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Lycopene Action On Cyclooxygenase and Lipid Peroxidation in Dexamethasone-Actuated Oxidative Worry in Wistar Rodents

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Abstract— This examination was gone for exploring lycopene action on cyclooxygenase and lipid peroxidation in dexamethasone-treated Wistar rodents. Twenty (20) male Wistar rodents weighing between 150g-250g were haphazardly chosen into four gatherings containing five rodents each. Control rodents got standard feed and water. Gathering two got 3mg/kg body weight of dexamethasone intraperitoneally every two days for 9 days. Gathering 3 got 3mg/kg body weight of dexamethasone intraperitoneally every two days for 9 days in addition to day by day oral organization of 1mg/kg of lycopene for 28 days. Results demonstrated that there was no critical distinction in the action of dexamethasone and lycopene on COX and THX-A 2 in every one of the gatherings. Dexamethasone expanded AST ALT and ALT level. Treatment with Lycopene fundamentally ($p<0.01$) diminished AST, ALT, and ALP in every one of the gatherings. Lactate dehydrogenase movement was fundamentally ($p<0.01$) diminished in the dexamethasone and further brought down upon treatment with lycopene when contrasted with the DEX gathering. Malondialdehyde (MDA) fixation in Dex was expanded ($p<0.01$), Catalase action was diminished while SOD focus was not modified. Treatment with lycopene Significantly ($p<0.01$) diminished serum MDA and expanded catalase fixation. Triglyceride and LDL segments of the lipid were raised in Dex with a diminished HDL however without modification in complete cholesterol level. Lycopene diminished the TC, LDL, and TG and fundamentally ($p<0.01$) expanded HDL. It is presumed that dexamethasone stifles cyclooxygenase articulation however potentiates lipid peroxidation and builds liver compounds. Lycopene hinders Cox movement, secure against lipid peroxidation and is hypolipidemic and hepatoprotective.

Keywords— Dexamethasone, Lycopene, Cyclooxygenase, Lipid peroxidation, Liver enzyme.

1. Introduction

Dexamethasoneis an engineered glucocorticoid regularly utilized for fiery issue, for example, asthma, hypersensitivity, contamination and alsoautoimmune malady, for example, rheumatoidarthritis, glomerulonephritis, sclerosis [1]. The counter inflammatoryproperty of glucocorticoids depends on its capacity to stifle the action of qualities that have a noteworthy job in irritation, for example, cytokines and nitricoxide synthase [2]. Cytokines especially, straightforwardly animates the arrangement of reactiveoxygenspecies [3,4] that are potentoxidative pressure markers. A few specialists nonetheless, have opined that one system by which dexamethasone does its calming action is by restraint of cyclooxygenase (COX) which is thought to be inconvenient to the body as a result of its association in the arrangement of prostaglandin E 2 and thromboxane however whose hindrance promptly gives alleviation to torment and side effects of irritation [5,6]. Past the mitigating property of dexamethasone, inquire about has additionally embroiled it to wear an inconvenient face. For example, glucocorticoid treatment, contingent upon the measurement, prompts genuine fundamental symptoms, for example, immunosuppression, hypertension, adrenal organ misery and steroid diabetes [7], upsets lipid digestion in this manner potentiating lipid

peroxidation and arrangement of receptive oxygen species [8].

Lycopene, a characteristic plant item with an abnormal state of the cancer prevention agent property has been accounted for to display free radical and singlet oxygen species searching property brought about by lipid peroxide [9]. The point of the present examination was to explore the MDA level, liver catalyst focus and cyclooxygenase/thromboxane A2 action in dexamethasone-treated rodents and the cancer prevention agent status of lycopene supplementation.

Experimental Design

Fifteen male Wistar rodents weighing somewhere in the range of 180 and 250 g were gotten from the creature House, Physiology Department, Cross River University of Technology, Calabar, Nigeria and arbitrarily chose into three gatherings of 5 rodents each. The creatures were housed in plastic confines and kept in room temperature of 28°C ± 2°C with 12 h light/dull cycle. Gathering 1 (control) was benefited from an ordinary rodent feed. Gathering 2 (Dex) got 3mg/kg body weight of dexamethasone intraperitoneally every two days while bunch 3 (Dex + Lyco) got 3mg/kg body weight of dexamethasone every 2 days in addition to day by day oral organization of 1mg/kg of lycopene for 28 days. All gatherings approached water and standard feed not obligatory. Moral endorsement for the examination was gotten from the Faculty of Basic Medical Science Animal Research Ethical Committee of Cross River University of Technology, Calabar, Calabar, Nigeria (endorsement number FBMS/CRUTECH/12/015).

Gathering of blood tests and biochemical investigation

Creatures were anesthesized, blood tests were gathered via heart cut into EDTA blood test bottles for assurance of COX, THX-A 2 and liver compounds and furthermore into plain jugs for lipid profile examination. Tests were permitted to represent two hours to clump. The blood was centrifuged at 2000rpm for 10 minutes to get the serum. The serum was put away at 10°C until further use. The strategies portrayed by [10,11] were utilized to decide all out cholesterol (TC) and triglyceride, individually. low thickness lipoprotein cholesterol was determined to utilize the condition of [12]

Assurance of superoxide dismutase/catalase movement and lipid peroxidation

Superoxide dismutase movement was controlled by the technique for [13] catalase was resolved utilizing the strategy for [14] Lipid peroxidation was dictated by the technique for [15]

Factual investigation

Information acquired were exhibited as the mean ± standard mistake of the mean (SEM). The measurable examination was finished utilizing the One-route investigation of change (ANOVA). The GraphPad Prism adaptation 5.0 for Windows (GraphPad Software, San Diego, CA, USA) was utilized for the examination. Bonferroni various correlation tests were additionally utilized for pair-wise examination, and contrasts were viewed as critical at $p < 0.01$.

2. RESULTS

2.1 Effect of dexamethasone and lycopene on lipid profile

The impact of dex and lycopene on complete cholesterol, triglyceride, HDL, and LDL is appeared table 1. All out cholesterol was not influenced by dex. Triglyceride and LDL were altogether ($p<0.01$) raised while

HDL was decreased. Treatment with lycopene altogether ($p<0.01$) diminished TC, TG and LDL. HDL was altogether ($p<0.01$) expanded.

Impact of dexamethasone and lycopene on liver chemicals

The aftereffect of dexamethasone treatment and lycopene organization on AST, ALT and ALP is appeared in figures 3, 4 and 5. All gatherings treated with dexamethasone altogether expanded serum AST, ALT, and ALP when contrasted and the control. Treatment with Lycopene altogether ($p<0.01$) diminished AST, ALT, and ALP in every one of the gatherings.

Impact of dexamethasone and lycopene on lipid peroxidation and lactate dehydrogenase

Lipid peroxidation item, MDA in the dexamethasone gathering was fundamentally ($p<0.01$) higher when contrasted with control and the test gathering. Treatment with lycopene Significantly ($p<0.01$) diminished serum MDA, (Figure 6). There was no noteworthy contrast in superoxide dismutase (SOD) focus in every one of the gatherings (figure 7) while the movement of catalase in DEX was fundamentally ($p<0.01$) decreased. Treatment with lycopene fundamentally ($p<0.01$) expanded catalase fixation when contrasted with the dexamethasone bunch as appeared in figure 8. Lactate dehydrogenase movement was significantly($p<0.01$) diminished in the dexamethasone bunch when contrasted with control and further brought down upon treatment with lycopene when contrasted with the DEX group (figure 9).

Plate 2: Photomicrograph of the liver of (A) control with typical Hepatic sinusioids. (B) Dexamethasone gathering (DM) demonstrating gross separation and consumption of hepatocytes, (C) Dex. gathering treated with Lycopene (DEX. + LYCO) demonstrated no armies. H and E. X100

3. Discussion

The utilization of Dexamethasone in the treatment of different incendiary conditions just as in relief from discomfort can't be overemphasized. This characteristic of glucocorticoid is because of dependent on its cell-explicitness relying upon the outflow of different receptor proteins and protein synthesis [16]. In this examination, we analyzed the movement of dexamethasone and impact of lycopene supplementation on cyclooxygenase/thromboxane A2 levels, serum liver catalyst fixation, and lipid peroxidation in Wistar rodents. Our outcomes on the cyclooxygenase and thromboxane action in both dexamethasone and the lycopene-enhanced gatherings did not demonstrate any measurable distinction when contrasted with control. The announced movement of dexamethasone as a mitigating operator on one hand, and as an oxidative pressure inducer then again, in this manner, raises some worry.

Some researchers, [17,18], have conjectured that dexamethasone actuates oxidative pressure and overproduction of responsive oxygen species and adds to the advancement of cardiovascular issues by means of upregulation of ACE articulation and angiotensin II type - 1 and α-1 receptors [19]. Expanded generation of this free radicals without hindrance by either endogenous or exogenous cancer prevention agents encroaches on the equalization of the invulnerable framework causing a separate in physiological movement and in the long run bringing about pernicious ambushes on indispensable organs [20].

The mind-boggling increment in lipid peroxidation and their items, triglyceride, and LDL and furthermore the decrease in catalase protein action, and HDL in dexamethasone-treated creatures saw in this investigation recommends a conceivable mutilation in the detoxification framework. Studies have demonstrated that lipid peroxides and their items can make huge damage film bound compounds and

biomolecules, for example, mRNA, and DNA [21].

The superoxide dismutase movement in this examination was not modified. Normally, endogenous cancer prevention agents, for example, superoxide dismutase, catalase, and glutathione, search and subdues the arrangement of ROS [22]. Catalase capacities to processes the hydrogen peroxide delivered at the course of peroxidation into water and oxygen while SOD rummages superoxide radicals and elevates quickly its transformation to hydrogen peroxide which is then detoxified by glutathione peroxidase [23,24]. The capacity of lycopene to ensure against free radical-instigated harm and lipid peroxidation procedure prompted by dexamethasone is prove by the noteworthy diminishing in the dimensions of peroxidation item, malondialdehyde (MDA) and basically, good tweak of lipid profile saw in this examination. The outcome acquired is in accordance with a previous detailed work by [25,26,27,28,29.] who exhibited the cancer prevention agent impact of lycopene on peroxidation of phospholipids, proteins, and nucleic acids. The complete cholesterol (TC), Triglyceride (TG) and low-thickness lipoprotein (LDL) were brought down while HDL fixation was expanded after lycopene supplementation. Flavonoids and saponins in plant items have been involved in the bringing down of lipids.

One exceptional component of decrease of the lipids is proposed to be by hindrance of hepatic HMG-CoA reductase [30] and furthermore the decrease of the awful cholesterol (LDL) by expanded hepatic detoxification or decontamination of LDL precursors. [31,32,33]. Similarly, our examination has demonstrated that dexamethasone treatment and lycopene supplementation did not change cyclooxygenase and thromboxane movement. This might be deciphered to mean restraint or a no action result. This is clear by different exploratory reports that the mitigating movement exhibited by dexamethasone in vivo, is by means of the concealment of basal constitutive cyclooxygenase union [34,35,36] and COX-mRNA [37]. Cyclooxygenase is the protein in charge of the development of prostanoids (thromboxane and prostaglandins). It catalyzes the change of arachidonic corrosive to frame prostaglandin E2 and thromboxane [5] whose organic activities incorporate vasoconstriction and is pathogenic in a different sickness like hepatic provocative process [38]and intense hepatotoxicity [39]

Strangely, accessible writing recommends that lycopene as a result of its phytochemical segments, for example, carotenoids, flavonoids, saponins and tannis[40] and its solid movement in rummaging singlet oxygen species [41]suppresses Cox-2,[42] prostaglandin E 2, ERK 1/2 phosphorylation [43], hence proposing a calming action.

Besides, the way that AST, ALT, and ALP were significantly raised in dexamethasone treatment in this examination presumes a conceivable damage to the liver. This is seen in the liver histology with gross seclusion and consumption of hepatocytes.

This might be viewed as one symptom of successful utilization of the manufactured glucocorticoid. All things considered, lycopene indicated evidently a positive natural action by switching the bargained liver respectability and by bringing down the AST, ALT and ALP catalysts. Aspartate aminotransaminase (AST) is to a great extent found in the muscles and liver parenchymal cells. At the point when there is a raised fixation in AST and serum alanine aminotransferase (ALT) it perpetually proposes conceivable liver ailment or harm.

Then again, ALP is regularly used to build up plasma film respectability with the end goal that any sensible change in its fixation may recommend harm to the plasma layer. In any case, there are likewise gives an

account of the opposite that Lycopene does not adjust AST, ALT and ALP. [44].

4. Conclusion

We infer that dexamethasone thromboxane A 2 action, initiates lipid to cause wrecking liver harm hepatoprotective, hypolipidemic and thromboxane A2 action. restrains COX and peroxidation and may while lycopene is hindered COX-2 and thromboxane A2 action.

5. References

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