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Ethanolic Stem Bark Extract of *Khaya Senegalenesis* Ameliorates Cerebral Ischemia in Wistar Rats

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ABSTRACT

Khaya senegalenesis is the most popular African medicinal meliaceous plants used traditionally in the management of neurodegenerative disorders. This study aimed to investigate the neuroregenerative effects of *Khaya senegalenesis* bark extract on cerebral ischemia in adult wistar rats. Twentyfive adult wistar rats weighing 300-400 g were randomly divided into five groups (each n=5), and in 2 phases of experiment. Phase 1 stroke induction (group B - E) and phase 2, treatment (group D & E) at low and high doses of 150 mg/kg and 250 mg/kg body weight of *Khaya senegalenesis*. 4 groups (groups B - E) were stroke induced by occlusion of middle cerebral artery through incision of the cervical region. Group A were control, administered distilled water, group B, stroke-induced and evaluation, group C, stroke-induced and untreated to assess recovery, group D and E, stroke-induced and treated. At the end of 2 weeks, the animals were sacrificed by cervical dislocations, blood sample collected to measure marker of oxidative stress, serum electrolytes and the brain tissues were perfused with phosphate buffer solution, harvested, processed and stained with H& E for histological observation. Normal histoarchitecture of cerebral cortex observed in control (group A) while group B revealed intact meninges, with congested meningeal vessels, mild tissue gliosis, lymphocytic infiltrates, show cerebral ischemia induced expression. Group C shows intact meninges, with moderate to severe lymphocytic infiltrates, vascular congestion, mild tissue oedema and gliosis. On treatment, low dose of *Khaya senegalenesis* shows minimal cellular proliferation and moderate gliosis while there was marked improvement in neurovascular unit in *Khaya senegalenesis* high dose. SOD, GSH, Na^+ and K^+ significantly decrease while MDA increase significantly in stroke induced rats. In the treated groups, SOD, GSH, Na^+ and K^+ values significantly increase while MDA significantly decrease. A dose of 250 mg/kg body weight of *Khaya senegalenesis* ethanol stem bark extract has been shown to ameliorate induced degenerative changes in the cerebral cortex causes by stroke-induced adult wistar rats.

KEYWORDS: Cerebral ischemia, Cerebral cortex, *Khaya senegalenesis*, Neurovascular unit.

INTRODUCTION

The therapeutic measures to treat stroke as the third most common cause of death remains a critical problem to both researchers and clinicians, due to failure in various clinical trials to combat the disease. Conventional single therapeutics measures used to treat stroke, has been reported to fail clinically.[1,2]

Stroke, also called brain attack, is currently reported as a serious burden on the society because it has been recognized as the major cause of dead and adult long-term disability in the world. Brain attack may result as clot breeches the blood supply to the brain, and also as a burst from the blood vessel supplying the brain. Several reports have shown that brain ischemia has a wide range of disturbances which include cardiovascular and respiratory disorders, brain trauma, and any condition leading to prolonged arterial hypotension or intracranial hypertension.[2-4] Cerebral ischemia is a condition that results from acute occlusion of blood vessels that supply the brain or severe narrowing leading to impaired delivery of blood flow.[5-7] Since the symptoms depend on the type of stroke and the area of the brain affected, knowing a particular blood vessel supplying specific area of the brain where brain attack occurs could help to identify the types of stroke.

Tissue-type plasminogen activator (t-PA) is reported to be the first medication approved by the FDA for the management of stroke, but still records a high limitation in clinical success.[1,2]

Thrombolytic therapy has been reported to be the most effective therapeutic strategy to prevent brain injury and validated a reduction in patient mortality of cerebral infarction. Notwithstanding, clinical trials of thrombolytic therapy of single therapeutic strategy have been reported disappointing because of its adverse effects after a two-week window of treatment. The use of medicinal plants in neuroregenerative and neurorepairs may also be adopted for effective neuronal protection therapy. Therefore, in search of these, herbal medicinal approaches have been adopted; these include herbal drugs that can ameliorate the neurological damage

associated with stroke.[2,8-10]

Khaya Senegalensis. (Family: Meliaceae) is one of the most important medicinal plants use in traditional system of medicine in Africa, due to its multifaceted medicinal usefulness. In Nigeria, it is widely used by different ethnic groups and is commonly known as African mahogany (English), Aganwo (Yoruba), Madachi (Hausa), and ono (Igbo). *K. Senegalensis* bark has been reported used in the treatment of different diseases; anti-sickling, anti-cancer effects, anti-hyperglycemic and as free radical scavenger.[11-18] Most inherently, its polyphenol composition and antioxidant properties have been evaluated and it showed high capacity to scavenge reactive oxygen species, hence its ability of chemopreventing diseases like neurodegenerative disorders, asthma, diabetes, cardiovascular disorders and different forms of cancer are validated.[19-23]

This study aimed at determining ameliorative effects of ethanolic stem bark extract of *khaya senegalenesis* on stroke-induced cerebral cortex in male wistar rats.

MATERIALS AND METHODS

Plants Material: Fresh bark of Khaya senegalensis was collected from Iyango forest in Cross River State, Nigeria. The plant material was identified and authenticated in the Department Botany of University of Lagos, Lagos, Nigeria, with ID No. 8004.

Extract Preparation: Fresh bark of *Khaya senegalensis* was well cleansed diced into smaller pieces using s sterile knife and air dried at room temperature for a period of two weeks. The stem bark was then oven dried at 50°C for 3hrs and thereafter crushed into semi powder using mortar and pestle. 244g of coarse powder of bark *Khaya senegalensis* was packed into a thimble and inserted to the Soxhlet extractor. The Soxhlet was inserted into the quick fit bottom flask containing solvent. The solution was left to concentrate using a rotary evaporator, 221g of the dried extract of *Khaya senegalensis*, was collected and preserved at 4°C for further use.

Laboratory Animals: Twenty-five adult healthy albino wistar rats weighing between 300 – 400 g were used for this study. The animals were obtained from the Laboratory animal Centre of the College of Medicine, University of Lagos, Lagos, state Nigeria. They were housed in well-ventilated plastic cages, kept and maintained under standard laboratory conditions. They were allowed free access to food and tap water *ad libitum*. Animal experiments were carried out in line with the guidelines of the Health Research Ethics Committee of the College of Medicine of the University of Lagos, Nigeria, with CMULHREC No. CMUL/HREC/06/18/362.

At the end of two weeks acclimatization, animals were weighted and randomly assigned to 5 groups (each n=5).

Surgical Procedure and Preparation of Stroke Model: The animals in 4 groups (groups B - E) were anesthetized using intramuscular atropine of 0.5mg/kg and 0.5mg/kg body weight of ketamine hydrochloride, after which they are carefully laid in supine position and their limbs spread outwardly, and cello tape was used to hold each limb firmly to the experimental platform. The skin at the cervical region carefully shaved and cleared with sterilizer. Thereafter, an operating microscope is set to view the area while carrying out surgical procedures. A midline neck incision about 3-4cm was carrying out from jaw to about one inch to the manubrium, the skin and the subcutaneous layer carefully reflected and exposed. On exposure of the subcutaneous layer, a midline neck incision of 3-4cm continue and carefully retracted to exposed the muscular layer, where underlying muscle fascia and submandibular glands and trachea were carefully retracted and clipped to exposed the vascular layer, then the vague nerve and the right common carotid artery (rCCA) traced and separated, and the rCCA temporarily ligated using a silk suture while occlusion process was carried out. The right external carotid artery (rECA) and the right internal carotid artery (rICA) were traced and isolated, following the course of rCCA. At the bifurcation of the rCCA, two temporary ligations were made on rECA, at the proximal and it distal end. Other details to occlude the right MCA was achieved by the methods Coyle[24] and Calloni *et al*.,[25]. Occlusion of the right middle cerebral artery (rMCA) was left for a period of 60 minutes and the mid-line neck incision was temporarily closed after the occlusion. After 60 minutes of occlusion, temporarily suture of the mid-line neck was carefully removed and occluding filament was removed. Other reperfusion method was done by the method of Calloni *et al*., (2010).[25] The mid-line neck incision was well suture and the animals were placed back in their groups; group C, stroke-induced and untreated to check recovery, group D and E, stroke-induced and treated.

Experimental Design and Treatment: The animals were grouped as follow; Phase 1 stroke induction (group B - E) and phase 2, treatment (group D & E) at low and high doses of 150 mg/kg and 250 mg/kg body weight of *Khaya senegalenesis* for a period of 2 weeks. 4 groups (groups B - E) were stroke induced by occlusion of middle cerebral artery through incision of the cervical region. Group A were normal control, administered distilled water, group B, stroke-induced and evaluation, group C, stroke-induced and untreated to check recovery, group D and E, stroke-induced and treated. At the end of 2 weeks, the animals were euthanized

Antioxidant Enzymes Assay: The following antioxidant enzymes activities were determined spectrometrically as follows:

Determination of Superoxide Dismutase (SOD) activity: Superoxide Dismutase activity was determined by its ability to inhibit the auto-oxidation of epinephrine determined by the increase in absorbance at 480nm as

described by Sun and Zigma (1978).[26] The reaction mixture (3 ml) contained 2.95 ml 0.05 M sodium carbonate buffer pH 10.2, 0.02 ml of liver homogenate and 0.03 ml of epinephrine in 0.005 N HCL was used to initiate the reaction. The reference cuvette contained 2.95 ml buffer, 0.03 ml of substrate (epinephrine) and 0.02 ml of water. Enzyme activity was calculated by measuring the change in absorbance at 480 nm for 5 min. $\Sigma = 4020 M^{-1} cm^{-1}$

Determination of Catalase activity (CAT): Catalase activity was determined according to Sinha, *et al*.,[27]. It was assayed colorimetrically at 620nm and expressed as µmoles of H_2O_2 consumed/min/mg protein at 25^0C. The reaction mixture (1.5ml) contained 1.0ml of 0.01M phosphate buffer (pH 7.0), 0.1ml of tissue homogenate and 0.4ml of 2M H_2O_2. The reaction was stopped by the addition of 2.0ml of dichromate-acetic acid reagent (5% potassium dichromate and glacial acetic acid were mixed in 1:3 ratio). $\Sigma = 40 M^{-1} cm^{-1}$.

Estimation of Reduced Glutathione (GSH): The reduced glutathione (GSH) content of liver tissue as non-protein sulphydryls was estimated according to the method described by Sedlak and Lindsay[28]. To the homogenate 10% TCA was added, centrifuged. 1.0ml of supernatant was treated with 0.5ml of Ellmans reagent (19.8mg of 5,5-dithiobisnitro benzoic acid (DTNB) in 100ml of 0.1% sodium nitrate) and 3.0ml of phosphate buffer (0.2M, pH 8.0). The absorbance was read at 412nm.
$\Sigma = 1.34 \times 10^4 M^{-1} cm^{-1}$.

Determination of Lipid Peroxidation: Malondialdehyde (MDA) an index of lipid peroxidation was determined using the method of Buege and Aust (1978).[29] 1.0 ml of the supernatant was added to 2 ml of (1:1:1 ratio) TCA-TBA-HCl reagent (thiobarbituric acid 0.37%, 0.24N HCl and 15% TCA) tricarboxylic acid- thiobarbituric acid-hydrochloric acid reagent boiled at 100^oC for 15 min, and allowed to cool. Flocculent materials were removed by centrifuging at 3000 rpm for 10 min. The supernatant was removed and the absorbance read at 532 nm against a blank. MDA was calculated using the molar extinction coefficient for MDATBA- complex of $1.56 \times 10^5 M^{-1} CM^{-1}$.

Tissue Processing Procedure: At the end of the stipulated 2 week of the administration, the animals were sacrificed cervical dislocation. Brain tissues were carefully harvested out of the mice, trimmed to remove any blood. The tissues were immediately fixed in phosphate buffer solution; after 72 hours, 2-3mm in thickness were dissected out and post fixed in another freshly prepared phosphate buffer solution and then transferred to a graded series of alcohol.

Phytochemical Screening: Liquid-Chromatography Electrospray – Ionization Mass Spectrometry (LC-ESI) - Qualitative and Quantitative phytochemical analysis of the ethanol bark extract *Khaya senegalensis* was carried out in accordance with Soni and Sosa[30]. And High performance liquid chromatography (HPLC) using reversed-phase ion pairing was adopted in accordance with Grindberg and Williams (2010).

Statistical Analysis: Data were presented as mean ± SEM; analysed using SPSS, ANOVA and Tukey's Post-Hoc test. Statistical Significance was set as $P<0.05^*$, $P< 0.001^*$

RESULTS

Oxidative stress markers: There was a significant decrease in SOD level, in stroke-induced animals (group B &C) compared to group A ($p< 0.0001$). the animals in groups D and E, that was treated with low and high dose of *Khaya senegalenesis* shows a significant increase in SOD level ($p<0.0001$) compared to group B and C (fig. 2a).

In figure. 2b, GSH level of the animals in groups B and C decrease significantly compared to group A ($p< 0.001$) and significantly increase in treatment groups D and E, treated with low and high dose of *Khaya senegalenesis* when compared to group B and C animals.

There was a significant increase ($p< 0.05$) in CAT level in group B and C animals compared to group A while in treatment groups D and E, with low and high dose of *Khaya senegalenesis* shows no significant ($p< 0.05$) compared to group B and C animals. A significant increase ($p< 0.05$) was observed in CAT level in the treatment groups D and E when compared to group A (fig. 2c).

MDA concentration in stroke induced animals (groups B and C), shows a significant increase in MDA concentration compared to group A while MDA concentration decrease significantly in treated animals in groups D compared to groups B and C animals and not significant between groups C and E (fig. 2d).

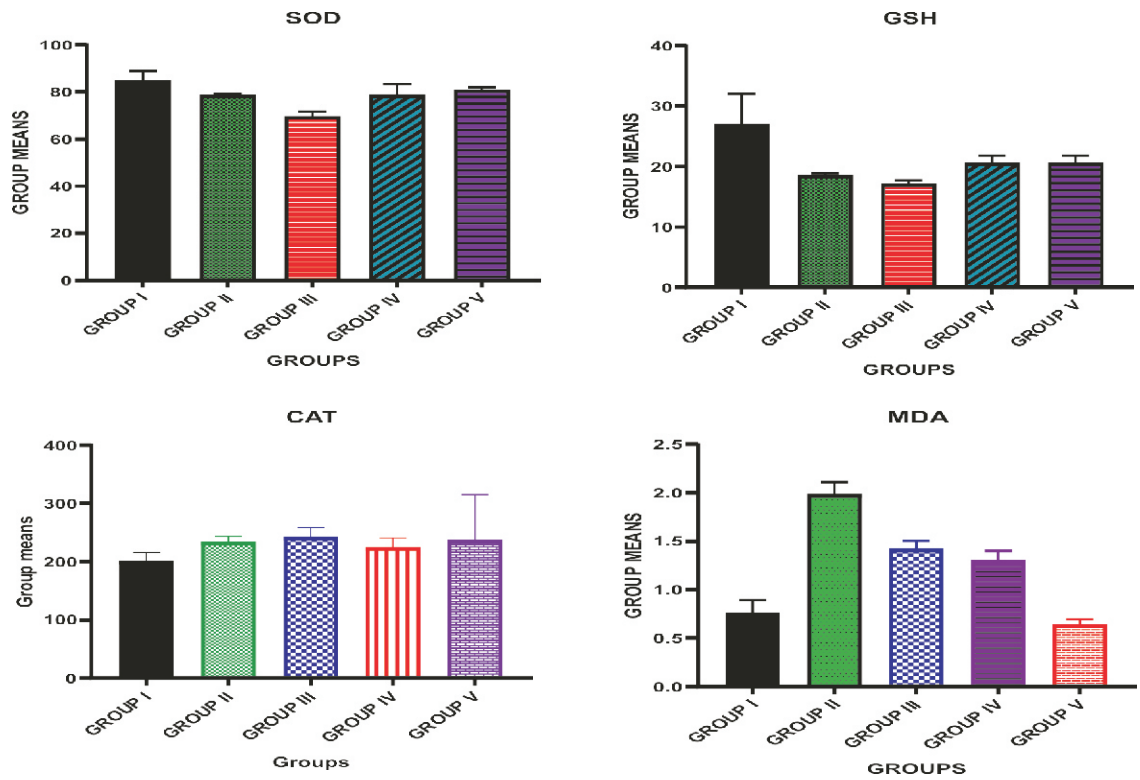

Figure 1: Oxidative stress marker. Group I (vehicle control), group II (negative control I), group III (negative control II), group IV (low dose of *Khaya senegalenesis*), Group V (High dose of *Khaya senegalenesis*). ($p< 0.05$)* = Significantly different from control, ($p< 0.001$) ** = significantly different from control, ($p< 0.0001$) ***. Figures are represented as mean ± SD.

Electrolytes Analysis of ethanol stem bark extracts *Khaya senegalensis*:

Table1 present the results for the electrolytes analysis of ethanol stem bark extracts *Khaya senegalensis*. There was a significant decrease in group B, in serum sodium, potassium and chlorine compared to group A. A significant increase was observed in serum sodium, potassium and chlorine in group C compared to group B. In treatment group D animals, serum sodium, potassium and chlorine increase significantly compared to stroke induce animals (group B and C). In group E treated animals, serum sodium and chlorine increase significant compared to group B and C (Tab. 1).

Table 1 : Electrolytes Analysis of ethanol stem bark extracts *Khaya senegalensis*

	ELECTROLYTES		
GROUPS	Na	K	Cl
GROUP I	133.00±1.4	11.2±0.1	95.00±1.4
GROUP II	$129.44±1.6^{\alpha}$	$8.1±2.1^{\alpha}$	$85.54±1.2^{\alpha}$
GROUP III	$134.50±2.1^{\alpha\alpha}$	$9.75±0.3^{\alpha\alpha}$	95.00±0.7
GROUP IV	$139.00±7.0^{\beta\phi}$	$8.7±3.1^{\beta}$	$102.00±5.6^{\beta\phi}$
GROUP V	$148.00±1.4^{\delta\theta}$	5.55±0.7	$110±50^{\delta\theta}$

Values are Mean ± SD. α significant difference compared to Group A vs Group B; αα significant difference compared to Group A vs Group C; β significant difference compared to Group II vs Group D; δ significant difference compared to Group B vs Group E; ϕ significant difference compared to Group C vs Group D; θ significant difference compared to Group C vs Group E. One-ANOVA followed by multiple comparison tests.

Phytochemical screening: Qualitative analysis of ethanol stem bark extract *Khaya senegalensis* shows the presence of Anthraquinone, Alkaloid, Terpernoid and Cardiac glycoside (Table 1). While quantitative analysis as shows total Alkaloid,and total Cardiac glycoside had higher values compared to total Anthraquinone and the total Terpernoid present (Table 2).

Table 2: Qualitative Phytochemical Analysis of ethanol stem bark extracts *Khaya senegalensis*

S/No	Phytochemicals	status
1	Saponin	-
2	Anthraquinone	+
3	Alkaloid	+
4	Terpernoid	+
5	Flavonoid	-
6	Tannin	-
7	Phenol	-
8	Steroid	-
9	Reducing sugar	-
10	Cardiac glycoside	+

Table 3: Quanlitative Phytochemical Analysis of ethanol stem bark extracts *Khaya senegalensis*

S/No	Phytochemicals	Quantity
1	Alkaloid (%)	29.56
2	Terpernoid (%)	17.63
3	Flavonoid (%)	14.80
4	Steroid (%)	11.66
5	Cardiac glycoside (%)	19.69

Histological Findings:

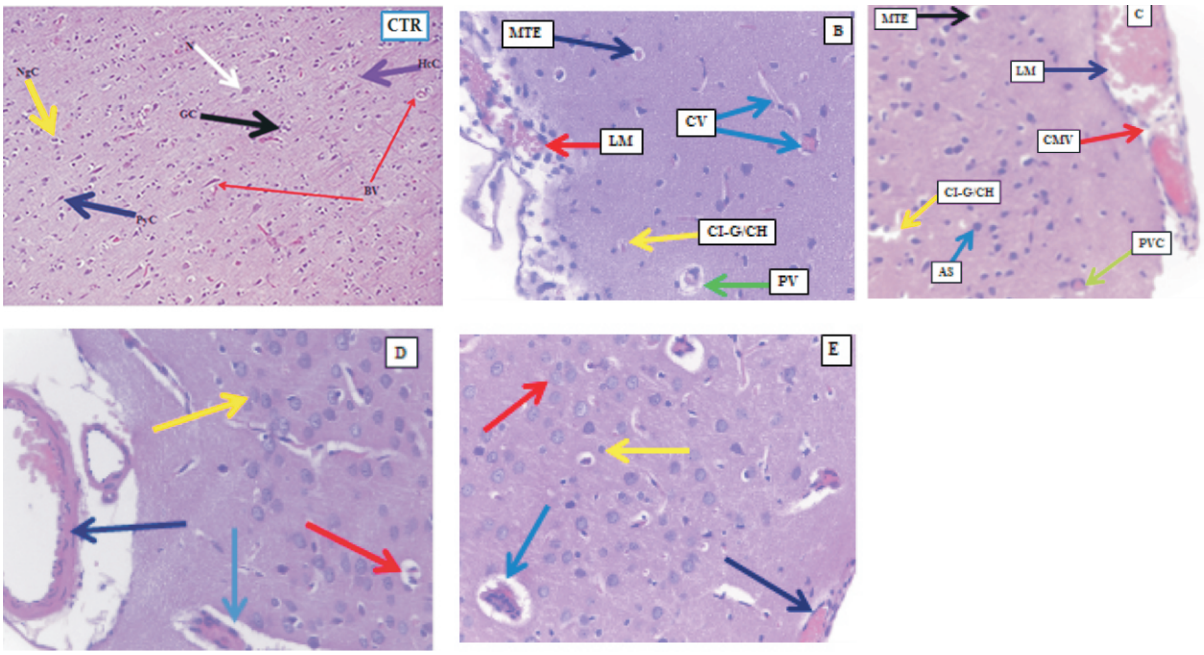

Figure. 2: Cerebral cortex of rats. Control Group A. Group B. cerebral cortex of stroke induced and evaluation, Group C. cerebral cortex of the stroke induced and assesses recovery, Group D. cerebral cortex of the treated group with low dose of KS and Group E. cerebral cortex of the treated group with high dose of KS.

The section of the Cerebral cortex shows normal granule cells (orange arrow) as well as neurogial (yellow arrow). The pyramidal cells (green arrow), blood vessels (blue arrow) and the horizontal cells appear normal and unremarkable. Group B. Photomicrograph of the cerebral cortex of the stroke induced and evaluation. The section of the cerebral cortex shows leptomeninges (red arrow), cortical infarct with gliosis and hyperplastic state of capillary endothelium (yellow arrow), vascular congestion (blue arrow), perivascular cuffing (green arrow), mild tissue oedema (dark blue). Group C, photomicrograph of the cerebral cortex of the stroke induced and assesses recovery. The section of the Cerebral cortex shows congestion meningeal vessels (red arrow), severe leptomeninges (dark blue), congestion meningeal vessels (red arrow), Asrocytosis (blue arrow), severe cortical infarct with gliosis and hyperplastic state of capillary endothelium (yellow arrow). Group D, photomicrograph of the cerebral cortex of the treated group with low dose of KS; the section shows meninges intact, with moderate gliosis. Group E, photomicrograph of the cerebral cortex of the treated group with high dose KS; the section shows meninges intact, with moderate gliosis.

DISCUSSION

A search for permanent treatment of stroke reminds a major challenge in neurodegenerative research. *Khaya Senegalensis* bark stem extract have been reported to have free radical scavenger activities, thereby its usefulness in chemopreventing diseases like neurodegenerative diseases has been demonstrated.[20,31,32] The present study found that animals (groups B and C) that were induced with stroke shows a significant increase in malondialdehyde (MDA) concentration compared to control (group I), revealing increase in lipid peroxidation. The finding in this present study is consistent with the report by İÇME *et al*.,[33] who reported in their study that increase in the levels of MDA as main byproducts of lipid peroxidation, is highly associated with stroke. Our study further shown a significant decrease in superoxide dismutase (SOD) levels in groups II and III compared to groups I. This finding agreed with the reports of studies similar to the present study.[33-36]

The animals treated with low and high dose of ethanolic stem bark extract of *khaya senegalenesis,* in this present study shows a decrease in the MDA concentration and a significant increase in SOD and GSH levels (figure 2). This in lined with reports by Mandel *et al*.,[20] Atawodi *et al*.,[32]; Kolawole *et al*.,[15] who reported in their studies that *Khaya Senegalensis* bark stem extract possess free radical scavenger activities.

Our findings in this study show a significant decrease in serum sodium and potassium in stroke induce animals (group B) when compared with the control. The result of this study is in agreement with other findings.[36-40] which reported that low level of sodium and potassium (hyponatremia and hypokalemia) is highly associated with stroke. Chlorine levels also decreased (hypochrloridemia) significantly in stroke induced animals, in this present study. In treatment group D and E (Low and High dose of *Khaya senegalenesis)*, this present study found serum sodium and chlorine to be statistically significant. Hypernatremia and hyperchrloridemia found in our study indicates that, in stroke condition (hyponatremia and hypokalemia) Khaya *senegalenesis* bark extract stimulate specific sodium transport systems in the blood brain barrier, which increase the rate of sodium influx from blood to brain, and thereby increase arterial pressure. Berghoff *et al*.,[41] and Gotoh *et al*.,[42] reported that sodium only cannot increase blood pressure in the absence of chlorine. In this study sodium levels increase in concomitant with chlorine. Potassium levels decreases significantly in the treatment group E (High dose of *Khaya senegalenesis*) animals. This decrease in potassium levels may have shown it to as a vasodilator agent. It therefore means administering *Khaya senegalenesis* at a high dose, it has the ability to dilate the arteries and cause blood flow, in stroke condition. Studies by Myles *et al*.,[44]; Shin *et al*.,[45]; Strong *et al*.,[46] have reported that increase in potassium levels is associated with vasoconstriction in cardiovascular accident. Serum chlorine levels significantly increase in the treatment group E (High dose of *Khaya senegalenesis*) animals. This increase in chlorine levels is found to have corroborative association with potassium in enhancing blood pressure. Our finding is in lined with findings by Overlack *et al*.,[47] who reported that in hypertensive humans, the reduction of blood pressure by dietary potassium is attenuated by potassium chloride compared with that of potassium citrate.

The result of this present study revealed the cerebral cortex leptomeninges, cortical infarct with gliosis and capillary hyperplasia, vascular congestion, perivascular cuffing and mild tissue edema (plate II), these features validate ischemic insult. This is in line with reports of Sicard *et al*.,[48]; Elkind,[49]; Welsh *et al*.,[50]; Calloni *et al*.,[25]; Peter,[52] and Wyde *et al*.,[53]. Cortical infarct with gliosis and hyperplastic state of capillary endothelium were highly prominent in the findings of the present study. This study also found fewer glial cells accumulating around blood vessels (perivascular cuffing) and Mild tissue edema or hyperemia within the perivascular cuffing. These findings are comparable with other studies[45,51,52].

Stroke induced animals and assesses recovery (Group C), in this our present study, had showed peak infiltration, congestion meningeal vessels, severe leptomeninges, reactive Astrocytes, severe cortical infarct with gliosis and hyperplastic state of capillary endothelium. This is consistent with other researchers [44,49,50,54,59]. Reactive Astrocytes were also found in this Group C wistar rats, exhibiting stellate morphology

which showed ischemic insult over a period of time While presenting infiltrates and congested meningeal vessels within the meninges and severe cortical infarct with gliosis and hyperplastic state of capillary endothelium were found in this study. These findings are comparable with the study of Ding,[60].

The treatment groups, this present study showed a minimal cellular proliferation and moderate gliosis in Groups D and E, treated animals. There was a neurorepair in the neurovascular unit and glail cells, in the animals administered with low and high dose of *Khaya senegalensis* compared with ischemia groups (Group B & C). A significant improvements in neurovascular unit and glail cells were found in the treated animals in Group E compared to Group D treated animals. When compared these findings with the positive control animals (Group A), we found a marked recovery in neurovascular unit. Findings in this present study are in line with others researchers.[15,20,31,32] Our study further indicates that ethanol stem bark extract of *Khaya senegalensis* has the ability to ameliorate inflammatory responses and also suppresses its activities. This is similar with the study of Kolawole *et al*.,[15] in their studies of Evaluation of Anti-inflammatory and Antinociceptive Potentials of *Khaya senegalensis* Stem Bark Aqueous Extract, who reported that *Khaya senegalensis* Stem Bark Aqueous extract, have anti -inflammatory effects. We thereby deduct in this present study that ethanol stem bark extract of *Khaya senegalensis* may work favorably like others anti- inflammatory drugs.

This study has validated induction of ischemic stroke (Group B & C), which shown multiple evidences of ischemia with infiltration and proliferation of immune cells, hyperplastic state of capillary endothelium. These ischemic insults were indication that there was an attempt for immune cells to eliminate cellular debris and pathogens and thereby increases the leukocytes aggregate and adhere to the vascular endothelium. In the treated animals, Groups D and E had shown minimal cellular proliferation and a neurorepair in neurovascular unit which showed that ethanol stem bark extract of *Khaya senegalensis* may possesses inhibitory properties by preventing further leukocytes migration and infiltration of hematogenous cells. This is in lined with others studies reported by Torres *et al*.,[54]; Wu *et al*.,[55]; Atawodi *et al*.,[32]; Knights *et al*,[56]; Olurishe *et al*.,[57]; Kolawole *et al*., [15]. The inhibitory properties of stem bark extract of *Khaya senegalensis*, observed in this present study may have attributed with its properties to inhibit cyclooxygenase (COX) enzymes which is a key enzyme in prostaglandin biosynthesis, catalyses oxygenation of arachidonic acid to prostaglandin G2 (PGG2) and reduction of PGG2 to Prostaglandin H2 (PGH2), an immediate precursor for production of eicosanoids. Our finding is in agreement with Park *et al*.,[31] Atawodi *et al*., [32] Knights *et al*,[56] ; Olurishe *et al*.,[57] whose studies reported similar findings with the present study.

CONCLUSION

In conclusion, a dose of 250 mg/kg body weight of *Khaya senegalenesis* ethanol stem bark extract has been shown to ameliorate induced degenerative changes in the cerebral cortex causes by stroke-induced adult wistar rats.

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