

Methylglyoxal as a Marker of NAFLD Severity in Patients with Type 2 Diabetes

LA Zvenigorodskaya^{*}, MV Shinkin, AV Petrakov, TV Nilova

Department of Clinical Medicine, University of Medical Clinical Research Center, Loginov, Moscow, Russia

ABSTRACT

Glycation is due to the ability of glucose to form irreversible chemical compounds with amino groups of lysine and arginine - methylglyoxal (MG).

Methylglyoxal (MGO) is the natural compound with the recipe CH3C(O)CHO. It is a diminished subordinate of pyruvic corrosive. It is a responsive intensify that is embroiled in the science of diabetes. Methylglyoxal is delivered modernly by corruption of starches utilizing overexpressed methylglyoxal synthase.

Vaporous methylglyoxal has two carbonyl gatherings, an aldehyde and a ketone. Within the sight of water, it exists as hydrates and oligomers. The arrangement of these hydrates is demonstrative of the high reactivity of MGO, which is applicable to its organic conduct.

Keywords: NAFLD; Type 2 diabetes; Glycosylation; Methylglyoxal

INTRODUCTION

In creatures, methylglyoxal is framed as a side-result of a few metabolic pathways.Methylglyoxal for the most part emerges as side results of glycolysis including glyceraldehyde-3-phosphate and dihydroxyacetone phosphate. It is likewise thought to emerge by means of the debasement of CH3)2CO and threonine. Illustrative of the heap pathways to MGO, 12-overlay increment of aristolochic corrosive caused methylglyoxal from 18 to 231 µg/mg of kidney protein in harmed mice. It might frame from 3-aminoacetone, which is a middle of the road of threonine catabolism, just as through lipid peroxidation. Be that as it may, the most significant source is glycolysis. Here, methylglyoxal emerges from nonenzymatic from glyceraldehyde phosphate phosphate end and dihydroxyacetone phosphate (DHAP), two intermediates of glycolysis. This transformation is the premise of a potential biotechnological course to the product synthetic 1,2-propanediol [1].

Non-alcoholic Greasy Liver Illness (NAFLD), otherwise called Metabolic (brokenness) related Greasy Liver Infection (MAFLD), unreasonable fat develop in the liver without another reasonable reason, for example, liquor use. There are two sorts; nonalcoholic greasy liver (NAFL) and non-alcoholic steatohepatitis (NASH), with the last likewise including liver inflammation. Non-alcoholic greasy liver sickness is less perilous than NASH and generally doesn't advance to NASH or liver cirrhosis. When NAFLD advances to NASH, it might in the long run lead to confusions, for example, cirrhosis, liver malignancy, liver disappointment, or cardiovascular disease [2].

Corpulence and type 2 diabetes are solid hazard factors for NAFLD. Other dangers incorporate being overweight, metabolic disorder (characterized as in any event three of the five after ailments: stomach stoutness, hypertension, high glucose, high serum triglycerides, and low serum HDL cholesterol), an eating regimen high in fructose, and more established age.NAFLD and alcoholic liver infection are sorts of greasy liver disease. Obtaining an example of the liver subsequent to barring other possible reasons for greasy liver can affirm the diagnosis [3].

Treatment for NAFLD is weight reduction by dietary changes and exercise. There is conditional proof for pioglitazone and nutrient E; bariatric medical procedure can improve or resolve extreme cases. Those with NASH have a 2.6% expanded danger of passing on per year [4].

NAFLD is the most widely recognized liver issue worldwide and is available in roughly 25% of the world's population. It is additionally exceptionally regular in created countries, for example, the United States, and influenced around 75 to 100 million Americans in 2017. Over 90% of large, 60% of diabetic,

Correspondence to: Zvenigorodskaya LA, Department of Clinical Medicine, University of Medical Clinical Research Center ,Loginov, Moscow, Russia, Tel: 89700000000; E-mail: jendis1@gmail.com

Received date: April 05, 2021; Accepted date: April 20, 2021; Published date: April 26, 2021

Copyright: © 2021 Zvenigorodskaya LA, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Citation: Zvenigorodskaya LA, Shinkin MV, Petrakov AV, Nilova TV (2021) Methylglyoxal as a Marker of NAFLD Severity in Patients with Type 2 Diabetes. Diabetes Case Rep. 6:140.

and up to 20% ordinary weight individuals create it. NAFLD is the main source of ceaseless liver disease and the second most normal purpose behind liver transplantation in the US and Europe as of 2017. NAFLD influences around 20 to 25% of individuals in Europe. In the United States, gauges propose somewhere in the range of 30 and 40% of grown-ups have NAFLD, and around 3 to 12% of grown-ups have NASH. The yearly monetary weight was roughly US\$103 billion in the US in 2016 [5].

Type 2 Diabetes (T2D) once in the past known as grown-up beginning diabetes, is a type of diabetes that is portrayed by high glucose, insulin opposition, and relative absence of insulin. Regular indications incorporate expanded thirst, visit pee, and unexplained weight reduction. Side effects may likewise incorporate expanded yearning, feeling tired, and wounds that don't recuperate. Frequently a side effects please gradually. Long haul inconveniences from high glucose incorporate coronary illness, strokes, diabetic retinopathy which can bring about visual impairment, kidney disappointment, and poor blood stream in the appendages which may prompt removals. The abrupt beginning of hyperosmolar hyperglycemic state may happen; in any case, ketoacidosis is phenomenal [6].

Type 2 diabetes basically happens because of heftiness and absence of activity. A few people are more hereditarily in danger than others. Type 2 diabetes makes up about 90% of instances of diabetes, with the other 10% due fundamentally to type 1 diabetes and gestational diabetes. In type 1 diabetes there is a lower all out degree of insulin to control blood glucose, because of an immune system instigated loss of insulin-creating beta cells in the pancreas. Analysis of diabetes is by blood tests, for example, fasting plasma glucose, oral glucose resilience test, or glycated hemoglobin (A1C) [7].

Type 2 diabetes is to a great extent preventable by remaining an ordinary weight, practicing routinely, and eating appropriately. Treatment includes exercise and dietary changes. On the off chance that glucose levels are not enough brought down, the medicine metformin is commonly suggested. Numerous individuals may in the end additionally require insulin infusions. In those on insulin, routinely checking glucose levels is exhorted; be that as it may, this may not be required in those taking pills. Bariatric medical procedure regularly improves diabetes in the individuals who are fat [8-10].

MATERIAL AND METHODS

98 patients with NAFLD and 52 patients with NAFLD were examined. Diabetes 2-type, and 22 healthy volunteers. Age 57.3 \pm 5.2years. BMI $34,85 \pm 1,79$. The diagnosis was established on the basis of laboratory data and examination results. The level of LPO was determined by the content of malonic dialdehyde (MDA).

RESULTS

In patients with NAFLD, the content of MG in the blood serum 520.75 \pm 114.35, in the control-69.02 \pm 6.67 nm/l (p=0.001). In T2DM patients the MG content was 292.11 \pm 16.34 nm/l. Observed correlation of MG and MDA (r=0,495).

CONCLUSIONS

Patients with NAFLD have a significant increase in MG b serum 7 times compared to the control. MG damage arginine residues of proteins, disrupts the transmission of the insulin signal, inhibits enzymes and plays a key role in the development of insulin resistance and hyperglycemia. Quantitative determination of MG in serum by HPLC method can be used as a prognostic and diagnostic test glucotoxicity.

REFERENCES

- Angulo P. Non-alcoholic fatty liver disease. N Engl J Med. 2002; 346:1221-1231.
- Manne V, Handa P, Kowdley KV. Pathophysiology of Nonalcoholic Fatty Liver Disease/Nonalcoholic Steatohepatitis. Clin Liver Dis. 2018;22:23–37.
- Ludwig J, Viggiano TR, McGill DB, Oh BJ. Non-alcoholic steatohepatitis: Mayo clinic experiences with a hitherto unnamed disease. Mayo Clin Proc. 1980;55:434–438.
- Kim WR, Lake JR, Smith JM, Skeans MA, Schladt DP, et al. OPTN/ SRTR 2015 Annual Data Report: Liver Am J Transplant. 2017;17:174–251.
- Day CP, James O. Steatohepatitis: A tale of two 'hits'? Gastroenterol. 1998;114:842–845.
- Buzzetti E, Pinzani M, Tsochatzis EA. The multiple-hit pathogenesis of nonalcoholic fatty liver disease (NAFLD) Metabolism. 2016;65:1038– 1048.
- Dongiovanni P, Anstee QM, Valenti L. Genetic Predisposition in NAFLD and NASH: Impact on Severity of Liver Disease and Response to Treatment. Curr Pharm Des. 2013;19:5219–5238.
- Sies H, Berndt C, Jones DC. Oxidative stress. Annu Rev Biochem. 2017;86:715–748.
- 9. Sies H. On the history of oxidative stress: Concept and some aspects of current development. Curr Opin Toxicol. 2018;7:122–126.
- 10. Lushchak VI. Free radicals, reactive oxygen species, oxidative stress and its classification. Chem Biol Interact. 2014;224C:164-175.