Journal of Diabetes & Clinical Practice

Wang, J Diabetes Clin Prac 2022, 5:6

Perspective Open Access

Lipid Metabolic Disorders and Liver Steatosis in DOCA-Salt Hypertensive Mice

Yan Wang*

Department of Internal Medicine, University of Utah and Salt Lake Veterans Affairs Medical Center, US

Abstract

Endogenous ligands for peroxisome proliferator-activated receptors include nitrooleic acid. The goal of the current investigation was to examine the therapeutic effects of OA-NO2 on lipid metabolism and hepatic steatosis in a mouse model of hypertension caused by deoxycorticosterone acetate salt. Male C57BL/6 mice were separated into two groups: one group got neither DOCA-salt nor OA-NO2, and the other received neither DOCA-salt nor OA-NO2 (control group). The hypertension was detected after a 3-week course of therapy with DOCA-salt + 1% sodium chloride in drinking water; however, OA-NO2 had no impact on the hypertension. Plasma triglyceride and total cholesterol levels in DOCA-salt-treated animals were considerably higher than those in control mice, but these parameters were markedly decreased after pretreatment with OA-NO2. Additionally, the histology of the liver showed increased lipid distribution along with more severe micro- and following DOCA-salt therapy, there was macro vesicular steatosis, which was consistent with the level of triglycerides and non-esterified fatty acids in the liver tissue.

Keywords: Endogenous Ligands; Hypertension; Doca-Salt Delivery; Dyslipidemia

Introduction

The mice that had received an OA-NO2 pretreatment had less liver damage and less hepatic lipid accumulation. Additionally, the expressions of gp91phox and p47phox dropped concurrently with the liver TBARS [1]. These results demonstrated that the antihyperlipidemic properties of OA-NO2 had a protective impact on liver damage caused by DOCA-salt delivery [2]. The most prevalent cardiovascular illness is hypertension, and it is expected that prevalence will rise across the board, particularly in emerging nations. Researchers are interested in the metabolic anomalies, particularly faulty lipid metabolism, in hypertension patients because of their close relationship [3]. The significant link between Dyslipidemia and hypertension is becoming more and clearer. Many hypertension patients have metabolic problems such as hypertriglyceridemia, hypercholesterolemia, and insulin resistance [4]. The greatest rates of morbidity and death among patients with cardiovascular disease are caused by hypertension and Dyslipidemia, which are significant risk factors for the condition. As a result, several investigations have been devoted to creating therapeutic medicines for hypertension and the lipid metabolism that it is associated with [5]. There are several traditional animal models for the study of hypertension [6]. Animal model of hypertension caused by deoxycorticosterone acetate salt model has been frequently used to research hypertension and the organ damage caused by hypertension. In the past, DOCAsalt hypertensive rats were shown to have rising levels of both circulation and membrane lipids before the onset of hypertension [7].

Discussion

In the meanwhile, DOCA-salt hypertensive mice showed altered levels of membrane phospholipids, phospholipid distribution, and fatty acid saturation [8]. The modifications in the lipid content of the membrane may modify membrane activity over time, affecting the control of blood pressure as well as the function of the heart, liver, and kidney organs [9]. The nitro alkene and nitrooleic acid derivatives of nitrated free fatty acids, in particular, are endogenous compounds with enticing signalling qualities. There are nitro alkenes. Animals we bought male C57BL/6 mice from Shandong University's Animal Center. Standard rat chow was used to feed all animals, and a 12-hour light/dark cycle was maintained [10]. All mice also had full access to water. All mouse-using methods were carried out in compliance with the rules and recommendations of the Provincial Hospital Affiliated to Shandong University's Ethics Committee. Materials two regioisomers of OA-NO2, 9- and 10-nitrooleic acids, are created in vivo by nitrating oleic acid in almost equal amounts. Both substances were bought from Cayman Chemical and utilised as a combination of their isomers after being dissolved in dimethyl sulfoxide. Animals used as sham operators served as controls. Following surgery, animals were given a normal salt diet for three weeks and drinking water containing 1% sodium chloride. Once a week, metabolic cages were used to measure body weight, urine volume, food intake, and water intake.

Conclusion

After the tests were complete, the animals were fasted for an entire night before blood was drawn by cutting a tiny cut in the tail with a razor blade. The mechanism of the hyperlipidaemia in DOCA-salt hypertensive animals was distinct from that of the obese Zucker rats. Hernandez discovered that, in contrast to control rats, DOCA-salt hypertensive rats had higher plasma levels of triglycerides and cholesterol as well as higher glucose, glycogen, and triglyceride levels and decreased citrate synthase and beta-hydroxyacyl-CoA dehydrogenase activities in the soleus muscle. These findings suggested that the hyperlipidaemia of the DOCA-salt hypertensive animals was caused by changes in metabolic enzymes.

***Corresponding author:** Yan wang, Department of Internal Medicine, University of Utah and Salt Lake Veterans Affairs Medical Center, US, E-mail: Yanwang90@gmail.com

Received: 01-Nov-2022, Manuscript No. jdce-22-82500; **Editor assigned:** 05-Nov-2022, PreQC No. jdce-22- 82500 (PQ); **Reviewed:** 12-Nov-2022, QC No. jdce-22-82500; **Revised:** 19-Nov-2022, Manuscript No. jdce-22-82500(R); **Published:** 30-Nov-2022, DOI: 10.4172/jdce.1000169

Citation: Wang Y (2022) Lipid Metabolic Disorders and Liver Steatosis in DOCA-Salt Hypertensive Mice. J Diabetes Clin Prac 5: 169.

Acknowledgement

None

Conflict of Interest

None

References

1. Patrick R, Adrian F H, Scott DS, Faiez Z (2019) Heart failure drug treatment. Lancet 393: 1034-1044.
2. Barry A B (2020) Evaluation and management of heart failure with preserved ejection fraction. Nat Rev Cardiol 17: 559-573.
3. John JVM, Scott DS, Silvio EI, Lars K, Mikhail NK, et al. (2019) Dapagliflozin in patients with heart failure and reduced ejection fraction. N Engl J Med 381: 1995-2008.
4. Milton P, Stefan DA, Javed B, Gerasimos F, Stuart JP, et al. (2020) Cardiovascular and renal outcomes with empagliflozin in heart failure. N Engl J Med 383: 1413-1424.
5. William TA, JoAnn L, Piotr P, Piergiuseppe A, Javed B, et al. (2021) Empagliflozin in heart failure with a preserved ejection fraction. N Engl J Med 385: 1451-1461.
6. Johann B (2021) Heart failure drug treatment: the fantastic four. Eur Heart J 42: 681-683.
7. Mahakpreet S, Anoop K (2018) Risks associated with SGLT2 inhibitors: an overview. Curr Drug Saf 13: 84-91.
8. Arsalan H, Muthiah V, Adebamike AO, Krishna KA, Andreas PK, et al. (2020) Antihyperglycemic therapies with expansions of US Food and Drug Administration indications to reduce cardiovascular events: Prescribing patterns within an academic medical center. J Cardiovasc Pharmacol 76: 313-320.
9. Rosario R, Stefani DD, Raffaello A, Cristina M (2012) Mitochondria as sensors and regulators of calcium signaling. Nat Rev Mol Cell Biol 13: 566-578.
10. Ajay NJ (2007) Surflex-Dock 2.1: Robust performance from ligand energetic modeling, ring flexibility, and knowledge-based search. J Comput-Aided Mol Des 21: 281-306.