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Khaya senegalensis FLAVONOID MITIGATE ALUMINUM CHLORIDE-INDUCED ISCHEMIC STROKE IN THE PARIETOTEMPORAL CORTEX IN WISTAR RATS

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Abstract

Restorative approaches to treat stoke is still the major challenges confronting stroke researches in attempt to manage and treat stroke. This study investigated ameliorative effects of *Khaya senegalensis* flavonoids on aluminum chloride-induced ischemic stroke in the parieto-temporal cortex. Thirty-five healthy adult male rats weighing 150–220g, were randomly assigned into five groups (n =6): Group 1: serve as control received 1 mg/kg of distilled water, Group 2 ($Alcl_{3\ -2Wks}$)i, received 100 mg/kg of $AlCl_3$ and 1 mg/kg of distilled water, orally for 2 weeks, sacrificed after 2wks), Group 3 ($Alcl_{3\ -2Wks}$)ii, received 100 mg/kg of $AlCl_3$ and 1 mg/kg of distilled water orally for 2 weeks (left untreated to check recovery), Group 4 ($KS_{FLAV\text{-}MD}$+ $AlCl_3$) received 100 mg/kg of $AlCl_3$ + 200 mg/kg of flavonoids orally for 2 weeks, designated as medium dose, Group 5 ($KS_{FLAV\text{-}HD}$ + $AlCl_3$) received 100 mg/kg of $AlCl_3$ + 300 mg/kg of flavonoids orally for 2 weeks, designated as High dose. After the administration, the animal sacrifice was done via cervical dislocation. Brain tissues were carefully harvested for homogenation assay while some were fixed in 10% neutral buffered formalin, processed and stained for histological studied. Results showed normal histoarchitecture of the cortical cells in group 1 (control) while the $Alcl_3$ treated group for 2 weeks and the group left untreated, revealed mild and marked distortion and cortical cells degeneration respectively with a significant ($p < 0.05$) depleted antioxidant enzymes (SOD, GSH, catalase) and elevated lipid peroxidation, MDA were observed compared to control. However, when treated with 200 mg/kg of KS $_{flavonoid}$ (($KS_{FLAV\text{-}Medium\ dose}$) and 300 mg/kg of KS $_{flavonoid}$ ($KS_{FLAV\text{-}\ High\ dose}$) the results showed graded morphological neurorestorative process of the cortical cells, from minimal to normal orientation of the cortical cells and significantly restored antioxidant levels and reduced elevated lipid peroxidation by $AlCl_3$. Findings suggest that *Khaya senegalensis* flavonoids possess neurorestorative and anti-oxidant activities, which could be of potential benefit in the treatment and management of ischemic stroke.

Keywords: Ischemic Stroke, Parietotemporal Cortex, Aluminum Chloride, Flavonoid

Introduction

Medicinal plants have historically played a pivotal role in disease prevention and management, especially in developing countries where access to conventional pharmaceuticals may be limited. The World Health Organization (WHO) estimates that over 80% of the global population relies on traditional plant-based medicines for primary healthcare (World Health

Organization, 2013). This widespread use is attributed to the affordability, accessibility, and cultural acceptability of herbal remedies. Furthermore, the resurgence of interest in phytomedicine across modern science is driven by growing evidence that plant-based compounds possess a broad spectrum of pharmacological activities, including antioxidant, anti-inflammatory, antimicrobial, hepatoprotective, and neuroprotective effects (Cowan, 1999; Rates, 2001).

Khaya senegalensis, commonly known as African mahogany, is one such medicinal plant with significant pharmacological potential. Belonging to the family Meliaceae, this tree is widely distributed across tropical Africa, especially in West African countries like Nigeria, Senegal, Ghana, and Mali. Traditionally, the bark and leaves of *K. senegalensis* have been employed in the treatment of fever, malaria, gastrointestinal disorders, inflammation, and liver diseases (Okoye *et al.,* 2010). Phytochemical screening of the plant has revealed the presence of several bioactive constituents including flavonoids, tannins, alkaloids, saponins, steroids, triterpenoids, and limonoids—all of which contribute to its therapeutic properties (Akinmoladun *et al.,* 2007; Oboh *et al.,* 2016).

The neuroprotective potential of *Khaya senegalensis* is largely attributed to its high flavonoid content. Flavonoids have been shown to cross the blood-brain barrier, where they exert antioxidant and anti-apoptotic effects, enhance cerebral blood flow, reduce neuroinflammation, and modulate neuronal signaling cascades (Spencer, 2008). These mechanisms are vital in the context of ischemic stroke, where oxidative stress and inflammatory processes contribute significantly to neuronal damage. Given these properties, there is growing interest in exploring *K. senegalensis* as a candidate for neuroregenerative therapy in stroke models.

Among the classes of bioactive compounds found in plants, flavonoids are particularly significant. These polyphenolic secondary metabolites are present in almost all vascular plants and are known for their strong antioxidant properties. Flavonoids neutralize free radicals, chelate metal ions, modulate enzyme activity, and influence cellular signaling pathways (Middleton *et al.,* 2000). Their role in neuroprotection has received increasing scientific attention, as they have been shown to protect neurons from oxidative damage, inhibit neuroinflammation, and promote neurogenesis (Spencer, 2008). In light of this, medicinal plants rich in flavonoids are being evaluated as potential therapeutic agents for the management of stroke and neurodegenerative disorders such as Alzheimer's and Parkinson's diseases (Maher, 2019).

Stroke is a sudden and acute neurological event characterized by the rapid loss of brain function due to an interruption in the blood supply to the brain (Campbell *et al.,* 2019). The cessation of cerebral perfusion leads to deprivation of oxygen and glucose, which are critical for neuronal metabolism, resulting in irreversible brain tissue damage within minutes (Virani *et al.,* 2021).

Ischemic stroke is a major global health problem and ranks as the second leading cause of death and the third leading cause of disability worldwide (Feigin *et al.,* 2022). It results from an abrupt occlusion of cerebral blood vessels, leading to a reduction or complete cessation of

blood supply to parts of the brain. This disruption in cerebral circulation causes hypoxia, glucose deprivation, excitotoxicity, oxidative stress, and ultimately neuronal death (Dirnagl *et al.,* 1999). The parietotemporal cortex is one of the brain regions that is highly susceptible to ischemic damage due to its high metabolic activity and vascularization (Kumral *et al.,* 2002). Injury to this region often results in cognitive deficits, including impaired memory, visuospatial disorientation, and sensory integration dysfunctions (Corbetta & Shulman, 2002).

Although numerous pharmacological interventions exist for the management of stroke, such as thrombolytics and neuroprotective agents, their clinical utility is limited by narrow therapeutic windows, high cost, and adverse side effects (Powers *et al.,* 2018). Hence, there is a compelling need for affordable, safe, and effective alternatives—especially those derived from natural sources—that can mitigate stroke-related brain injury and enhance recovery outcomes.

In experimental models, neurotoxic agents like aluminum chloride ($AlCl_3$) are commonly used to simulate oxidative and ischemic brain injuries. Aluminum, a non-essential and neurotoxic metal, accumulates in the brain following chronic exposure and induces severe neuronal dysfunctions. Its neurotoxicity is mediated through several mechanisms including oxidative stress, mitochondrial dysfunction, inflammation, disruption of calcium homeostasis, and inhibition of essential enzymes such as acetylcholinesterase (Yokel & Florence, 2006; Exley, 2013). Studies have shown that $AlCl_3$ administration in laboratory animals leads to histopathological alterations in the cerebral cortex and hippocampus, mimicking features of stroke and neurodegeneration (Shati, 2011). As such, aluminum-induced models provide a useful platform for evaluating the efficacy of neuroprotective agents.

Given the known neurotoxic effects of aluminum Yokel & Florence, 2006; Exley, 2013and the limited efficacy of conventional stroke therapies, it is both timely and necessary to explore alternative interventions. This research, therefore, seeks to evaluate the ameliorative effects of *Khaya senegalensis* flavonoids on aluminum chloride-induced ischemic stroke in the parietotemporal cortex of Wistar rats, using a combination of histological and biochemical techniques. The study aims to provide scientific insight into the potential role of *K. senegalensis* in neuroprotection, and by extension, contribute to the development of plant-based interventions for stroke management.

Materials and Method

Plant material

Fresh bark of *Khaya senegalensis* was gotten from a Forest at Ukele community of Cross River State. The tree bark was identified and authenticated in the Department of Botany, University of Lagos, Nigeria and stored in the herbarium; LUH 8004 - *Khaya senegalensis* (KS).

Animals

All protocols and treatment procedures of the experiment were approved by the Federal University Wukari Research Ethics Committee of the College of Health Sciences with protocol number FUW/CHS/HREC/JUNE/2025/015/VOL1. Total of 35 male Wistar rats weighing

150 - 220 g, were obtained from the animal house of College of Health Sciences, Federal University Wukari and were kept in well-ventilated plastic cages, kept and maintained under standard laboratory conditions in the animal house of the department of Anatomy of the faculty of Basic Medical Sciences of the Federal University Wukar. They were allowed free access to food and tap water ad libitum.

Materials and chemicals used

Cages, surgical gloves, cotton wool, face masks, spatula, oral cannula, feeding plates, distilled water, sensitive weighing balance, and web cam, syringe (1ml, 2ml, 5ml, and 10ml), feed, towel, mortar and pestle, normal saline, 10% formal-saline solution, phosphate-buffered saline (PBS), sample bottles, dissecting set.

Chemicals used

Aluminum chloride (AlCl3) was obtained from the Histology laboratory of Department of Human Anatomy, Federal University Wukari, Wukari, Taraba state, Nigeria.

Extracts Preparation

Fresh bark of *Khaya senegalensis* was well cleansed and diced into smaller pieces using a sterile knife to aid the drying process and after which they were air dried at room temperature for a period of four weeks. The stem bark was then oven dried at 50c for 3hrs and thereafter crushed into semi powder using a grinding machine. 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 and the dried extract of *Khaya senegalensis* yielded 221g, was collected and preserved at 4℃ for further use. And extraction of flavonoids was carried out according to Ushie *et al.***,** 2022.

Dose Preparation of $Alcl_3$

The experimental rat's weights were measured and an average of 165.2g was taken. The dose of induction of the rats was calculated using the average weight of the experimental rats, the concentration of aluminum chloride dissolved in water.

Phytochemical Screening of *Khaya Senegalensis* bark

The phytochemical screening of the ethanolic extract was investigated using standard qualitative procedures (Trease& Evans, 1989; Sofowara, 1993) as reported by Kumar *et al.*, 2015; Lukpata *et al.*, 2020.

Experimental Design and Treatment

A total number of thirty - five healthy adults male wistar rats weighing 150 - 220 g, were used for this experiment and they were grouped into five groups (n = 6). The experiment was divided into 2 phases. Phase I: induction of experimental ischemic stroke, $Alcl_3$ was administered using a single dose of 0.5 mg/kg of $Alcl_3$ for 14 days and a control (CTL) group received 0.5 ML of normal saline for 28 days. Phase II: treatment groups, after induction of experimental stroke, the rats were divided into 3 subgroups; ($Alcl_3$ group (ischemic stroke rats), which was left untreated to check recovery, and two treatment groups, $KS_{FLAV\text{-}MD}$ + $AlCl_3$ and $KS_{FLAV\text{-}HD}$ + $AlCl_3$ groups) received 200 mg/kg and 300 mg/kg as medium and high doses respectively of KS_{FLAV}. Choice of dose selection was based on previous studies by Nwosu *et al.*, (2012), who reported that the LD50 of the extract is greater than 3000mg/kg body weight.

Induction of Experimental Ischemic Stroke

$AlCl_3$ at a single dose of 0.5 mg/100g body weight was administered daily for 14 days. $AlCl_3$ was dissolved in 150 ML of water and dose was calculated by simple proportion based on animal weight and administered via oral route with the use of a metal oropharyngeal can nula. Close daily food and water monitoring was done after $AlCl_3$ administration.

Dose treatment

Group 1: Control (received distilled water).

Group 2: $Alcl_{3\ -2Wks(i)}$ received 100 mg/kg body weight of $AlCl_3$ orally for 2 weeks, sacrificed after 2wks to evaluate ischemic stroke.

Group 3: $Alcl_{3\ -2Wks(ii)}$ (received 100 mg/kg body weight of $AlCl_3$ orally for 2 weeks, left untreated to check recovery).

Group 4: $KS_{FLAV\text{-}MD}$ + $AlCl_3$ **(**received 100 mg/kg body weight of $AlCl_3$ + 200 mg/kg of flavonoids orally for 2 weeks, designated as medium dose.

Group 5: $KS_{FLAV\text{-}HD}$ + $AlCl_3$ **(**received 100 mg/kg body weight of $AlCl_3$ + 300 mg/kg of flavonoids orally for 2 weeks, designated as High dose.

Acute Toxicity Test

The acute toxicity effect of the extract was determined using the fixed dose protocol of the Organization of Economic Co-operation and Development (OECD) guidelines for testing of chemicals, TG420 (OECD & Staff, 2001) for oral administration.

Doses of *khaya senegalensis* flavonoid extract and Aluminum Chloride ($AlCl_3$) was determined based on prior toxicity and efficacy studies, which ensured the safety and pharmacological relevance of treatment regimens. (Agyare *et al.* 2013). In the case of aluminum chloride, acute toxicity studies in rodents have shown that the oral LD_{50} is approximately 3,311–3,470 mg/kg body weight in rats, indicating that it has moderate acute toxicity when ingested (Fisher Scientific, 2020; Ricca Chemical, 2025; Sigma-Aldrich, 2024; Spectrum Chemical, 2020).

Conversely, acute toxicity studies on the aqueous stem bark extract of Khaya senegalensis reveal an LD_{50} greater than 5,000 mg/kg body weight in rats, with no significant adverse clinical or histopathological changes, suggesting that the bark extract is relatively safe at therapeutic doses (Akintola *et al.*, 2013)

Dose Standardization

For the treatment group; each wistar rat was treated with *Khaya senegalensis* flavonoid, group 4; medium dose of khaya senegalensis, group 5; high dose of *khaya senegalensis* flavonoid, received 200mg/kg and 300mg/kg per body weight as medium and high doses of *Khaya senegalensis* once a day for a period of fourteen (14) days.

Animal Sacrifice

The animal sacrifice was done via cervical dislocation. Subsequently, the skull was opened to harvest the whole brain which was carefully divided into two through the longitudinal fissure one part was fixed in the perfusion fixative (10% formal saline) and the other part was homogenized with mechanical homogenizer in a Phosphate buffer saline solution and collected in a plain tube. Subsequently the homogenized brain tissue was centrifuged at 3500rpm to separate debris or insoluble materials, the resultant supernatant was taken for biochemical analysis. Afterwards, for routine histological processing was carried out in the Neuro-Lab, Akure Ondo state Nigeria, while biochemical analysis was done at Lively Stone Lab, Port-Harcourt Rivers state Nigeria. After staining with H and E microscopic slides where viewed under the microscope using X400 and photomicrographs where taken using MD900 Amscope® digital camera.

Results

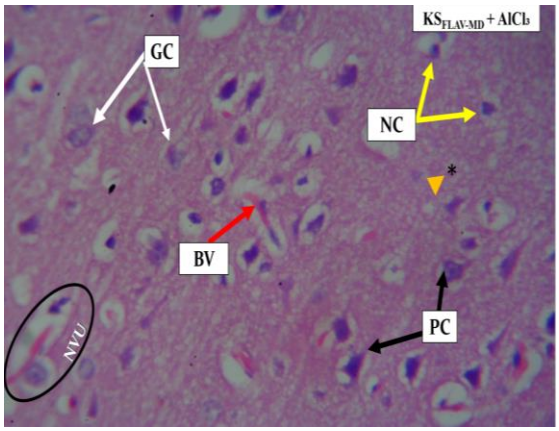
Biochemical assay: in our study the control group demonstrated normal antioxidant balance with significantly higher activities of superoxide dismutase (SOD), glutathione (GSH), and catalase, alongside a low malondialdehyde (MDA) level. In contrast, the result of the Oxidative Stress marker revealed a significantly decrease ($p<0.05$) in antioxidant activities; superoxide dismutase (SOD), glutathione (GSH), and catalase (CAT) and significantly ($p<0.05$) increase in MDA level in the group that received 100 mg/kg body weight of $AlCl_3$ orally for 2 weeks, sacrificed after 2wks ($Alcl_{3\ -2Wks(i)}$). In $Alcl_{3\ -2Wks(ii)}$ group which received 100 mg/kg body weight of $AlCl_3$ orally for 2 weeks, and left untreated for 2 weeks to check recovery revealed that after 2 weeks, there was partial improvement in SOD and MDA values, although catalase remained severely diminished, indicating sustained oxidative burden and potential compensatory mechanisms. The administration of *Khaya senegalensis* flavonoids in $AlCl_3$-treated rats produced notable ameliorative effects. At medium dose, there was a mild increase in SOD and catalase compared to the $AlCl_3$ groups, and MDA levels were reduced, suggesting a degree of neuroregeneration. On treatment with $KS_{FLAV\text{-}HD}$ shows a significant improvement in antioxidant enzyme activities and a further reduction in MDA, reflecting more restorative effect.

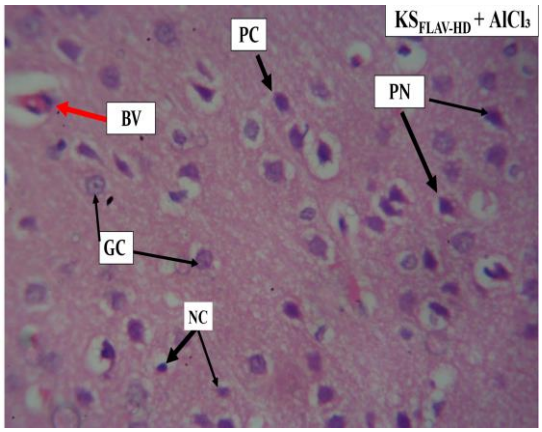
Table 1: Effect administration aluminium chloride and *Khaya senegalenesis* flavonoid on Oxidative Stress marker

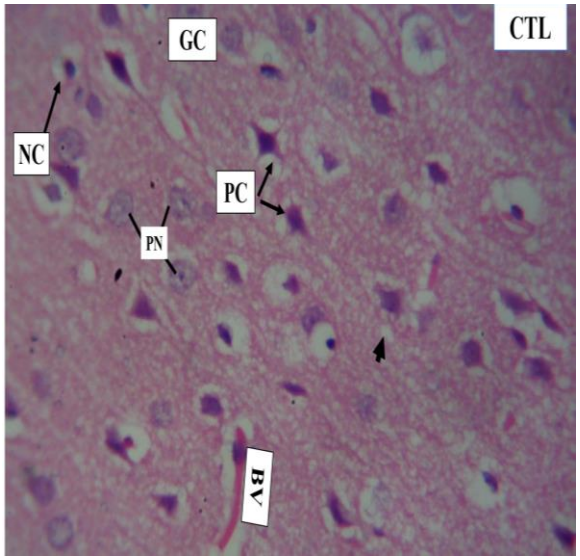
Groups	SOD(U/ml)	GSH (mmol)	MDA (umol)	Catalase (u/ml)
Control	4.11±0.12	1.79±0.25 [a]	0.86±0.12 [a]	198.99±47.09 [a]
$Alcl_{3\ -2Wks(i)}$	0.27±0.03*	0.18±0.04*	5.00±0.27*	56.92±7.43*
$Alcl_{3\ -2Wks(ii)}$	1.28±0.01*[a]	0.44±0.04*	1.27±0.18 [a]	12.21±0.01*
$KS_{FLAV-MD} + AlCl_3$	1.03±0.02*[a]	0.35±0.01*	1.22±0.01 [a]	8.76±0.04*
$KS_{FLAV-HD} + AlCl_3$	0.83±0.02*[a]	0.50±0.03*	1.27±0.12[a]	4.84±0.02*

n=3; mean ± SEM, one-way ANOVA, *= $p<0.05$; significance difference when compared to control; a=$p<0.05$ when compared to $AlCl_3$ (2Wks).

Histological studies: In fig Figure 1, A photomicrograph of a section in the parieto-temporal cortex), Control (CTL)received distilled water, showing a normal architecture of cerebral cortex, featuring the pyramidal cell, Neuroglial cell, With dense nuclei granule cell, With pale open face nucleus. $Alcl_3$ treated group which received 100 mg/kg body weight of $AlCl_3$ orally for 2 weeks, sacrificed after 2wks ($Alcl_{3\ -2Wks(i)}$), revealed cortical cells degeneration characterized by astrocytosis, Lymphocytes Infiltrates and Vacuolated Neutrophils, pyramidal cell with irregular shape and surrounded by pericellular halos, Perivascular Edema, Perivascular Cuffing and shrunken Granule cells deeply stained. $Alcl_{3\ -2Wks(ii)}$ group which received 100 mg/kg body weight of $AlCl_3$ orally for 2 weeks, and left untreated for 2 weeks to check recovery, showed marked cortical cells degeneration characterized by astrocytosis, degenerate pyramidal neuron with irregular shape and surrounded by pericellular halos, Perivascular Edema, Perivascular Cuffing and shrunken Granule cells deeply stained. $KS_{FLAV-MD} + AlCl_3$ treated rats showed less or normal neurovascular unit, Pyramidal cell, Neuroglial cells and Neutrophils vacuolated, Granule cells. Pyramidal cell, Neuroglial cells and, Granule cells (GC) visibly showed open face nuclei and basophil cytoplasm, prominent nucleoli. $KS_{FLAV-HD} + AlCl_3$ treated rats. Showing more prominent orientation of blood vessels, Pyramidal cell, Neuroglial cells and Neutrophils vacuolated, Granule cells. Pyramidal cell, Neuroglial cells darkly stained and Granule cells visibly showing open face nuclei and basophil cytoplasm, prominent nucleoli.


KS$_{FLAV-MD}$ + AlCl$_3$
GC
NC
BV
PC
NVU


KS$_{FLAV-HD}$ + AlCl$_3$
PC
BV
PN
GC
NC


CTL
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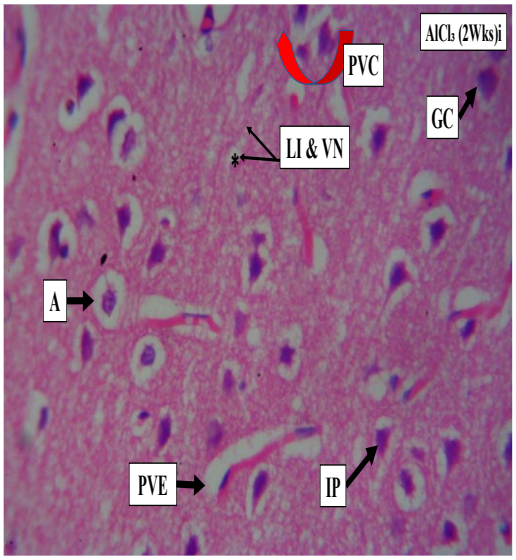
AlCl$_3$ (2Wks)i
PVC
GC
LI & VN
A
PVE
IP

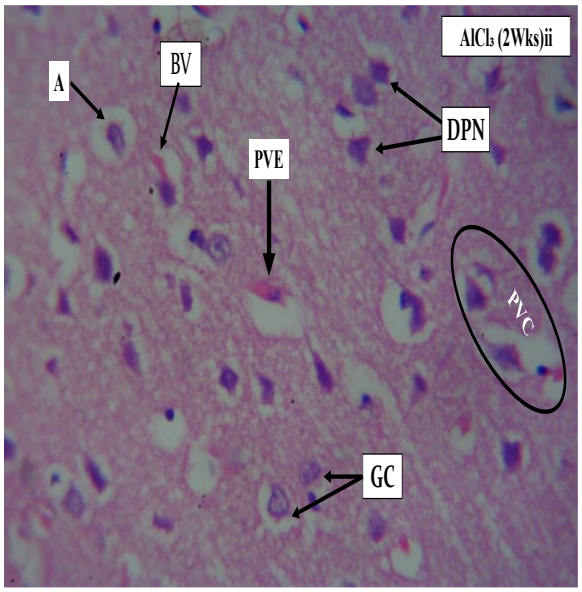
AlCl$_3$ (2Wks)ii
A
BV
DPN
PVE
PVC
GC

Fig 1: A photomicrograph of a section in the parieto-temporal cortex of male Wister rats. Control (CTL) showing normal architecture of cerebral cortex, pyramidal cell (P), Neuroglial cell (N), With dense nuclei granule cell (G), Alcl3 -2Wks(i) showing mild cortical cells degeneration characterized by astrocytosis (A), Lymphocytes Infiltrates (LI) and Vacuolated Neutrophils (VN), pyramidal cell with irregular shape (IP) and surrounded by pericellular halos, Perivascular Edema (PVE), Perivascular Cuffing (PVC) and shrunken Granule cells (GC). Alcl3 -2Wks(ii) showing marked cortical cells degeneration characterized by astrocytosis(A), degenerate pyramidal neuron with irregular shape (DPN) and surrounded by pericellular halos, Perivascular Edema (PVE), Perivascular Cuffing (PVC) and shrunken Granule cells (GC). KSFLAV-MD + $AlCl_3$ treated rats,Showing less or normal neurovascular unit (NVN), Pyramidal cell (PC), Neuroglial cells (NC) and Neutrophils (N) vacuolated, Granule cells (GC). Pyramidal cell (PC), Neuroglial cells (NC) and, Granule cells (GC) visibly showing open face nuclei and basophil cytoplasm, prominent nucleoli. KSFLAV-HD + $AlCl_3$ treated rats. Showing more prominent orientation of blood vessels (BV), Pyramidal cell (PC), Neuroglial cells (NC) and Neutrophils (N) vacuolated, Granule cells (GC). Pyramidal cell (PC), Neuroglial cells (NC) darkly stained and Granule cells (GC) visibly showing open face nuclei and basophil cytoplasm, prominent nucleoli. (H & E X400)

Discussion

In the past years, Cerebrovascular accident (CVA) is considered to be the third most common cause of mortality in the developed world. Current treatments such as tissue plasminogen activator (tPA/Alteplase) is the gold standard for acute ischemic stroke if administered within 4.5 hours, but its effectiveness declines rapidly with delayed administration, with high risk of intracerebral hemorrhage (about 6%), not to mention systemic bleeding and other complications (Hacke *et al.*, 2008).

In our present study, the biochemical analysis of oxidative stress biomarkers is presented in Table 1. The control group demonstrated normal antioxidant balance with significantly higher activities of superoxide dismutase (SOD), glutathione (GSH), and catalase, alongside a low malondialdehyde (MDA) level, reflecting a healthy redox state. This finding aligns with earlier reports that physiological antioxidant systems maintain cellular homeostasis under normal conditions (Halliwell & Gutteridge, 2015). In contrast, aluminum chloride ($AlCl_3$) administration for two weeks caused a marked depletion of antioxidant defenses, as indicated by significantly decreased SOD, GSH, and catalase levels, while MDA was drastically elevated compared to the control ($p < 0.05$). This observation is consistent with previous studies that demonstrated $AlCl_3$-induced oxidative damage in neuronal tissues through lipid peroxidation and suppression of endogenous antioxidants (Ebhodaghe *et al.,* 2019; Suryavanshi et al., 2022). In $Alcl_{3\text{ -2Wks(ii)}}$ group which received 100 mg/kg body weight of $AlCl_3$ orally for 2 weeks, and left untreated for 2 weeks to check recovery, there was partial improvement in SOD and MDA values, although catalase remained severely diminished, indicating sustained oxidative burden and potential compensatory mechanisms. The administration of Khaya senegalensis flavonoids in $AlCl_3$-

treated rats produced notable ameliorative effects. At medium dose, there was a modest increase in SOD and catalase compared to the $AlCl_3$ groups, and MDA levels were reduced, suggesting a degree of neuroprotection. At high dose, flavonoid treatment resulted in greater improvements in antioxidant enzyme activities and a further reduction in MDA, reflecting a stronger restorative effect. These findings corroborate earlier reports that flavonoids exert antioxidant and neuroprotective effects by scavenging free radicals, enhancing endogenous enzymatic defenses, and reducing lipid peroxidation (Spencer, 2008; Oyelami *et al.*, 2018). Specifically, extracts of Khaya senegalensis have been documented to contain polyphenolic compounds with potent antioxidant capacity, which contribute to protection against chemically induced oxidative insults (Abdullahi *et al.,* 2019; Ibrahim *et al.,* 2021).

The present study histological findings of parieto-temporal cortex in group 1 (control) showed normal histoarchitecture of the cortex with normal pyramidal cell, neurovascular unit, neuron, blood vessel, neuroglial cell with dense nuclei, granule cell, pale open face nucleus and blood vessels. This is in line with the work done by Ebhodaghe & Enogieru, 2024.
Also, in the $Alcl_3$ treated groups for 2 weeks, showed mild and marked cortical cells degeneration characterized by astrocytosis, Lymphocytes Infiltrates and Vacuolated Neutrophils, pyramidal cell with irregular shape and surrounded by pericellular halos, Perivascular Edema, Perivascular Cuffing and shrunken Granule cells deeply stained. This suggest that there was neurodegeneration. The result of our present is consistent with the study Okhah and Enogieru (2023) who observed that, atrophy and vacuolation of astrocytes and pyramidal cells were seen.

On treatment, it was observed in this study that, the parieto-temporal cortex of $Alcl_3$ and KS_{FLAV} treated rats ($KS_{FLAV\text{-}MD}$ + $AlCl_3$) unveils less or normal neurovascular unit, Pyramidal cell, Neuroglial cells and Neutrophils vacuolated, Granule cells. Pyramidal cell, Neuroglial cells and, Granule cells (GC) visibly showed open face nuclei and basophil cytoplasm, prominent nucleoli. This suggests that low dose (200 mg/kg) of *khaya Senegalensis*and have low ameliorative effect on aluminum chloride ($AlCl_3$)-induced stroke. This study correlates with Onu *et al.,* (2013) who also observed that for *khaya Senegalensis* is dose dependent.

In addition,on treatment with 300 mg/kg of *khaya senegalensis* on aluminum chloride ($AlCl_3$)-induced stroke in the parieto-temporal cortex of adult wistar rats ($KS_{FLAV\text{-}HD}$ + $AlCl_3$). It reveals the ameliorative effects of *khaya Senegalensis* aluminum chloride ($AlCl_3$)-induced stroke which fully unveils the in the parieto-temporal cortex of $KS_{FLAV\text{-}HD}$ + $AlCl_3$ treated rats. Showing more prominent orientation of blood vessels, Pyramidal cell, Neuroglial cells and Neutrophils vacuolated, Granule cells. Pyramidal cell, Neuroglial cells darkly stained and Granule cells visibly showing open face nuclei and basophil cytoplasm, prominent nucleoli.

This result implies that *khaya Senegalensis* 300 mg/kg have the efficacy to curtail and eradicate the toxic effect of induced stroke by aluminum chloride. This is in line with Onu *et al.,* (2013) who suggest that the aqueous stem bark extract of K. senegalensis may affect the cellular integrity of vital organs of the body.

Conclusion

In this our study, and from the results obtained above, *Ks*FLAV have the efficacy and potency to ameliorate aluminum chloride ($AlCl_3$)-induced ischemic stroke on the parieto-temporal cortex. The result showed that at 100 mg/kg of aluminum chloride, stroke can be induced in fourteen days.

In conclusion, *Khaya senegalensis* flavonoids at dose 200 mg/kg have shown neuroregenerative and neurorepairs role on neurodegenerative changes in the parieto-temporal cortex, by regenerating pyramidal cell/granule cells and neurovascular unit.

Recommendation to Further Study

Additional studies involving different animal models and prolonged treatment periods should be conducted to establish optimal dosage, safety profiles, and possible toxicological limits before progressing to human clinical trials. Also, the compatibility of this herb and any other drug that induce stroke is recommended.

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