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Exploring Moringa Oleifera Usefulness In Ameliorating Induced Retinopathy In Experimental Animals

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Abstract: *Retinopathy is a major problem of vision. It could lead to loss of vision. It is simply the degeneration of the retina. This can be as a result of assault or trauma to the eye or its retina. It can also be as a result of complications of health including diabetes. A totally damaged retina is an indication of total loss of visual perception, which also implies total loss of vision. It is however helpful to prevent the retina from degenerating. This experiment is an attempt to explore the potentials of the phytochemicals of Moringa oleifera [moringa] in its ethanolic extract to ameliorate retina degeneration caused by sodium iodate [NaIO3] toxicity. Twenty four adult male animals weighting between 180 and 200g were divided into Groups A-D. Group A animals served as the Control group; they were only fed ad libitum. Group B animals were administered 50mg/kg body weight of sodium iodate [NaIO3] through the tail vein to induce retinpathy. Group C animals were administered sodium iodate [NaIO3, 50mg/kg BW] once to induce retinopathy and thereafter administered a daily dose of a low dose of the ethanoic extract of moringa [200mg/kg BW] daily with an oral cannula.Group D animals were admisnitred sodium iodate once [NaIO3, 50mg/kg BW] to induce retinopathy and therefater a high dose of the ethanoic extract of moringa [500mg/kg BW] daily, with an oral cannula. Moringa administration lasted ten days after animals were injected [NaIO3]. Results incidate that the $NaIO_3$-induced retinoathy was only fairly ameliorated by moringa extract.*

Key Words: *Retina, Retinopathy, Moringa, Sodium Iodate, Eye*

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I. Introduction

Retinopathy is a term used to basically describe damage and degeneration to the eye retina which may lead to vision impairment or loss due to compromised photoreception. Retinopathy often refers to retinal vascular disease, macular degeneration, or damage to the retina caused by abnormal blood flow. Frequently, retinopathy is an ocular manifestation of systemic disease as seen in diabetes or hypertension. Retinopathy-including macular, degeneration is a degerative disease of the retina and the leading cause of irreversible blindness in developed countries. It is a clinically and genetically highly heterogenous disorder affecting the people world wide. Retinal degeneration is characterised by the progressive degeneration of photoreceptor cells in the retina. It first influences rods and later results in the loss of cones. Eventually, cone degeneration affects the central vision, leading to complete blindness.

Macular degeneration is a leading cause of irreversible blindness in the developed world. The retinal pigment epithelium (RPE) is a critical site of pathology (Zhou, *et al*., 2014). Retinotoxicity can be due to natural factors, induced, and due to complications such as trauma, hypertension and diabetes. Diabetic retinopathy accounts for about 5% of blindness worldwide and is designated a priority eye disease by the World Health Organization (WHO, 2017). Retinotoxicity can also be induced by endogenous and exogenous agents in laboratory animals. Sodium Iodate (NaIO3) has proven to be an effective way to induce retinopathy in animal models.

Retinipathy is a major vision disorder. Risk factors include cardiac defects, high exposure to oxygen, infection, low birth weight and prematurity (Karma *et al*., 2009). Epidemiology reports show that retinopathy of prematurity prevalence varies, from 5–8% in developed countries with adequate neonatological facilities, to up to 30% in middle-income developing countries (Gergely and Gerinel, 2010). There is also evidence that the population of premature infants at risk of severe ROP varies depending on the level of neonatal intensive care being provided (Gilbert *et al.,* 2005). The sodium iodate retinopathy model is also an evidence that it could be linked to toxicities.

Sodium Iodate is a white crystalline salt (Budavari, 1996). It is an organic salt, iodic acid, and sodium salt (Registry of Toxic Effects of Chemical Substances [RTECS], 1992). Salts acts as a carrier of iodine, which helps to populate areas that are deficient of iodine. Sodium Iodate is achieved by adding 20 to 80 mg of iodine per kilogram of sodium salt. $INaO_3$ is a powerful oxidizer which is a fire hazard, when it comes in contact with

flammable or combustible materials, it triggers fire (Sax, 1975). $INaO_3$ is very toxic. The oral lethal dose for human is 50 – 500mg/kg (Gosselin *et al.,* 1976). A reported case of a 22year old man, who consumed a concentrated solution of potassium Iodate indicated that he developed nausea, diarrhea and had immediate loss of visual sharpness. Opthalmoscopic examination revealed extensive retinal damage accompanied with photoreceptor layer degeneration (Singalavanija *et al.,* 1994: Singalavanija *et al.,* 2000: Tong, 1995). Also, in the 1920s, there was an incidence involving Iodate- induced blindness in China; in which there was pigment epithelium damage as a result of the toxicity of Pregl's solution (Lewis`, 1996).

There are experimental evidences of sodium iodate is retinotoxicity (Armstrong *et al.,* 1982). In rabbits, sodium Iodate develops or causes retinal pigment degeneration (Orzalesi and Calabria, 1967). In a research where rabbits were administered sodium Iodate intravenously, atrophy and marked pigmentation of the retina was observed (Wada, 1960). Adverse ocular effects is not limited to the pigment epithelium of the retina, it also affects the blood- retinal barrier, rhodopsin regeneration, ERG effects (Sorsby and Reading, 1964: Sorsby, Campochiaro *et al.,* 1986; Korte *et al*., 1989).

Moringa oleifera (*M. oleifera*) is commonly known as *Moringa*, it has different names is different languages; in English is called Horseradish tree, Ben tree and Drumstick tree, in Spanish it is called Moringa, Ben and Angela, in Hindi it is called Saguna and Sainjna (Mishria *et al,*. 2011). It originates from South Asia; it is a small, fast growing, evergreen plant. Antioxidant properties of moringa has been established: ethanol and aqueous methanol extract of dried leaves have been found to have radical scavenging and antioxidant properties (Anwar, 2007). The leaves are natural sources of antioxidants (Lalas and Tsakins, 2002: Siddhuraja and Becker, 2003).

Histologically, the retina has 10 distinct histological layers as listed below:

1. The retina pigment epithelium layer (RPE): RPE cells are held together by junctional complexes to form a continuous epithelial monolayer; with tight junctions. This contributes to the formation of the outer blood-retina barrier between the outer retina and its choroidal blood supply (Thuman *et al.,* 2006: Kanski and Miewski, 2002).
2. The photoreceptor layer: consisting of the rods and the cones.
3. The outer limiting membrane (OLM): a pseudo membrane that separates the processes of the rod and cone from a layer of densely packed nuclei. It is formed by junctional complexes between adjacent Muller cells; also between Müller and photoreceptor cells.
4. The outer nuclear layer (ONL): consisting of the cell bodies of the rod and cone photoreceptors (Kolb *et al.,* 1995)
5. Outer plexiform layer (OPL): it has two components consisting of the projection of the photoreceptor cells, horizontal and bipolar cell axons and their synaptic connections. The terminals of the photoreceptor cells form the synapses between the photoreceptor, bipolar and horizontal cells (Wurtz *et al.,* 2000: Roof *et al.,* 2000).
6. Inner nuclear layer (INL): it consists of the nuclei of the amacine, bipolar, horizontal, interplexiform and Muller cells (Newman, 2000: Wurtz *et al.,* 2000)
7. Inner plexiform layer (IPL): it is the second retinal processing layer with bipolar, amacrine and ganglion cells. The synapses between the dendrites of ganglion cells and amacrine cells and axons of bipolar cells are seen; it shows sublayering into six lamina. This facilitates parallel representation and processing of the photoreceptor input through specific synapses between the bipolar, amacrine, and ganglion cells in each of the six lamina of the IPL (Zhu, 2000).
8. Ganglion cell layer: it consists of the nuclei of the ganglion cells (1.2 million cells). Presence of displayed amacrine cells, astrocytes, endothelial cells and pericytes. In the perifoveal macula, there are about eight to ten rows of nuclei (60- 80µm), this makes this part very thick. Outside the macula, the row decreases to just one role (10–20 µm) (Sharma *et al.,* 2003).
9. Inner limiting membrane: it acts as the boundary between the retina and the vitreous body. The glial cells (astrocytes and muller cells) form the layer (Kolb *et al.,* 1995).
10. Nerve fiber layer (NFL): the ganglionic axons travel towards the optic nerve head within the nerve fiber layer. The axons are accompanied by astrocytes in the nerve fiber layer and are separated into small bundles by the cellular processes of Müller cells and the internal limiting membrane (Radius and Andrson, 1979).

The study was primarily aimed at studying the effects the oral treatment of the ethanoic extract of Moringa oleifera will have on the Sodium Iodate induced retinopathy in Wistar rats.The study involved the investigation of the biochemical and histoarchitectural changes in the retina in the retinopathy rat model following oral treatment with the ethanolic extract of moringa.

II. Materials And Methods

Plant Extract Preparation

The *M.oleifera* leaves were obtained from the Nigeria Natural Medicine Development Agency Federal Ministry of Science and Technology. It is situated at 9, Kofo Abayomi street, Victoria Island Lagos. The collected plants were taken to the Botany Department at the School of Biological Sciences, Obafemi Awolowo University for identification.

The leaves were cleaned, air-dried for 2 weeks after which it was pulverized into dry powder using industrial laboratory grinder. Extraction of the phytochemicals was done by soaking the powder in ethanol for 72 hours after which the extract was filtered. Extract was obtained by filtration followed by evaporation of the solvent in an oven at 40°C. The paste was weighed and used to prepare stock solution. The paste was dissolved in distilled water for the preparation of the stock solution.

Animals Grouping and Treatments

The rats were kept and cared for in Babcock University Animal house. In the animal house, rooms were allocated to each department. The room was well ventilated with room temperature 29 + 2ºC and relative humidity of 40 - 70%, with a 12 hr natural light dark cycle.

The rats were provided with plastic cages with wood shafts as their beddings and wire screen tops, which had a part designated for feed and another part designated for water. The wood shafts were replaced every 2 days to maintain good hygiene, and the rats were provided with big pellet feed and water daily.

The protocol of experimentation was according to the guide to the care and use of animals in research and teachings approved by the Babcock University Research Ethic Committee (BUHREC). The animals were divided into 4 groups, 8 per group, having a total of 32 rats. Group A is the control group and Group B, C, D are the experimental group; the table below shoes animal grouping and treatment regimen.

GROUPS	TREATMENT
A	Control: distilled water only along side with feed for 11 days.
B	Sodium Iodate (50mg/kg body weight) (Zhou *et al.,* 2014) only for 1 day along side with feed.
C	Sodium Iodate (50mg/kg body weight) (Zhou *et al.,* 2014) for 1 day and Ethanolic extract of *M .oleifera* leaves (500mg/kg body weight) (Owolabi *et al.,* 2014) for 10 days along side with feed.
D	Sodium Iodate (50mg/kg body weight) (Zhou *et al.,* 2014) for 1 day and Ethanolic extract of *M .oleifera* leaves (200mg/kg body weight) (Owolabi *et al.,* 2014) for 10 days along side with feed.

The control group animals were administered an equal volume of distilled water for 11 days while the experimental groups were administered an equal volume of Sodium Iodate and also Group C and D were administered *M.oleifera*.

Sacrifice of Experimental Animals and Organ Harvest

The experimental subjects were euthanized through cervical dislocation 24 hours after the last administration. The fur on the head of the rat was scraped off, and then the neck of the rat was cut off. The skull of the rat was opened up, the brain was removed and then the left and right retina of the subject was extracted from the eye socket in the skull and it was weighed. One of the retinas was fixed in sucrose solution and stored in the freezer and the other retina was fixed in 10% formol saline. Then some retinas were taken for homogenate, and neurotransmitter assays and others were then processed for histological analysis. The eye was homogenized and a homogenous solution was obtained.

The GABA Neurotransmitter Assay [Elisa, 2015]

The GABA neurotransmitter was assayed for in this study. Tissues were rinsed in ice-cold PBS (0.01 mol/ L, pH 7.0- 7.2) to remove excess blood thoroughly and weighed before homogenization. The tissues were minced to small pieces and homogenized in 5- 10ml PBS with a glass homogenizer on ice. The resulting suspension was sonicated with an ultrasonic cell disrupter or subjected to two freeze- thaw cycles to further break the cell membranes. After that, the homogenates were centrifuged for 5 minutes. The supernatant was removed and assayed.

All reagents, samples and standards were prepared; 50µL standard or sample was added to each well. Then 50 µL prepared Detection Reagent was added to A immediately; shaken, mixed and incubated for 1 hr at 37^0 C. Then it was aspirated and washed 3 times. Then 100µL prepared Detection B Reagent was added, and was incubated for 30 minutes at 37^0C. Then it was aspirated and washed 5 times. Added to this was 90µL substrate solution, and incubated 15- 25 minutes at 37^0C. Then 50µL Stop solution was added. Reading was done at 450nm immediately.

Glutamate Procedure (Sigma- Aldrich, 2016)

Procedure started with dilution of 10ml of the 0.1 M Glutamate Standard with 990 µL of the Glutamate Assay Buffer to prepare a 1mM standard solution. Add 0, 2, 4, 6, 8 and 10 µL and the 1m M standard solution into 96 well plate, generating 0 (blank), and 2, 4, 6, 8 and 10 nmole/well standards. Add glutamate Assay buffer to each well to bring the volume to 50µL.

The Reaction Mixes according to the scheme was set up. 1.00µL of Reaction Mix was required for each reaction (well). 100µL of the appropriate Reaction Mix was added to each of the wells. It was mixed well using a horizontal shaker by pipetting and incubating the reaction for 30 minutes at 37^0C.The plate was protected from light during incubation. The absorbance at 450nm was measured(A_{450})

Tissue Processing for Histological Procedure: Haematoxylin and Eosin

The general morphology and histoarchitecture of the retina of the various groups was demonstrated using Haematoxylin and Eosin staining method. The retina was excised and fixed in 10% formal saline to preserve it and also to inactivate degrading enzymes. The retina was dehydrated by passing them through increasing concentrated alcohol solutions ending in 100%. The alcohol content of the retina was then removed in toluene in a process known as clearing. After clearing, the retina placed in melted paraffin until they were completely infiltrated in the substance. The Paraffin infiltrated tissues were then placed in a small mold with melted paraffin and allowed to harden. The resulting paraffin blocks were trimmed to expose the tissue for sectioning on a microtome; the tissues were cut between 1 and 10nm and then mounted on a glass slide .

The slides were deparaffinized in 3 changes of xylene for 3 minutes each. The slides were hydrated in 100% alcohol and 95% alcohol, 2 changes for 3 minutes each, and rinsed in distilled water until ripples disappeared from slides. The slides were placed in Hematoxylin for 8 – 15 minutes. The slides were rinsed in tap water until the water ran clear. The slides were decolorized in 1% acid alcohol, 3-6 quick dips. Differentiation was checked for microscopically. The nuclei were viewed as distinct and the cytoplasm was uncolored. The slides were rinsed in tap water until ripples disappeared from the slides. The slides were dipped in Bluing A gent 3- 5 long dips. This process was followed by washing the slides in luke- warm tap water for 5 3ioiminutes (37^0- 40^0 C). The slides were stained in Eosin for 30 seconds to 2 minutes. The slides were hydrated in 95% alcohol and absolute alcohol, 3 changes each for 2 minutes. The slides were cleared. It was done in 3 changes of xylene for 2 minutes each. A cover glass was mounted on each slide.
(Luna, 1992 : Sheehean *et al*,. 1980)

Histochemistry: Periodic Acidic Sciff

Reagents included 0.5% Periodic Acid Solution [Periodic acid 0.5gm]; distilled water [100.0ml] and The Schiff Reagent [Pararosaniline HCL, 1% and soium metabislfite 4%, in hydrochloric acid 0.25 mol/L] and Hematoxlin Solution [Certified hematoxylin 6g/L, sodium iodate 0.6g/L, aluminium sulfate 52.8g/L and stabilizer].

The sectioned tissues were deparaffinized and hydrated to water to deionized water. The slides were immersed in 0.5% periodic solution for 5 minutes at standard room temperature. After that, the slides were rinsed in distilled water several times. Then the rinsed slides were immersed in Schiff reagent for 15 mins at standard room temperature. Then the slides were washed with water for 5 mins. The slides were then counterstained in Hematoxylin solution for 90 seconds. Then the slides were rinsed with water.

Immunohistochemistry: Glial Fibrillary Acidic Protein (GFAP) (Newcomer Supply, 2015)

The Sections were deparaffinised thoroughly in three changes of xylene, 3 minutes each and they were hydrated through two changes each of 100% and 95% ethyl alcohols, 10 dips each. Then, it was washed well with distilled water. Then endogenous peroxide was quenched with 2 drops of freshly made 3% hydrogen peroxide for 5 minutes. The slides were washed gently in distilled water and were rinsed in two different changes of Tris buffered saline. The excess buffer was tapped off. The Glial fibrillary acidic protein primary antibody was applied and incubated at room temperature for 30mins. The slides were rinsed in two changes of buffer solution. The excess buffer was tapped off. The amplifier was applied to Incubate for 10 minutes. The slides were rinsed in two changes of buffer. The excess buffer was tapped off; HRP polymer was applied. It was then incubated for 10 minutes. The slides were rinsed in two changes of buffer.

Prepared required quantity of DAB substrate chromogen was added: 4ml deionized water, 2drops acetate buffer, 1 drop AEC chromogen, 1 drop 3% hydrogen peroxidase. Other parts of the procedure was as follows: 2 drops substrate reagent was applied and then incubated up to 10 minutes. The slides were checked microscopically for adequate chromogenic development. Then when the sufficient staining has been achieved, the slides were rinsed in deionized water for five minutes. The excess was wiped off. Then it was counterstained lightly with Mayer's hematoxylin for 2 minutes. It was rinsed in gently running tap water to blue the sections. Then it was dehydrated in two changes each of 95% and 100% ethyl alcohol and cleared in three changes of

xylene, 10 dips each. Glycerol gelatin or other compatible aqueous mounting media was applied and then carefully covered with coverslip.

Statistical Analysis of Quantitative Results

The data gotten from the entire test was expressed as mean ± Standard Error of Mean (S.E.M). The stastical signifance was evaluated by one way analysis of variance (ANOVA) using Graph Pad Prism (version 5). A value $p < 0.05$ was considered statistically significant.

III. Results

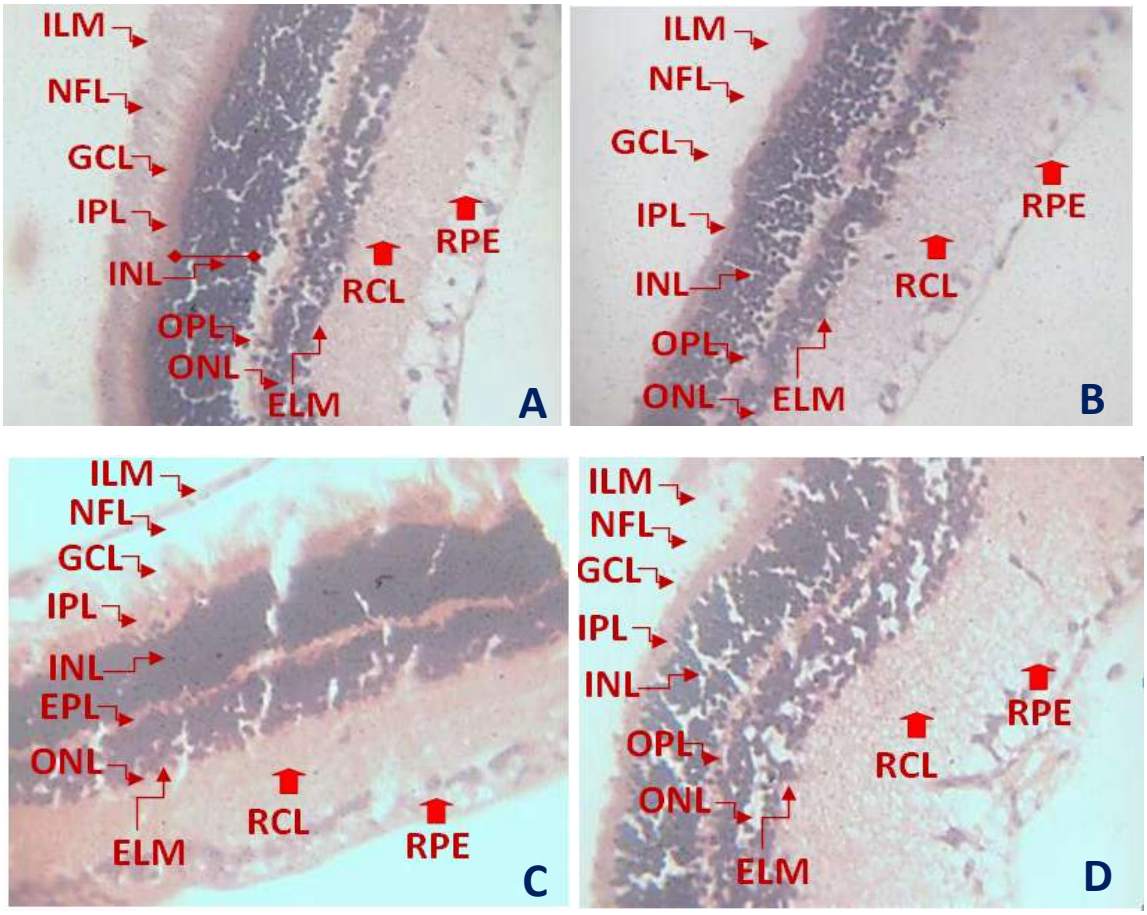

Figure 1: Photomicrographs of the retina of the experimental animals [H&E; X640]. Retinal epithelium is distorted in all treated groups. The inner lining epithelium, nerve fibre layer. Ganglion cell layer and part of the inner Plexiform layers are destroyed by sodium iodate. Treatment could not sufficiently prevent the damage.

Legend:

Retinal layers are indentified for the innermost to the outermost as follows [McCaa, 1982]:

[1] Inner Limiting Membrane [**ILM**] with basement membrane reinforced by the Müller cells; [2] Nerve Fibre Layer [**NFL**] that consists of the axons of ganglion cells; [3] Ganglion Cell Layer [**GCL**] that has the nuclei of ganglion cells; [4] Inner Plexiform Layer [**IPL**] containing the synapse of the bipolar cell axons and dendrites of the ganglion and amacrine cells; [5] Inner Nuclear Layer [**INL**] consisting of nuclei and perikarya of amacrine cells, bipolar cells and horizontal cells; [6] Outer Plexiform Layer [**OPL**] consisting of the projections of rods and cones forming synapses with dendrites of bipolar cells; [7] Outer Nuclear Layer [**ONL**] consisting of photoreceptor cell bodies- rods and cones; [8] External Limiting Membrane [**ELM**] which is the layer that separates inner segment portions of the photoreceptors from their cell nucleus; [9] Layer of rods and cones [**RCL**] which are the photoreceptors; [10] Retinal Pigment Epithelium [**RPE**] which is single layer of cuboidal cells demarcating the retina from the choroid.

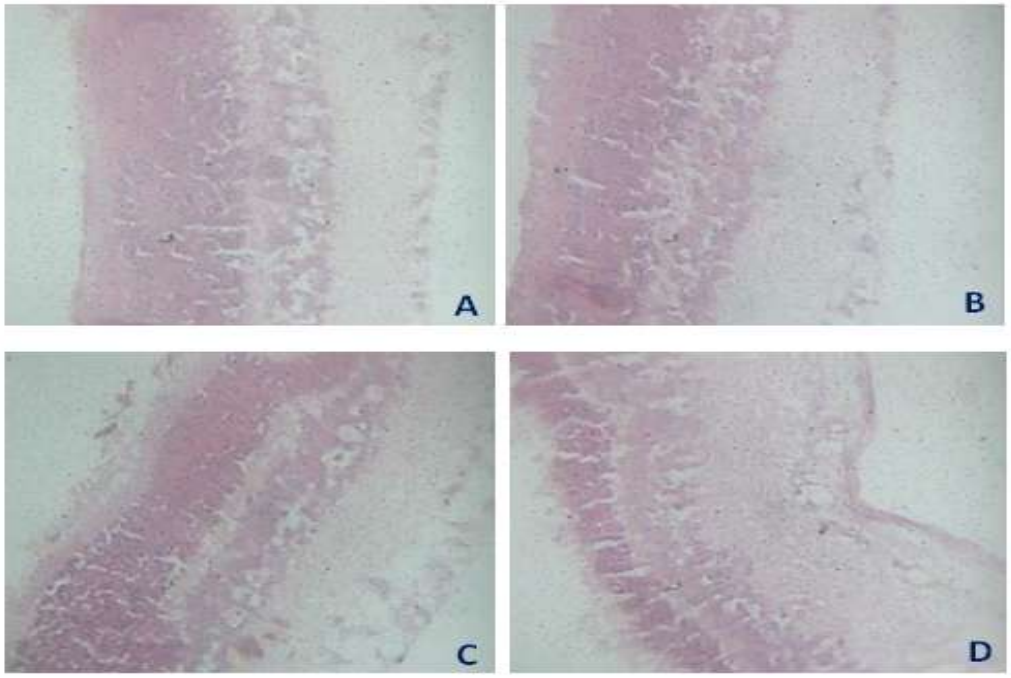

Figure 2: Photomicrographs of the retina of the experimental animals [PAS; X640].

Retinal epithelium in A is normal and structurally suitable to serve as a standard reference for the other experimental groups; Photomicrographs of the retina of the Group B experimental animals [PAS X1600] shows that retinal cells are relatively poorly demonstrated for PAS; **p**hotomicrographs of the retina of the Group C experimental animals [PAS; X1600] shows that retina is poorly demonstrated for PAS; photomicrographs of the retina of the Group D experimental animals [PAS; X1600] shows that retinal stained for PAS showing an improvement over the Groups B and C.

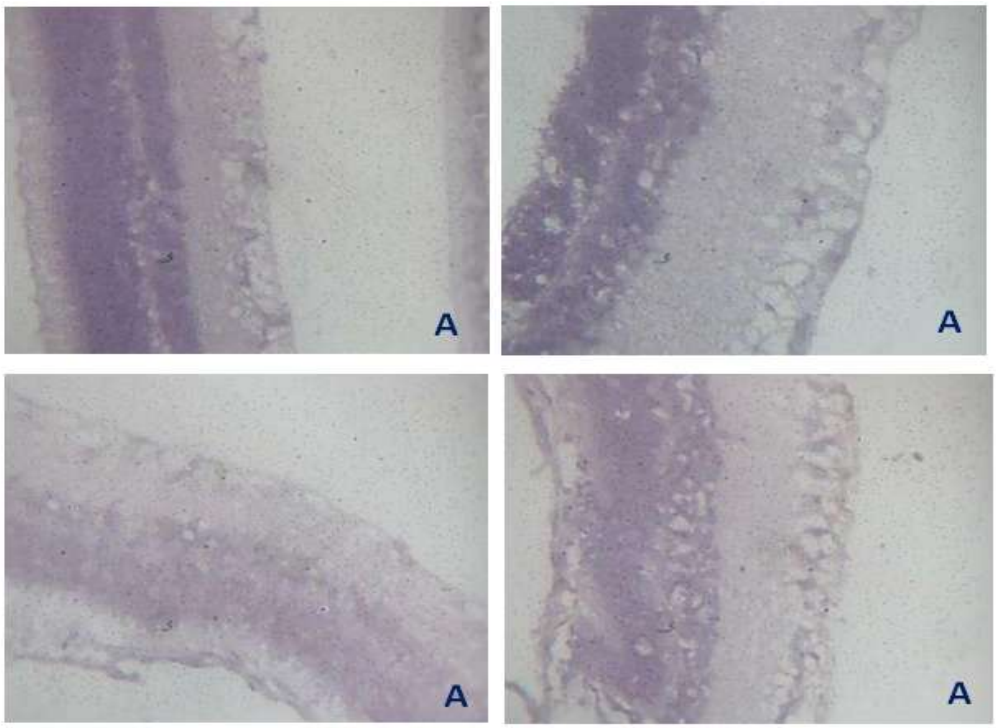

Figure 3: Photomicrographs of the retina of the Groups A-D experimental animals [GFAP; X640]. In Group B, retina Outer Segments of Rods and Cones was ameliorated. This effect is mildly ameliorated in Groups C and D. [GL= Ganglion Cells; BCN= Bipolar Cell Nuclei; NRC= Nuclei of Rods and Cones; OSRC= Outer Segments of Rods and Cones; PL= Pigmented Layer].

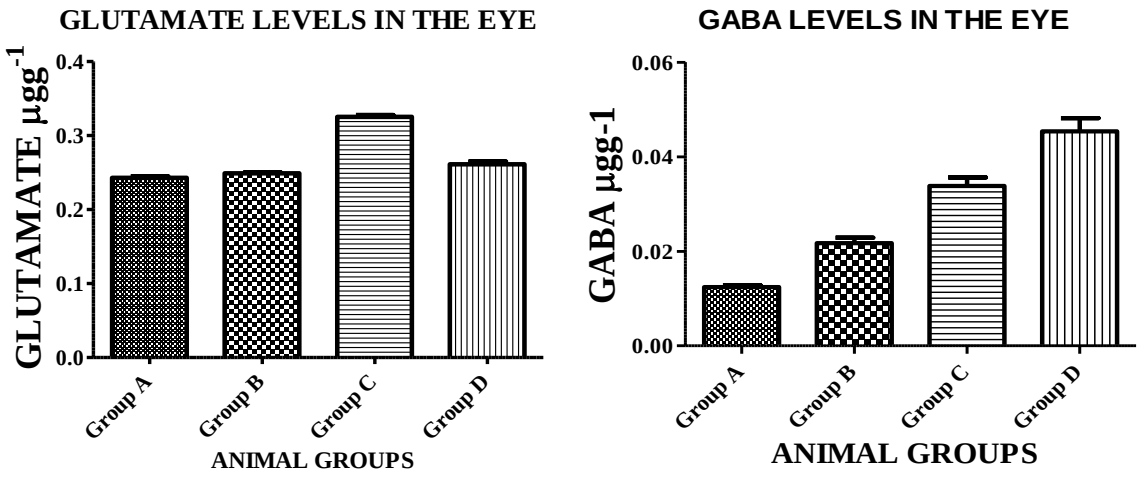

Figure 4: Bar charts showing the levels of glutamate and GABA in the eye. Treatment disrupted glutamate activities in the eye while GABA was increased in all groups level to the control,

IV. Discussion

The histology of the retina shows that the retina is structurally organised in highly defined layers that are made of cells and their processes. In histological demonstration of the retinal histology, especially in the haematoxylin and eosin [H&E] staining technique typically employed in routine histological demonstrations, a number of features are found within the layered retina; these include the ganglion cells layer [GL], the layer of bipolar cell nuclei [BCN], the layer of nuclei of rods and cones [NRC], the outer segments of rods and cones [OSRC] and the pigmented layer [PL]. In a cross section the ganglion layer is the most superficial while the pigmented layer is the deepest. Also, in typical degeneration of the retina such as macular degeneration or induced retinopathy, the pigmented layer degeneration is a typical sign of impending retina degeneration linkable to blindness. Also for the photoreceptor cells, the rods are first vulnerable to destruction, then the cones. These features are observable in the current experiment's results.

General Structure of Tissues

The retina of the control group [Group A] animals is normal as it has the basis layers of the retina properly defined as listed above. This implies that it is normal and structurally suitable to serve as a standard reference for the other experimental Groups B-D. In Group B the retina histological organisation is distorted and disrupted. The retina is reduced in thickness and the pigment layer is structurally distorted. Ganglion layer is relatively lost and barely observable. These features are typical of retinal degeneration. Also, they show that the administered agent, sodium iodate caused retinal damage that can further deteriorate the retina, leading to blindness due to compromised photoreception. The retina is also distorted in the Group C animals, yet less severe when compared to the Group B when the thickness of the retina and the degree of ganglion layer loss is considered. However, the pigment layer remains distorted and this is also an indication of impeding or possible retinal degeneration. Therefore, retinal degeneration is only fairly limited. Hence, the intervention that was employed might not be potent enough to have arrested retinal degeneration following sodium iodate induced damage. It could only either limit the rate of deterioration or limit the extent.

The retina was relatively more preserved in the Group D when compared with its histological demonstration in Groups B and C as previously described. Ganglion layer was preserved to a lager extent; also the pigmented layer. These two features are important in determining potential or impending further retinal degeneration. Therefore, it could be inferred that the intervention in the Group D prevented or ameliorated retinal degeneration that was induced by sodium iodate administration. To this end, the higher dose of the intervention in Group D resulted in better protection of the retina. It is also possible, that retina degeneration might be prevented in this group as the pigmented layer was relatively preserved relative to the other treated groups and when measure with the original reference control group.

Sodium iodate, therefore initiated retina degeneration in the experimental animals. Higher dose of the intervention agent preserved the retina from extensive degeneration. Therefore, the ability of the intervention to prevent retina degeneration was dose dependent. The intervention agent, therefore showed potentials to ameliorate retinal degeneration. This would be due to its phytochemistry, and especially the antioxidant properties of the active ingredients (Zhou *et al.,* 2014).

Special Features- PAS Expression

Normal PAS demonstration of the control group animals' retina could serve as a suitable standard reference for the other groups [B-D]. Certain layer expressed PAS poorly in Group B especially the rods and cones layers as well as the pigmented layer. These are evidences of retinal damage. PAS expressions improved in the Group C over the Group B while the expression in the Group D was also an improvement over the Group C. These observations are in line with the structural aberrations observed in the H&E demonstrated photomicrographs. They also showed that the reported abnormalities affected tissue function relative to glycogen and mucosubstances properties within the tissues (Zhou *et al.,* 2014). Results also showed that the higher dose of the extract protected the retina to a greater extent.

Special Immunohistochemical Feature

The Glial acidic fibrillary protein demonstration of the retina and the photomicrographs of the retina of the Groups A-D experimental animals [Figure 3] correlate with other structural and functional observations. In Group B, retina Outer Segments of Rods and Cones was disrupted. This effect is ameliorated in Groups C and D; however, effects was relatively more effective in the latter.

Biochemical Alterations in the Eye

Glutamate is normally an excitatory neurotransmitter in the retina (Makoto, 2013). Alteration is Glutamate activities are indications of toxicities and accompanying complications in the retina. Interestingly, levels were increased the more when treatments were administered. While acute efflux and alterations in glutamate occurs in toxic retinal assault; glutamate alterations might have therapeutic effects in repair (Dhingra and Vardi, 2012). Glutamate is crucial to retinal functional integrity and photoreception; a report suggested that mGluRs could play a trophic role in retinal development and after damage. (Dhingra and Vardi, 2012). Again, fluctuations in retinal glutamate activities and metabolism have been associated with retinal detachment, and glutamate efflux is linked to excitotoxicity in the neural retina (Sherry and Townes-Anderson, 2000).

GABA is retinal major inhibitory neurotransmitter. Its ration relative to glutamate is an indication of retinal functional integrity and a marker for abnormalities (Guoping *et al.,* 2017). This ration is a marker of retinal development and myopia (Guoping *et al.,* 2017). The current investigation shows alterations that indicate retinopathy; thus giving a correlating biochemical basis for the observed structural alterations. There are however insufficient evidences to suggest that the administered agents could effectively reverse the induced damage to the retina.

V. Conclusion

The current investigation shows that the sodium iodate model of retinopathy resulted in the damage of the retina from the innermost layers. It also shows that the antioxidant rich *Moringa oleifera* could mildly retard the rate or the extent of retinopathy. However, there is not enough evidence to suggest effective repair or an almost perfect protection.

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