ISSN: 2322-0066

Research and Reviews: Research Journal of Biology

Factor's Associated with Type 3 Diabetes and Type 2 Diabetes

Barkha Gupta [1]* and Praneeth Kamarapu[2]

[1]Btech (Biotech), Amity Institute of Biotechnology, Amity University, India
[2]Department of pharmaceutical sciences, The University of Greenwich, UK

Review Article

Received: 18/08/2016
Accepted: 19/08/2016
Published: 29/08/2016

***For Correspondence**

Barkha Gupta, Amity Institute of Biotechnology, Amity University, Noida-201313, India
Tel: +9911212976;
E-mail: barkhagupta000@gmail.com

Keywords: Diabetes, Alzheimer, Hyperglycaemia, Aerophilous, Histopathological, Psychological.

ABSTRACT

There are growing link between type 2 Diabetes and Alzheimer's disease. After the review of researches related to diabetes term given Type 3 diabetes to AD. There are various factors associated with the diabetes which can lead to conditions similar in Alzheimer's disease. Risk factor included in Type 3 diabetes is genetic factor, environmental and metabolic activity. In case of type 2 diabetes Amyloid formations occurs which deposits to the brain resulting conditions like memory loss and inhibits cell to cell signalling.

INTRODUCTION

Diabetes mellitus (DM) is impaired hypoglycaemic agent secretion and variable degrees of peripheral hypoglycaemic agent resistance resulting in hyperglycaemia [1]. Early symptoms square measure regarding hyperglycaemia and embrace thirst, polyphagia, polyuria, and blurred vision. Later complications embrace vascular malady, peripheral pathology, renal disorder, and predisposition to infection. Designation is by activity plasma aldohexose [2,3].

TYPES OF DIABETES

Type 1 diabetes mellitus

In Type 1 DM (beforehand called adolescent onset or insulin - subordinate), insulin generation is missing a result of immune system pancreatic β-cell pulverization potentially activated by an ecological presentation in hereditarily defenceless individuals [4,5]. Type one polygenic disorder sometimes develops in youngsters or young adults and

can't be corrected with diet or a modification in life-style. Sort one is genetically preset, and needs daily hypoglycaemic agent injections to balance aldohexose levels [6].

Type 2 diabetes mellitus

Type 2 pair of polygenic disorder happens once the cells cannot establish or absorb the hypoglycaemic agent place out by the exocrine gland, and frequently happens with avoirdupois, and a robust case history of polygenic disorder [7]. The nice news is that in its earlier stages, Type 2 DM is less complicated to manage with diet and life-style modifications [8].

Type 3 diabetes mellitus

Type 3 DM is the term used for Alzheimer's disease. It is the case of insulin resistance to the brain.

With sort three polygenic disorder, the researchers at Brown grad school and Rhode Island Hospital discovered that hypoglycaemic agent isn't solely discharged from the exocrine gland, however conjointly the brain. This opens an entire new perspective on each polygenic disorder and Alzheimer's – the progressive disorder inflicting loss of memory, conception and even temperament changes [8,9].

Type 3 DM is the term used for Alzheimer's disease. It is the case of insulin resistance to the brain.

INSULIN RESISTANCE TO THE BRAIN

Insulin resistance (IR) is mostly seemed to as a pathological situation throughout which cells fail to reply to the conventional movements of the inner secretion hormone. The frame produces hormone once aldohexose begins to be loose into the blood from the digestion of carbohydrates in the food plan. Unremarkably this hormone reaction triggers aldohexose being taken into body cells, for use for electricity, and inhibits the frame from mistreatment lipids as energy [10].

Alzheimer's disease (AD) has characteristic histopathological, molecular, and organic chemistry abnormalities, together with cell loss; voluminous neurofibrillary tangles; dystrophic neurites; amyloid precursor macromolecule, amyloid-β (APP-Aβ) deposits; enlarged activation of prodeath genes and sign pathways; impaired energy metabolism; mitochondrial dysfunction; chronic aerophilous stress; and DNA harm. Gaining a much better understanding of AD pathological process would force a framework that mechanistically interlinks of these phenomena [11,12].

TYPE 2 DIABETES MELLITUS (T2DM) AND ALZHEIMER'S DISEASE

Type two polygenic disorders severally is one among the danger consider AD [13]. Alzheimer's sickness (AD) is that the most typical reason behind insanity in North America. Growing proof supports the thought that AD could be a metabolic sickness mediate by impairments in brain hypoglycaemic agent responsiveness, aldohexose utilization, and energy metabolism that result in exaggerated aerophilous stress, inflammation, and worsening of hypoglycaemic agent resistance. Additionally, metabolic derangements directly contribute to the structural, functional, molecular, and organic chemistry abnormalities that characterize AD, as well as neuronic loss, conjugation disconnection, alphabetic character hyperphosphorylation, and amyloid-beta accumulation. as a result of the basic abnormalities in AD represent effects of brain hypoglycaemic agent resistance and deficiency, and also the molecular and organic chemistry consequences overlap with sort one and sort two polygenic disorder, we

propose the term "Type three diabetes" to account for the underlying abnormalities related to AD-type neurodegeneration [13,14].

T2DM causes brain endocrine resistance, aerophilic stress, and psychological feature impairment, however its mixture effects fall so much in need of mimicking AD. Intensive disturbances in brain endocrine and insulin-like protein (IGF) communication mechanisms represent early and progressive abnormalities and will account for the bulk of molecular, organic chemistry, and histopathological lesions in AD [15].

It's been reviewed that brain polygenic disorder created by neural structure administration of streptozotocin shares several options with AD, together with psychological feature impairment and disturbances in neurotransmitter physiological condition [16].

Insulin functions by dominant neurochemical unharness processes at the synapses and activating sign pathways related to learning and memory.

IAPP amyloid formation in type 2 diabetes may occur under circumstances of genetic variance, which results in a relative decreased affinity of the chaperone protein pathway for trafficking of IAPP [17].

It's been suggested by Schwartz that there can be a relation between pancreatic islets and amyloid deposits in brain.

ASSOCIATION OF TYPE 3 DM WITH METABOLIC SYNDROMES

Metabolic syndrome and Alzheimer's sickness are life vogue associated disorders that depends upon numerous environmental and genetic factors.

An association between metabolic syndrome and specific single-nucleotide polymorphisms (SNPs) within the cistron INPPL1, secret writing for SHIP2, a SH2 domain-containing B vitamin 5-phosphatase concerned in hypoglycaemic agent signalling, has been delineated [18].

Resistance to hypoglycaemic agent and insulin-like protein as being a key a part of the progression of Alzheimer's sickness [19]. Whereas sort one and sort two polygenic disorder are characterized by symptom (increased blood sugar), a separate study, disbursed by the University of Pennsylvania and revealed in 2012, excluded individuals with a history of polygenic disorder, indicating that Alzheimer's will develop while not the presence of great symptom within the brain [20-22].

CONCLUSION

T3DM or AD is associated with the many factors related to type 2 diabetes. This included various metabolic disorders factors , environmental and genetic factors. AD is characterised by a discount within the utilization of aldohexose, and treatment with internal secretion has been related to improved memory. Insulin, vital in memory process, crosses the barrier and is even made in brain tissue itself. AD patients have less internal secretion and fewer internal secretion receptors than non-AD patients, and correction of internal secretion levels improves psychological feature. In some cases It can be controlled by few changes in lifestyle as diet, exercise, and medicines that cut back aldohexose levels, as well as hypoglycemic agent and oral antihyperglycemic medication.

REFERENCES

1. Janet LP and Le Anne MH. Validity of the Community Integration Questionnaire as a Measure of Participation in Persons with Diabetes Mellitus. J Diabetes Metab. 2016;7:2.
2. Tagliente I, et al. Management and Treatment of Type 1 And 2 Diabetes. State of Art Gen Med (Los Angeles). 2016;4:2.
3. Bayramova AN. Gastroenterological Diseases as a complications of Type 2 Diabetes Mellitus. Journal of Gastrointestinal & Digestive System. 2016.
4. Mitchell S, et al. Women in Control: Pioneering Diabetes Self-Management Medical Group Visits in the Virtual World. J Clin Trials. 2016;6:2167-0870.
5. Norouzi Z, et al. The Prevalence of Depression in Patients with Diabetes Mellitus Type II in the Shahid Rahimi Hospital of Khorramabad, Iran. Epidemiology (Sunnyvale). 2016;6:2161-1165.
6. Szybinski Z. Primary Prevention of Obesity and Type 2 Diabetes Mellitus. Epidemiology (Sunnyvale). 2016;6:2161-1165.
7. Nayeri A, et al. Type 2 Diabetes Mellitus is an Independent Risk Factor for Postoperative Complications in Patients Surgically Treated for Meningioma. Journal of Neurology & Neurophysiology. 2016.
8. Ignacio AB, et al. Diabetes Mellitus and Neuromuscular Blockade:Review. Journal of Diabetes & Metabolism. 2016.
9. Tong M, et al. Targeting Alzheimer's Disease Neuro-Metabolic Dysfunction with a Small Molecule Nuclear Receptor Agonist (T3D-959) Reverses Disease Pathologies. J Alzheimers Dis Parkinsonism. 2016;6:2161-0460.
10. Biessels GJ and Kappelle LJ. Increased risk of Alzheimer's disease in Type II diabetes: insulin resistance of the brain or insulin-induced amyloid pathology?. Biochemical Society Transactions. 2005;33:1041-1044.
11. Cifuentes D, et al. Targeting Hypertension to Manage Alzheimer's Disease: Rational and Promise. J Alzheimers Dis Parkinsonism. 2016;6:2161-0460.
12. Glenner GG and Wong CW. Alzheimer disease: initial report of the purification and characterization of a novel cerebrovascular amyloid protein. Biochem Biophys Res Commun. 1984;120:885–890.
13. Accardi G, et al. Can Alzheimer disease be a form of type 3 diabetes?. Rejuvenation research. 2012;15:217-221.
14. Suzanne M. Type 3 diabetes is sporadic Alzheimer′ s disease: Mini-review. European Neuropsychopharmacology. 2014;24:1954-1960.
15. Kroner Z. The relationship between Alzheimer's disease and diabetes: Type 3 diabetes?. Alternative Medicine Review. 2009;14:373.
16. Suzanne M and Wands JR. Alzheimer's disease is type 3 diabetes—evidence reviewed. Journal of diabetes science and technology. 2008;2:1101-1113.
17. Butler PC, et al. Islet amyloid polypeptide (IAPP) and insulin secretion. In Molecular Biology of Diabetes.
18. Masters CL, et al. Neuronal origin of a cerebral amyloid: neurofibrillary tangles of Alzheimer disease contain the same protein as the amyloid of plaque cores and blood vessels. EMBO J. 1985;4:2757–2763,
19. Masters CL, et al. Amyloid plaque core protein in Alzheimer disease and Down syndrome. Proc Natl Acad Sci USA. 1985;82:4245–4249.

20. Vanhanen M, et al. Association of metabolic syndrome with Alzheimer disease A population-based study. Neurology. 2006;67:843-847.
21. Draznin B, et al. Humana Press. 1994;32:381–398.
22. Schwartz P. Senile cerebral, pancreatic insular and cardiac amyloidosis. Trans N Y Acad Sci. 1965;27:393–413.