Review Article

Journal Of Clinical & Medical Images Case Reports

Open Access, Volume - 1

A review of high glycemic index (gi) in relation to risk of parkinson's disease

Mohsen Jafarzadehgharehziaaddin[1]; Mohammad Amin Parizad[2]; Marzieh Marahem[3]*; Samad Beheshtirouy[4]; Asma Jafarzadeh[5]

[1]Department of Education and Psychology, University of Aveiro, Campus Universitário de Santiago, [3810-193] Aveiro, Portugal.

[2]Assistant of Neurosurgery, Department of Neurosurgery, Tabriz University of medical sciences, Tabriz, Iran.

[3]Department of physiology and pharmacology, Tabriz University of medical sciences, Tabriz, Iran.

[4]Department of Cardiothoracic Surgery, Imam Reza Hospital, Tabriz, University of Medical Sciences, Tabriz, Iran.

[5]Department of Anesthesiology, Imam Reza Hospital, Tabriz, University of Medical Sciences, Tabriz, Iran.

Received Date : Nov 11, 2021
Accepted Date : Dec 13, 2021
Published Date : Dec 20, 2021
Archived : www.jcmimagescasereports.org

***Corresponding Author:** Marzieh Marahem, Department of physiology and pharmacology, Tabriz University of medical sciences, Tabriz, Iran.
Email: marziehmarahem@yahoo.com

Abstract

Objective: The objective was to investigate whether a diet with high glycemic index (GI) is associated with Parkinson's disease (PD) and changes that happen at levels of methylglyoxal (MGO), oligomeric α-synuclein (α-syn), and DJ-1.

Materials and Methods: This reviewed study was carried out by doing search in Scholar Google, PubMed and Elsevier databases using keywords such as Parkinson's disease, high glycemic index (GI), methylglyoxal (MGO), α-synuclein (α-syn), DJ-1. Approximately 40 articles were selected that were fully reviewed.

Conclusions: High GI diets may be risk factor for developing PD by increasing a-syn oligomerization and advanced glycated end products (AGEs) formation. Intake of dietary fiber. Appear to be beneficial in reducing PD.

Keywords: Alpha-synuclein; DJ-1; glycemic index; glycemic load; methylglyoxal; Parkinson's disease.

Introduction

Parkinson's disease (PD) is the most common neurodegenerative disorders with a worldwide prevalence in the millions [1,2]. The frequency increases with age, affecting 1% of the population over the age of 60 [3]. Genetic, family history of disease and environmental factors such as diet smoking, exposure to solvents and metals, organophosphates are linked to PD [4]. PD was first described by James Parkinson in 1817 [5,6]. Characteristic features of PD include neuronal loss in the substantia nigra [7] and widespread intracellular protein a-synuclein (a-syn) accumulation [8,9]. a-syn may contribute to PD pathogenesis in a number of ways, but it is generally thought that its aberrant soluble oligomers conformations that mediate disruption of cellular homeostasis and neuronal death [10]. Studies indicate that glucose metabolism imbalance and subsequent hyperglycaemia lead to biochemical abnormalities that may associated with PD [11-13]. Researchers about the pathogenesis of age- related noted that glucose rich diet can increase up the generation of AGEs in the brain [14,15]. The major precursor in the formation of AGEs is Methylglyoxal (MGO) [17]. Excessive levels of MGO may directly damage neurons through increasing oxidative stress [17]. DJ-1 is another protein involved in PD pathogenesis and acts as a neuroprotective agent against the toxicity of MGO and also as a major ant glycation in eliminate glycated a-syn. Function of DJ-1 is still not fully understood [18]. In some studies have shown that mutations DJ-1 caused disease with parkinsonian features [19,20]. However, the cause of PD is not fully known [21, 22] and Recently, the management of age-related diseases such as PD has been associated with dietary factors for help to reduce or contrast the symptom of PD and the related pathological mechanisms [23].

Discussion

PD is a neurodegenerative condition which there are currently no fully therapies. The incidence of PD is increasing and ex-

Citation: Mohsen Jafarzadehgharehziaaddinm, Mohammad Amin Parizad, Marzieh Marahem, Samad Beheshtirouy, Asma Jafarzadeh. A review of high glycemic index (gi) in relation to risk of parkinson's disease. J Clin Med Img Case Rep. 2021; 1(1): 1050.

pected to double worldwide by 2040 [24, 25]. Diet has recently gained importance as a potential therapeutic approach to treat PD and also as a risk factor for develop. higher dietary GI was inversely associated with prevalence of PD. The impact of carbohydrate quality only might be important rather than both quality and quantity. The number of epidemiological studies of the associations between GI and GL and PD is limited. A prospective study by Murakami et al showed that dietary GI or GL carbohydrates might be decrease the risk of PD by an insulin secretion- induced increase in brain dopamine [26]. one pervious study using ELISA reported the presence of significantly elevated levels of oligomeric forms of a-syn in plasma sample obtained from 34 PD patients compared with 27 controls [27]. Recent studies have suggested that, oligomeric a-syn-induced toxicity causes neuronal death related to PD through mitochondrial dysfunction, altered membrane permeability, and produce high levels of reactive oxygen species [28]. Several previous in vitro studies suggested that DJ-1is an antioxidant protein and free radical scavenger. It can protect against cell dead induced by oxidative stress [29-32]. Waragai et al. reported high levels of DJ-1 in the CSF and plasma of PD patient [33]. The studies conducted by Maita, et al. did not find any significant difference between the level of DJ-1 in control and patients group and also were not found in correlations of levels DJ-1 with age, level of oxidative stress and clinical severity [34]. Although the interactions between these factors have not been completely elucidated, it seems that excessive levels of MGO cause a condition such as chronic glycative stress. In this condition, the elevated level of MGO leads to the accelerated oligomerization of a-syn. Consequently, an increase in DJ-1 level could be expected a compensatory response to modulating increasing levels of MGO and oligomeric a-syn toxicity. Regarding the correlation between GI and oligomeric a-syn, in 2010, Münch, et al reported association between glycation and glycoxidation and pathogenicity of PD and also showed that there is accumulation of aggregates of an intracellular protein,a-syn and lewy bodies that trigger dopaminergic neurons death [35]. The mechanisms by which dietary GI correlated with DJ-1 may be indirect, via increase glycosylation of a-syn, inflammation, and oxidative stress associated with PD, which can stimulate elevated DJ-1 levels as a protective response [18, 36, 37]. wang, et al (2020) showed that DJ-1 can play an antiapoptotic and anti-oxidative stress role in pericytes exposed to high glucose [18]. In a study by Renaud et al, investigated relation between hyperglycemia and nigrostriatal dopaminergic neurodegeneration and they found that elevated levels of glucose lead to the death of dopaminergic neurons in culture through oxidative mechanisms [38].

Conclusion

The results of this study suggest that high GI diets may be risk factor for developing PD by increasing a-syn oligomerization and advanced glycated end products (AGEs) formation and elevated levels of DJ-1 for modulating this condition are expected. In addition, Intake of dietary fiber. appear to be beneficial in reducing PD.

The main diagnosis methods are serology and imaging. ELISA (Enzyme Linked Immunosorbent Assay) for Echinococcus IgG with sensitivity of 95% and specificity of 94%, Immunoelectrophoresis (IEP) and Haemagglutination test are some of serological studies [3]. None of these studies were positive in our case and it could be explained by isolation of the parasite from the host immune system by cyst capsule (in 50% of cases). Additionally, inadequate T-cell activation and cytokine production is another explanation [1, 4]. About Imaging modalities, it is possible to benefit from plain X-Ray, Ultrasonography (USG), CT and MRI. There is Gharbi's classification for hydatid cyst masses in USG. The main features of hydatid cysts on USG are daughter cysts, detached membrane, and double-line sign [6]. CT could discover smaller cysts which are in different organs simultaneously, differentiate the parasitic cysts from non-parasitic, detect invasion of cyst to osseous and other structures especially when the cysts calcified and utilize in treatment follow-up. Water lily sign is a characteristic feature of hydatid cyst in MRI as a detached laminated membrane which attenuation of linear area within the cyst was increased [6]. In this case, this feature was not detected and regarding high prevalence of hydatidosis in northwest of Iran and presentation of multiple cysts in various organs, Echinococcusis was the most probable diagnosis. Treatment of hydatidosis depends on various factors. Surgery is the most recommended and optimal method in cases like this [9]. In cysts with types 4 and 5 of Gharbi's classification, posterior or centrally positioned, presentation of more than three cysts, large cysts, cysts with heavy calcification, biliary and pulmonary communication of cysts and peritoneal rupture are indications of open cystectomy. In Gharbi's type 1 and 2 cysts, anterior or peripheral cysts, one or two cysts, small cysts and with or without minimal calcification, the laparoscopic approach is an optional method [5]. The surgery could be done by En bloc method which is defined as excision of cyst with whole and continuous shell of healthy tissue or by simple deroofing and enucleation of the cyst; especially, in firmly embedded cysts such as intraperitoneal cysts attached to viscera [3]. Irritation of area with hypertonic saline 20%, silver nitrate 0.5%, formalin, aqueous iodine and etc. is beneficial to prevent secondary and recurrent cysts [6]. PAIR is an alternative therapy for hydatid cysts. It is defined as Puncture-Aspiration-Injection-Reaspiration and performed as USG-guided percutaneous aspiration, injection of 95% ethanol and reaspiration [3]. Small studies for PAIR advocated that this is a safe technique without complication or recurrence. However, in larger studies it has been not recommended. These differences could be explained by dependency of technique to how experted is the performer [1, 10]. In the presented case according to high prevalence of hydatidosis in the area, hydatid cyst considered as the most probable diagnosis preoperatively and after whole intact cystectomy, the area irrigated with hypertonic saline. Anthelminthic drugs are suitable in sporadic small cysts and to prevent the recurrence after surgery [4]. In this case 6 months of Albendazole prescribed.

There have been few literatures of paraspinal hydatidosis which two of them presented as primary and solitary cyst [1, 3]. Some of them raised in cervical [1,7] and some in lumbar [2,3,4,5,8,9,10] areas. But in our case the cyst presented in

thoracic area. In conclusion, hydatid cyst should be considered as one of the differential diagnosis of all cystic lesions, especially in endemic areas which it provides the opportunity of being technically prepared for the type of intervention and the way of treatment.

References

1. Howell N, Elson JL, Chinnery PF, Turnbull DM. mtDNA mutations and common neurodegenerative disorders. Trends in Genetics. 2005; 21(11):583-6.

2. De Lau LM, Breteler MM. Epidemiology of Parkinson's disease. The Lancet Neurology. 2006;5(6):525-35.

3. Van Den Eeden SK, Tanner CM, Bernstein AL, Fross RD, Leimpeter A, Bloch DA, et al. Incidence of Parkinson's disease: variation by age, gender, and race/ethnicity. American journal of epidemiology. 2003; 157(11):1015-22.

4. Lim S-Y, Tan AH, Ahmad-Annuar A, Klein C, Tan LC, Rosales RL, et al. Parkinson's disease in the Western Pacific Region. The Lancet Neurology. 2019; 18(9):865-79.

5. Sauerbier A, Qamar MA, Rajah T, Chaudhuri KR. New concepts in the pathogenesis and presentation of Parkinson's disease. Clinical Medicine. 2016; 16(4):365.

6. Khandhar SM, Marks WJ. Epidemiology of Parkinson's disease. Disease-a-month. 2007;53(4):200-5.

7. Dickson DW, Fujishiro H, Orr C, DelleDonne A, Josephs KA, Frigerio R, et al. Neuropathology of non-motor features of Parkinson disease. Parkinsonism & related disorders. 2009; 15:S1-S5.

8. Ebrahimi-Fakhari D, Wahlster L, McLean PJ. Protein degradation pathways in Parkinson's disease: curse or blessing. Acta neuropathologica. 2012; 124(2):153-72.

9. Vivacqua G, Suppa A, Mancinelli R, Belvisi D, Fabbrini A, Costanzo M, et al. Salivary alpha-synuclein in the diagnosis of Parkinson's disease and Progressive Supranuclear Palsy. Parkinsonism & related disorders. 2019; 63:143-8.

10. Xu J, Kao S-Y, Lee FJ, Song W, Jin L-W, Yankner BA. Dopamine-dependent neurotoxicity of α-synuclein: a mechanism for selective neurodegeneration in Parkinson disease. Nature medicine. 2002;8(6):600-6.

11. Sandyk R. The relationship between diabetes mellitus and Parkinson's disease. International Journal of Neuroscience. 1993; 69(1-4):125-30.

12. Videira PA, Castro-Caldas M. Linking glycation and glycosylation with inflammation and mitochondrial dysfunction in Parkinson's disease. Frontiers in neuroscience. 2018; 12:381.

13. Schapira AH, Tolosa E. Molecular and clinical prodrome of Parkinson disease: implications for treatment. Nature Reviews Neurology. 2010; 6(6):309-17.

14. Yamagishi S-i, Ueda S, Okuda S. Food-derived advanced glycation end products (AGEs): a novel therapeutic target for various disorders. Current pharmaceutical design. 2007; 13(27):2832-6.

15. Bettiga A, Fiorio F, Di Marco F, Trevisani F, Romani A, Porrini E, et al. The modern western diet rich in advanced glycation end-products (AGEs): An overview of its impact on obesity and early progression of renal pathology. Nutrients. 2019; 11(8):1748.

16. Lv L, Shao X, Chen H, Ho C-T, Sang S. Genistein inhibits advanced glycation end product formation by trapping methylglyoxal. Chemical research in toxicology. 2011; 24(4):579-86.

17. Sharma N, Rao SP, Kalivendi SV. The deglycase activity of DJ-1 mitigates α-synuclein glycation and aggregation in dopaminergic cells: Role of oxidative stress mediated downregulation of DJ-1 in Parkinson's disease. Free Radical Biology and Medicine. 2019; 135:28-37.

18. Wang W, Zhao H, Chen B. DJ-1 protects retinal pericytes against high glucose-induced oxidative stress through the Nrf2 signaling pathway. Scientific Reports. 2020;10(1):1-13.

19. Puschmann A. Monogenic Parkinson's disease and parkinsonism: clinical phenotypes and frequencies of known mutations. Parkinsonism & related disorders. 2013; 19(4):407-15.

20. Hague S, Rogaeva E, Hernandez D, Gulick C, Singleton A, Hanson M, et al. Early-onset Parkinson's disease caused by a compound heterozygous DJ-1 mutation. Annals of neurology. 2003; 54(2):271-4.

21. Harris MA, Tsui JK, Marion SA, Shen H, Teschke K. Association of Parkinson's disease with infections and occupational exposure to possible vectors. Movement disorders. 2012; 27(9):1111-7.

22. Fahn S, Cohen G. The oxidant stress hypothesis in Parkinson's disease: evidence supporting it. Annals of neurology. 1992; 32(6):804-12.

23. Ciulla M, Marinelli L, Cacciatore I, Stefano AD. Role of dietary supplements in the management of Parkinson's disease. Biomolecules. 2019; 9(7):271.

24. Dorsey ER, Bloem BR. The Parkinson pandemic—a call to action. JAMA neurology. 2018; 75(1):9-10.

25. Feigin VL, Nichols E, Alam T, Bannick MS, Beghi E, Blake N, et al. Global, regional, and national burden of neurological disorders, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. The Lancet Neurology. 2019; 18(5):459-80.

26. Murakami K, Miyake Y, Sasaki S, Tanaka K, Fukushima W, Kiyohara C, et al. Dietary glycemic index is inversely associated with the risk of Parkinson's disease: a case–control study in Japan. Nutrition. 2010; 26(5):515-21.

27. El-Agnaf OM, Salem SA, Paleologou KE, Curran MD, Gibson MJ, Court JA, et al. Detection of oligomeric forms of α-synuclein protein in human plasma as a potential biomarker for Parkinson's disease. The FASEB journal. 2006; 20(3):419-25.

28. Salahuddin P, Rabbani G, Khan RH. The role of advanced glycation end products in various types of neurodegenerative disease: a therapeutic approach. Cellular & molecular biology letters. 2014;19(3):407-37.

29. Taira T, Saito Y, Niki T, Iguchi-Ariga SM, Takahashi K, Ariga H. DJ-1 has a role in antioxidative stress to prevent cell death. EMBO reports. 2004; 5(2):213-8.

30. Canet-Avilés RM, Wilson MA, Miller DW, Ahmad R, McLendon C, Bandyopadhyay S, et al. The Parkinson's disease protein DJ-1 is neuroprotective due to cysteine-sulfinic acid-driven mitochondrial localization. Proceedings of the National Academy of Sciences. 2004; 101(24):9103-8.

31. Yokota T, Sugawara K, Ito K, Takahashi R, Ariga H, Mizusawa H. Down regulation of DJ-1 enhances cell death by oxidative stress, ER stress, and proteasome inhibition. Biochemical and biophysical research communications. 2003; 312(4):1342-8.

32. Kinumi T, Kimata J, Taira T, Ariga H, Niki E. Cysteine-106 of DJ-1 is the most sensitive cysteine residue to hydrogen peroxide-mediated oxidation in vivo in human umbilical vein endothelial cells. Biochemical and biophysical research communications. 2004; 317(3):722-8.

33. Waragai M, Wei J, Fujita M, Nakai M, Ho GJ, Masliah E, et al. Increased level of DJ-1 in the cerebrospinal fluids of sporadic Parkinson's disease. Biochemical and biophysical research communications. 2006; 345(3):967-72.

34. Maita C, Tsuji S, Yabe I, Hamada S, Ogata A, Maita H, et al. Secretion of DJ-1 into the serum of patients with Parkinson's disease. Neuroscience letters. 2008; 431(1):86-9.

35. Münch G, Westcott B, Menini T, Gugliucci A. Advanced glycation endproducts and their pathogenic roles in neurological disorders. Amino acids. 2012; 42(4):1221-36.

36. Anderson C, Milne GL, Park Y-MM, Sandler DP, Nichols HB. Dietary glycemic index and glycemic load are positively associated with oxidative stress among premenopausal women. The Journal of nutrition. 2018; 148(1):125-30.

37. Arikawa AY, Jakits HE, Flood A, Thomas W, Gross M, Schmitz KH, et al. Consumption of a high glycemic load but not a high glycemic index diet is marginally associated with oxidative stress in young women. Nutrition Research. 2015; 35(1):7-13.

38. Renaud J, Bassareo V, Beaulieu J, Pinna A, Schlich M, Lavoie C, et al. Dopaminergic neurodegeneration in a rat model of long-term hyperglycemia: preferential degeneration of the nigrostriatal motor pathway. Neurobiology of aging. 2018; 69:117-28.