknown. Previous findings have accumulated sufficient evidence that insulin controls the admission of glucose into the brain system [4].

The trials, however, have drawbacks in that they did not directly measure brain glucose concentration. Furthermore, there has been no study on the effect of hypertensive medications on cerebral glucose levels [5]. The metabolic activity of the grey and striatum of the brain is significantly reliant on constant glucose delivery. The striatum metabolizes glucose at a different pace than the grey matter. The relationship between cerebral glucose concentration and various glucose metabolization rates, as well as the role of insulin in controlling regional cerebral glucose metabolism in humans, remain unknown. This was impossible due to the lack of direct techniques for measuring brain glucose concentrations [6].

However, the studies had a flaw in that they did not directly detect brain glucose levels. Furthermore, no study on the effect of hypertensive medicines on cerebral glucose levels has been conducted. The metabolic activity of the brain's grey and striatum is heavily reliant on continual glucose supply [7]. The striatum processes glucose at a slower rate than the grey matter. The link between cerebral glucose concentration and different rates of glucose metabolization, as well as the role of insulin in modulating regional cerebral glucose metabolism in humans, are unclear [8].

The lack of direct procedures for monitoring brain glucose concentrations made this unfeasible. However, the studies had a flaw in that they did not directly detect brain glucose levels [9,10]. Furthermore, no study on the effect of hypertensive medicines on cerebral glucose levels has been conducted. The metabolic activity of the brain's grey and striatum is heavily reliant on continual glucose supply. The striatum processes glucose at a slower rate than the grey matter [11]. The link between cerebral glucose concentration and different rates of glucose metabolization, as well as the role of insulin in modulating regional cerebral glucose metabolism in humans, are unclear [12]. Due to the absence of direct procedures for monitoring brain glucose concentrations, this was difficult.

The capacity of the immobilized enzyme on the electroactive surface to exchange redox equivalents with the electroactive surface at an appropriate electron transfer rate governs these enzyme-based sensors [13]. Direct Electron Transfer (DET) between the enzyme's active site and the electroactive surface is the most basic electron transfer method. The DET would include Flavin Adenosine Dinucleotide (FAD), the active site in glucose oxidase (the immobilized enzyme), and the electrode surface for a glucose sensor [14]. The FADs were extensively buried inside the protein shell, resulting in a low electron transfer rate. Because the FAD centers were oxidized by dispersed O_2, the glucose concentrations could be estimated by measuring the amount of O_2 consumed or H_2O_2 created. However, because the O_2 concentration in the brain's microenvironment changes, this form of measurement creates mistakes.

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Received: 22-Jun-2023, Manuscript No. JAP-23-25259; **Editor assigned:** 26-J-2023, Pre QC No. JAP-23-25259(PQ); **Reviewed:** 10-Jul-2023, QC No. JAP-23-25259; **Revised:** 17-Jul-2023, Manuscript No. JAP-23-25259(R); **Published:** 24-Jul-2023, DOI: 10.35248/1920-4159.23.15.361

Citation: Lebbar M, (2023) The Effect of Insulin and Hypertensive Medications in Cerebral Glucose. J Appl Pharm. 15:361.

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