JIRBDAI Journal of Institutional Research, Big Data Analytics and Innovation

Histopathological Assessment of the Protective Effects of *Kigelia africana* Fruit and Leaf Aqueous Extracts on Hepatic, Renal, and Pancreatic Tissues in Streptozotocin-Induced Diabetic Rats

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ABSTRACT

Diabetes mellitus remains a major global health burden, and the associated multi-organ complications affecting the liver, kidney, and pancreas contribute significantly to morbidity and mortality. Medicinal plants, particularly *Kigelia africana*, have been traditionally used in sub-Saharan Africa for the management of diabetes and related disorders. This study evaluated the histopathological effects of aqueous fruit and leaf extracts of *K. africana* on the liver, kidney, and pancreatic tissues of streptozotocin-induced diabetic rats. Diabetes was induced in Wistar rats by a single intraperitoneal injection of streptozotocin (55 mg/kg). Animals were grouped into six groups: normal control (Group A), diabetic control (Group B), diabetic treated with glibenclamide 1000 mg/kg (Group C), diabetic treated with *K. africana* fruit extract 1000 mg/kg (Group D), diabetic treated with *K. africana* leaf extract 1000 mg/kg (Group E), and diabetic treated with combined fruit and leaf extract 1000 mg/kg (Group F). Aqueous extracts were administered orally for 28 consecutive days, and tissues were processed for haematoxylin and eosin (H&E) histopathological examination. Group B (diabetic control) showed marked degeneration including hepatocyte necrosis, renal tubular and glomerular damage, and atrophy of the islets of Langerhans. Group D (fruit extract) demonstrated near-normal histological architecture in all three organs, comparable to Group C (glibenclamide). Group F (combined extract) showed partial tissue restoration. Group E (leaf extract alone) exhibited persistent degenerative changes including necrobiotic hepatocyte alterations, renal tubular degeneration, and pancreatic islet inflammation. The aqueous fruit extract of *K. africana* exhibited significant hepatoprotective, nephroprotective, and pancreato protective effects in STZ-induced diabetic rats, supporting its traditional use as an antidiabetic agent. The leaf extract alone did not confer equivalent protection, indicating differential bioactivity between plant parts.

KEYWORDS: *Kigelia africana*; diabetes mellitus; streptozotocin; histopathology; liver; kidney; pancreas; islets of Langerhans

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https://doi.org/10.5281/zenodo.21132037

I. INTRODUCTION

Diabetes Mellitus (DM) is a chronic metabolic disorder defined by persistent hyperglycaemia resulting from defective insulin secretion, impaired insulin action, or both. According to the 11th edition of the International Diabetes Federation (IDF) Diabetes Atlas, approximately 589 million adults aged 20 to 79 years were living with diabetes in 2024, representing a global prevalence of 11.1%, with projections indicating this figure will rise to 853 million by 2050 (IDF, 2024). Alarmingly, an estimated 43% of affected individuals remain undiagnosed, and the economic burden of diabetes associated health expenditure reached approximately USD 1 trillion in 2024 (IDF, 2024). The majority of the global burden is concentrated in low- and middle-income countries, where health system capacity is most constrained. Chronic hyperglycaemia in diabetes is associated with severe multi-organ complications. Progressive damage to the liver, kidney, and pancreas constitutes a particularly critical dimension of diabetic pathology. Hepatic dysfunction in diabetes arises from fatty infiltration, hepatocyte degeneration, and impaired glucose metabolism. Diabetic nephropathy, characterized by glomerular damage, tubular atrophy, and proteinuria, remains a leading cause of end-stage renal disease worldwide. The pancreas, as the site of insulin production within the islets of Langerhans, undergoes progressive beta-cell loss, further perpetuating metabolic dysregulation (Lenzen, 2008; Szkudelski, 2001).

Current pharmacological management of diabetes, including sulfonylureas such as glibenclamide, while effective, is associated with adverse effects including hypoglycaemia and gastrointestinal intolerance (Groop & Neugebauer, 1996). The growing global interest in medicinal plants as adjunct or alternative therapies has led to systematic investigations of botanicals with demonstrated hypoglycaemic and organ-protective properties. Previous work from our group has demonstrated the utility of natural product-based interventions in modulating haematological and histological parameters in experimental rodent models (Eli et al., 2024; Luka et al., 2017), providing a framework within which the present study is situated. *Kigelia africana* (Lam.) Benth., belonging to the family Bignoniaceae and commonly referred to as the sausage tree, has a pan-African distribution and has been extensively used in traditional medicine across sub-Saharan Africa for the treatment of diabetes mellitus, skin disorders, and inflammatory conditions (Bello et al., 2016; Atawodi & Olowoniyi, 2014). Phytochemical investigations have revealed the presence of iridoids, flavonoids, naphthoquinones, sterols, phenylpropanoids, and phenolic compounds, which are believed to underlie its diverse pharmacological activities (Bello et al., 2016). Previous studies have demonstrated the blood glucose-lowering capacity of *K. africana* extracts in both alloxan and streptozotocin (STZ)-induced rodent models (Muyenga et al., 2015; Fagbohun et al., 2020; Abdu et al., 2020). However, the comparative organ-protective effects of its constituent parts, notably the fruit versus the leaf, have not been systematically characterised using histopathological methodology. This study therefore investigated the histopathological effects of separately prepared aqueous extracts of the fruit and leaf of *K. africana*, as well as their combination, on the liver, kidney, and pancreas of STZ-induced diabetic rats, with glibenclamide serving as the pharmacological reference standard.

II. RELATED WORKS

Kigelia africana has attracted increasing scientific attention as a multi-target medicinal plant. Bello et al. (2016) conducted a comprehensive phytochemical and pharmacological review documenting extensive traditional use for diabetes management and the presence of antioxidant secondary metabolites that could plausibly modulate oxidative stress-driven diabetic complications. Atawodi and Olowoniyi (2014) similarly confirmed the antidiabetic,

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hepatoprotective, and anti-inflammatory properties of *K. africana* through multiple experimental systems. Building on ethnobotanical evidence, Muyenga et al. (2015) demonstrated the blood glucose-lowering effect of crude aqueous *K. africana* fruit extracts in alloxan-induced diabetic mice. Fagbohun et al. (2020) subsequently investigated the hexane fraction of *K. africana* fruit extract in STZ induced diabetic Wistar rats and reported significant reductions in fasting blood glucose alongside ameliorative effects on haematological parameters and histological regeneration in the renal cortex and liver. Abdu et al. (2020) further demonstrated hepatoprotective and nephroprotective biochemical effects of *K. africana* stem bark extract in alloxan-induced diabetic rats, reinforcing the multi-organ protective potential of the plant.

The relevance of plant-based extract interventions in the modulation of haematological and histological parameters in diabetic rodent models is also supported by broader experimental evidence. Eli et al. (2024) demonstrated significant effects on haematological and histological parameters in albino rats administered formulations derived from malted and fermented cereal flours supplemented with legumes, underscoring the capacity of natural product interventions to exert measurable histological effects in experimental models. Similarly, Luka et al. (2017) reported biochemical amelioration in alloxan-induced diabetic rats treated with aqueous seed extract of *Terminalia catappa*, providing additional precedent for aqueous plant extract use in experimental diabetes models. Streptozotocin is among the most widely used agents for inducing experimental diabetes in rodents because of its selective cytotoxicity to pancreatic beta cells via GLUT2-mediated uptake, DNA alkylation, and subsequent reactive oxygen species generation (Szkudelski, 2001; Lenzen, 2008). The STZ model closely recapitulates beta-cell destruction and ensuing organ complications observed in clinical Type 1 diabetes, making it appropriate for evaluating putative organ protective plant extracts.

More recent evidence continues to substantiate the antidiabetic and organ-protective potential of K. africana and related phytochemical classes. Muyenga et al. (2024) demonstrated that fractionated aqueous K. africana fruit extracts inhibit alpha-glucosidase activity in vitro, providing a plausible carbohydrate-digestive enzyme-inhibitory mechanism that complements the organ-protective effects reported here. Orabi et al. (2023) reported comparative antioxidant and cytotoxicity profiling across K. africana plant organs, confirming that different plant parts possess quantitatively distinct phytochemical and antioxidant capacities, lending independent support to the differential fruit-versus-leaf protective effects observed in the present study. Abbas et al. (2024), in a comprehensive review, catalogued over 140 bioactive compounds across K. africana plant parts and reaffirmed its antioxidant, anti-inflammatory, and antidiabetic pharmacological activities, further supporting the mechanistic plausibility of the histological protection described in this study.

III. METHODOLOGY

A. Ethical Approval

Ethical clearance for the use of laboratory animals in this study was obtained from the Ethical Committee Animal Experimental Unit, Faculty of Pharmaceutical Sciences, University of Jos, Nigeria (Reference Number: **UJ/FPS/F17-00379**, dated 02/03/2020), in compliance with the Institutional Animal Care and Use Committee (IACUC) guidelines in collaboration with the Office of Laboratory Animal Welfare (OLAW). All experimental procedures were strictly conducted in accordance with the approved protocol.

https://doi.org/10.5281/zenodo.21132037

B. Plant Material Collection and Identification

Fruits and leaves of *K. africana* were collected and authenticated by a botanist. Plant materials were washed with distilled water, shade-dried, and ground into fine powder using an electric blender.

C. Preparation of Aqueous Extracts

Distilled water (500 mL) was added separately to 100 g each of powdered *K. africana* leaf and fruit. Both mixtures were boiled separately in conical flasks for 30 minutes. After the set time, the mixtures were filtered, and the filtrate was designated as the stock solution. The crude extracts were dried in an oven at 40°C, weighed, and stored for subsequent use.

D. Experimental Animals

Adult male Wistar rats (180 to 220 g) were used for the study. Animals were housed under standard laboratory conditions with a 12-hour light/dark cycle and given free access to standard rat chow and clean drinking water.

E. Experimental Induction of Diabetes

Diabetes was induced by a single intraperitoneal injection of streptozotocin (STZ) at a dose of 55 mg/kg body weight, freshly dissolved in cold 0.1 M citrate buffer (pH 4.5). Animals were left for 72 hours, after which diabetes was confirmed using a one-touch glucometer. Animals with fasting blood glucose values equal to or greater than 200 mg/dL were considered diabetic and included in the study.

F. Experimental Design

Animals were randomly assigned to six groups of six rats each as follows:

Group A: Normal Control (received vehicle only)
Group B: Diabetic Control (no treatment)
Group C: Diabetic + Glibenclamide (1000 mg/kg)
Group D: Diabetic + K. africana fruit extract (1000 mg/kg)
Group E: Diabetic + K. africana leaf extract (1000 mg/kg)
Group F: Diabetic + K. africana combined fruit and leaf extract (1000 mg/kg)

All treatments were administered orally once daily for 28 consecutive days. After the last treatment, all animals were sacrificed by cervical decapitation and blood samples and organs were collected for analysis.

G. Histopathological Examination

Animals were sacrificed by cervical decapitation, and the liver, kidney, and pancreatic tissues were rapidly excised and washed with ice-cold 0.9% NaCl (w/v). A portion of each tissue was fixed in 10% neutral buffered formalin. After fixation, tissues were dehydrated through ascending grades of alcohol, cleared in xylene, embedded in paraffin wax, and sectioned at 5 micrometres. Sections were stained with haematoxylin and eosin (H&E) and examined under a light microscope (Eli et al., 2024; Bancroft & Layton, 2013).

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IV. RESULTS AND DISCUSSION

A. Histopathological Findings

(a). Liver (Plate 1)

Plate 1 presents the histological findings in liver tissues across all six groups. Group A (normal control) showed a normal hepatic lobular architecture with centrally located central veins, radially arranged hepatocytes, and well-defined portal triads (Plate 1A). Group B (diabetic control) exhibited severe hepatocytic degeneration, including areas of tissue necrosis and atrophy of hepatocytes, consistent with diabetic liver pathology characterized by oxidative stress and impaired hepatic function (Plate 1B). Group C (glibenclamide-treated) demonstrated nearly normal hepatocyte architecture with evidence of cellular regeneration around the portal areas (Plate 1C). Importantly, Group D (K. africana fruit extract) also showed near-normal hepatocyte morphology and marked regeneration of periportal cells (Plate 1D), a finding comparable to the standard drug group, consistent with reports by Fagbohun et al. (2020), who documented histological liver regeneration in K. africana-treated diabetic rats. In contrast, Group E (leaf extract) demonstrated necrobiotic changes in hepatocytes, proliferation of newly formed bile canaliculi at the portal area, and multiple forms of hepatocellular degeneration (Plate 1E), indicating that the aqueous leaf extract, at the tested dose, was not effective in protecting the liver from STZ-induced damage. Group F (combined extract) exhibited almost normal radially arranged hepatocytes, suggesting partial hepatoprotection attributable to the fruit component (Plate 1F).

(b). Kidney (Plate 2)

Plate 2 presents the renal histological findings. Normal glomerular and tubular architecture was observed in Group A (Plate 2A). Group B (diabetic control) showed degenerative changes in renal glomeruli and tubular epithelial cells, consistent with diabetic nephropathy (Plate 2B). These findings align with the established renal toxicity of streptozotocin (Lenzen, 2008). Group C (glibenclamide) showed improved, nearly normal renal tissue (Plate 2C). Group D (fruit extract) showed marked renal protection, with glomeruli morphologically comparable to those of the normal control (Plate 2D), consistent with the nephroprotective findings of Fagbohun et al. (2020). Group E (leaf extract) exhibited continued tubular epithelial degeneration and atrophy of the glomerular tuft (Plate 2E). Group F (combined extract) showed gradual regeneration of the glomerular tuft (Plate 2F), suggesting partial but incomplete nephroprotection.

(c). Pancreas (Plate 3)

Plate 3 illustrates the pancreatic histological findings. Group A (normal control) displayed well-preserved islets of Langerhans surrounded by normal exocrine acini (Plate 3A). Group B (diabetic control) showed marked degeneration and atrophy of the islets of Langerhans (Plate 3B), consistent with the STZ mechanism of selective beta-cell destruction via GLUT2-mediated DNA alkylation and reactive oxygen species generation (Szkudelski, 2001; Lenzen, 2008). Group C (glibenclamide) demonstrated normal islets with a normal population of beta cells (Plate 3C), reflecting the known K-ATP channel inhibitory mechanism of sulfonylureas (Groop & Neugebauer, 1996). Group D (fruit extract) showed normal-appearing islets with a beta-cell population comparable to the normal control, indicating clear protection from the damaging effects of streptozotocin (Plate 3D). Group E (leaf extract) showed inflammation of the islets and acini cells (Plate 3E), suggesting failure to protect against STZ-mediated destruction. Group F

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(combined extract) showed improved but less pronounced islet restoration compared to Group D (Plate 3F).

B. Discussion

The results demonstrate a clear and consistent pattern: the aqueous fruit extract of *K. africana* confers significant histological protection to the liver, kidney, and pancreas in STZ-induced diabetic rats, whereas the aqueous leaf extract at the equivalent dose does not. These findings are broadly consistent with the accumulating literature on *K. africana* fruit as a multi-organ protective agent in experimental diabetes (Fagbohun et al., 2020; Bello et al., 2016; Atawodi & Olowoniyi, 2014). The fruit has been reported to contain higher concentrations of naphthoquinones, iridoids, and phenolic acids than the leaf, which may collectively attenuate oxidative stress-mediated tissue damage. These observations are consistent with broader experimental evidence demonstrating the capacity of natural product-based interventions to modulate organ histology in rodent models (Eli et al., 2024; Luka et al., 2017). The performance of Group D in matching Group C (glibenclamide) histologically across all three organs is particularly encouraging. Glibenclamide, as the only oral antidiabetic drug on the WHO Essential Medicines List, represents a robust clinical benchmark, and the fruit extract's comparability to this standard supports its potential utility in the management of diabetic organ complications. The partial protection in Group F may reflect pharmacodynamic interactions between fruit and leaf bioactives, or dilution of the fruit extract's active constituents within the combined formulation. This has practical implications for the formulation of K. africana based remedies, suggesting that fruit specific preparations may be superior to whole plant combinations for organ protection in diabetes. Further phytochemical fractionation and dose-response studies are warranted. Mechanistically, the organ-protective effects observed with the fruit extract are plausibly attributable to its relatively high content of naphthoquinones, iridoids, flavonoids, and phenolic acids (Bello et al., 2016; Orabi et al., 2023). These compound classes are established in the broader phytochemistry literature as potent scavengers of reactive oxygen species and as modulators of the Nrf2-mediated antioxidant response pathway, which would be expected to limit STZ-induced lipid peroxidation and oxidative damage to hepatocyte, tubular epithelial, and beta-cell membranes (Szkudelski, 2001; Lenzen, 2008). Naphthoquinones and phenolic acids have additionally been reported to attenuate pro-inflammatory NF-κB signaling and cytokine release, which may account for the reduced inflammatory infiltration observed histologically in Group D relative to Group E (Atawodi & Olowoniyi, 2014). At the enzymatic level, the alpha-glucosidase inhibitory activity reported for K. africana fruit fractions (Muyenga et al., 2024) offers a complementary, non-histological mechanism by which the fruit extract could moderate postprandial glycaemic excursions and thereby indirectly reduce glucotoxic stress on the liver, kidney, and pancreas. The comparatively lower concentration of these protective phytochemicals reported for K. africana leaf tissue relative to fruit tissue (Orabi et al., 2023) offers a plausible phytochemical explanation for the persistent degenerative changes observed in Group E. These mechanistic pathways are inferred from the established pharmacology of the relevant phytochemical classes and from the histological pattern observed in this study; targeted biochemical assays are required to confirm them directly and are identified as a priority for future work (Section C).

https://doi.org/10.5281/zenodo.21132037

C. Study Limitations

This study was designed as a descriptive histopathological comparison of K. africana fruit and leaf extracts across six treatment groups. Three related limitations should be considered when interpreting the findings. First, histopathological assessment was qualitative rather than semi-quantitatively scored; a blinded severity-grading protocol scored independently by two histopathologists, with reported inter-rater agreement, would strengthen confidence in the reported group differences and is recommended for follow-up work. Second, the conclusions rest on histological evidence alone, without concurrent biochemical assays of hepatic (ALT, AST), renal (urea, creatinine), or oxidative-stress (malondialdehyde, superoxide dismutase, catalase) status; a biochemically integrated follow-up study is planned to corroborate the present histological findings. Third, because the histological findings reported are qualitative pattern descriptions rather than measured continuous variables, inferential statistical testing was not applied to the present dataset; where quantitative endpoints (e.g., islet counts, glomerular diameter, hepatocyte necrosis index) are introduced in future work, between-group comparisons will be analysed by one-way ANOVA with Tukey's HSD post-hoc test ($p < 0.05$). These limitations define the scope of the present descriptive study and the priorities for the quantitative, mechanistic follow-up study outlined above; they do not undermine the histological observations reported, which were consistent across three independent organ systems and were benchmarked against an active pharmacological comparator (glibenclamide, Group C).

D. Histopathological Plates

(a). Plate 1: Histological Presentation of Liver Structures

(A) Liver of rat from Group A (normal control) showing normal histological hepatic architecture. (B) Hepatic tissue of rat from Group B (diabetic control) with degenerative changes of hepatocytes, tissue necrosis, and atrophy. (C) Liver of rat from Group C showing near-normal hepatocytes and cellular regeneration around the portal area. (D) Rat from Group D showing near-normal hepatocytes and regeneration of cells around the portal area. (E) Group E rat showing necrobiotic changes of hepatocytes, proliferation of newly formed bile canaliculi at the portal area, and different forms of necrobiotic hepatocellular changes. (F) Group F rat showing almost normal radially arranged hepatocytes. (H&E, x40)

(b). Plate 2: Histological Presentation of Kidney Structures

(A) Group A (normal control) showing normal glomerular and renal tissue structure. (B) Renal tissue of Group B (diabetic control) showing degenerative changes of renal glomeruli and epithelial cells of renal tubules. (C) Group C showing improved and almost normal renal tissues. (D) Renal tissue of Group D showing marked protection against diabetic changes, with glomeruli comparable to those of the normal control. (E) Renal tissue of Group E showing degeneration of epithelial cells of renal tubules and atrophy and collapse of the endothelial cells of the glomerular tuft. (F) Renal tissue of Group F showing gradual regeneration of glomerular tuft. (H&E, x100)

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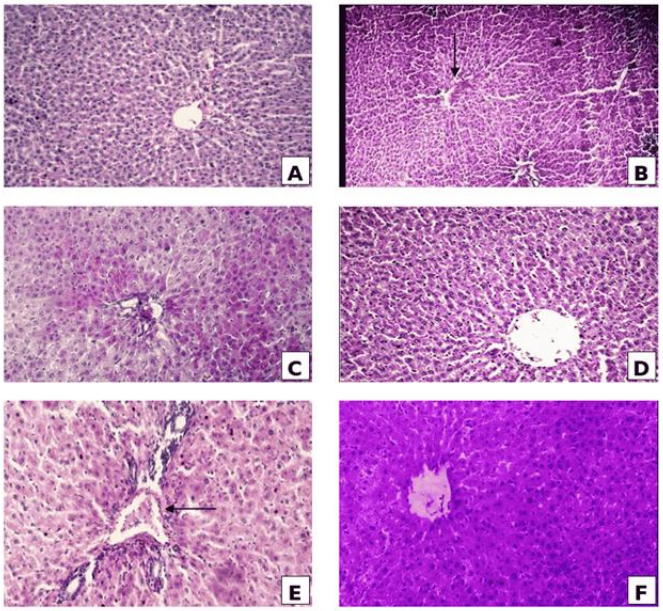

Plate 1: Histological Presentation of Liver Structures

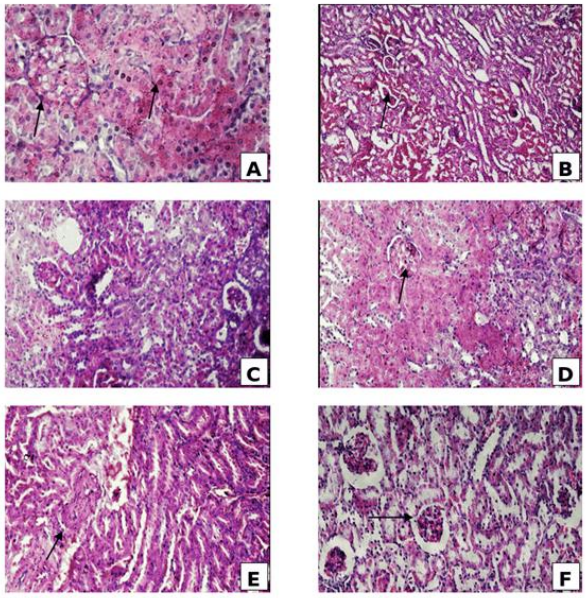

Plate 2: Histological Presentation of Kidney Structures

E. Plate 3: Histological Presentation of Pancreas Structures

(A) Group A (normal control) showing normal pancreatic structure and exocrine acini surrounding the islets of Langerhans. (B) Group B (diabetic control) showing marked degeneration and atrophy of islets of Langerhans. (C) Group C showing normal islets of Langerhans with a normal population of beta cells. (D) Group D showing normal islets of Langerhans with a beta-cell population similar to the normal control, indicating protection from the damaging effects of streptozotocin. (E) Group E showing inflammation of islets of Langerhans and acini cells. (F) Group F showing improved islets of Langerhans. (H&E, x200)

https://doi.org/10.5281/zenodo.21132037

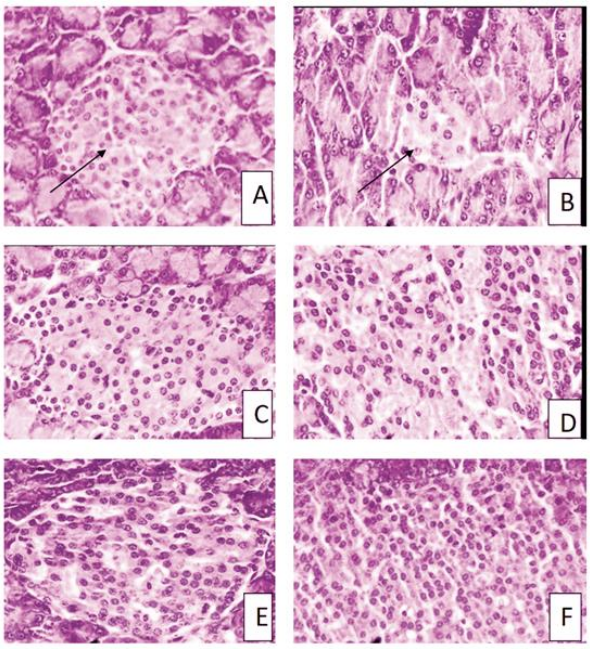

Plate 3: Histological Presentation of Pancreas Structures

V. CONCLUSION

This study provides histopathological evidence that the aqueous fruit extract of *Kigelia africana* exerts consistent, qualitatively marked protective effects on the liver, kidney, and pancreas of STZ-induced diabetic rats, with efficacy comparable to the standard antidiabetic drug glibenclamide. The fruit extract preserved hepatocyte architecture, maintained glomerular and tubular integrity, and protected the islets of Langerhans from STZ-mediated destruction. The aqueous leaf extract did not confer equivalent organ protection at the tested dose, and the combined extract produced only partial protection. These findings validate the ethnobotanical use of *K. africana* fruit in the management of diabetes and its complications. Building on the limitations outlined above, future work should prioritize isolating the specific phytochemical fractions responsible for these protective effects, establishing dose-response relationships, and pairing histological assessment with biochemical and semi-quantitative validation to fully characterise the therapeutic potential of *K. africana* fruit extract in diabetic complications.

DECLARATIONS

Ethical Approval: Ethical clearance was obtained from the Ethical Committee Animal Experimental Unit, Faculty of Pharmaceutical Sciences, University of Jos, Nigeria (Reference: UJ/FPS/F17-00379, 02/03/2020). All procedures complied with IACUC and OLAW guidelines.

Conflict of Interest: The authors declare no conflict of interest.

Funding: This research received no external funding.

https://doi.org/10.5281/zenodo.21132037

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