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Hepatoprotective Effects of *Vernonia amygdalina* Leaf Extract in Alloxan-Induced Diabetic Rats: A Histopathological Study

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ABSTRACT

Diabetes mellitus constitutes a major metabolic disorder characterised by persistent hyperglycaemia, heightened oxidative burden, and progressive impairment of vital organ function. The present investigation examined the capacity of an ethanolic leaf extract derived from *Vernonia amygdalina* to attenuate hepatic enzyme dysregulation and oxidative stress in a rodent model of alloxan-induced diabetes. A total of twenty-four male Wistar rats were assigned at random to four experimental groups (n = 6 per group): an untreated normoglycaemic group (Group I), an untreated diabetic group (Group II), and two groups administered graded doses of the ethanolic extract at 200 mg/kg (Group III) and 400 mg/kg (Group IV) respectively. Outcome measures encompassed body mass, relative liver weight, serum hepatic enzyme profiles (AST, ALT, ALP), oxidative stress indices (MDA, SOD), and microscopic evaluation of hepatic tissue architecture. Statistical comparisons employed one-way ANOVA with post-hoc Tukey's HSD test for biochemical parameters; findings were expressed as mean ± SEM and deemed statistically significant at $p \leq 0.05$. In untreated diabetic animals, significant elevations in AST, ALT, ALP, and MDA levels alongside depleted SOD activity were recorded relative to normoglycaemic controls. Microscopic examination disclosed hepatocellular degeneration, sinusoidal congestion, and inflammatory infiltration consistent with oxidative injury and lipid peroxidation. Administration of the plant extract substantially reversed these disturbances in a dose-dependent manner: serum enzyme and MDA concentrations fell markedly, SOD activity recovered, and histological sections revealed progressive mitigation of hepatic injury with increasing dose. The study concludes that ethanolic extract of *V. amygdalina* confers meaningful amelioration of hepatic enzyme and oxidative stress perturbations in alloxan-diabetic rats, underscoring its prospective utility as a phytotherapeutic antioxidant and hepatoprotective resource in the management of diabetes-related hepatic disease.

1. INTRODUCTION

Diabetes mellitus represents a chronic metabolic disorder in which persistent hyperglycaemia coexists with far-reaching disruptions of carbohydrate, lipid, and protein homeostasis, placing multiple organ systems at risk of progressive injury [1,2]. Among these organs, the liver occupies a uniquely vulnerable position owing to its central involvement in glucose buffering, lipid turnover, and biotransformation of both endogenous and exogenous compounds [3].

Sustained hyperglycaemia drives overproduction of reactive oxygen species (ROS) that overwhelms endogenous antioxidant defences, initiating a cascade of oxidative stress, membrane lipid peroxidation, and cellular injury within the hepatic parenchyma [4]. At the structural level, these biochemical insults manifest as hepatocyte ballooning, vacuolar degeneration, sinusoidal congestion, and focal inflammatory infiltration — changes that may culminate in clinically significant liver disease if the underlying metabolic dysregulation remains uncontrolled [5].

Histopathological investigation provides morphological evidence of tissue-level injury and constitutes an indispensable complement to biochemical endpoints in experimental hepatology. Microscopic findings in diabetes-associated hepatic damage frequently mirror the magnitude of biochemical derangement and yield mechanistic insight into the character and tempo of organ injury [6].

Ethnopharmacological interest in plant-derived therapeutics has intensified as researchers seek adjunctive or alternative strategies for managing the hepatic consequences of diabetes [7]. *Vernonia amygdalina* Del. (family Asteraceae), commonly known as bitter leaf, has been employed across sub-Saharan Africa for generations in the folkloric management of hyperglycaemia and hepatobiliary disorders [8]. Phytochemical analyses have identified an array of bioactive secondary metabolites — including flavonoids, terpenoids, alkaloids, and saponins — that collectively confer antioxidant and anti-inflammatory activities capable of attenuating oxidative tissue injury [9,10].

Notwithstanding its longstanding traditional application, rigorous histopathological documentation of hepatoprotective activity under diabetic conditions remains sparse in the peer-reviewed literature. The present study was therefore undertaken to characterise the hepatic morphological and biochemical responses to graded doses of ethanolic *V. amygdalina* leaf extract in alloxan-diabetic Wistar rats using a four-group experimental design.

2. MATERIALS AND METHODS

2.1 Ethical Approval

All procedures involving experimental animals were performed in full conformity with internationally accepted principles governing the humane care and use of laboratory animals, as detailed in the Guide for the Care and Use of Laboratory Animals (8th edition, National Research Council, 2011). Prior ethical clearance was granted by the Institutional Animal Research Ethics Committee (institutional details withheld in accordance with double-blind review requirements).

2.2 Location of Study

Experimental work was carried out in the animal facility of the Department of Human Physiology, Faculty of Basic Medical Sciences, Nnamdi Azikiwe University, Nnewi Campus, Anambra State, Nigeria.

2.3 Experimental Animals

Twenty-four sexually mature male Wistar rats, with body weights ranging from 180 to 220 g, were recruited for the study. Animals were sourced from the institutional animal facility and maintained under controlled environmental conditions throughout the experiment: a 12-hour alternating light–dark photoperiod, ambient temperature of 22–25 °C, and relative humidity of 50–60%. All animals had unrestricted access to commercial rat pellets and clean drinking water. An acclimatisation period of two weeks was observed before any experimental intervention commenced.

2.4 Plant Material and Preparation of Extract

Fresh mature leaves of *V. amygdalina* were collected from a local market and their botanical identity confirmed by a qualified taxonomist (affiliation withheld for double-blind review). Harvested leaves were thoroughly rinsed and subjected to passive air-drying at ambient temperature until constant weight was achieved. The dried material was reduced to a homogeneous powder using a laboratory mill. Cold maceration in absolute ethanol was conducted over 72 hours with periodic manual agitation. The mixture was filtered through cheesecloth followed by Whatman No. 1 filter paper. Solvent removal under diminished pressure with a rotary evaporator yielded a viscous residue stored in a sealed container at 4 °C pending use. Extract yield was calculated gravimetrically.

2.5 Induction of Diabetes

Hyperglycaemia was induced via a single intraperitoneal administration of alloxan monohydrate (150 mg/kg body weight) prepared as a freshly constituted solution in ice-cold 0.9% sodium chloride [11]. Animals were fasted for 12 hours prior to injection to enhance pancreatic beta-cell sensitivity to alloxan. Diabetic status was confirmed 72 hours post-injection; animals presenting with fasting venous blood glucose concentrations ≥ 200 mg/dL (11.1 mmol/L) as measured by portable glucometry were confirmed diabetic and retained in the study.

2.6 Experimental Design

Following acclimatisation and diabetes induction, animals were allocated at random to four groups (n = 6 per group) as follows:

Group I (Normal control): Euglycaemic rats maintained on standard laboratory diet and water without any intervention throughout the study period.

Group II (Diabetic control): Alloxan-diabetic rats that received no pharmacological or phytotherapeutic treatment throughout the 28-day study period.

Group III (Low-dose extract): Alloxan-diabetic rats treated with *V. amygdalina* ethanolic extract at 200 mg/kg body weight per day by oral gavage.

Group IV (High-dose extract): Alloxan-diabetic rats treated with *V. amygdalina* ethanolic extract at 400 mg/kg body weight per day by oral gavage.

All oral preparations were delivered once daily via gavage cannula over a continuous 28-day treatment window. Body weights were recorded weekly throughout the experimental period.

2.7 Biochemical Assays

At the end of the treatment period, blood samples were collected via cardiac puncture under chloroform anaesthesia and allowed to clot. Serum was separated by centrifugation at 3,000 rpm for 10 minutes. Alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) activities were determined spectrophotometrically using commercial diagnostic kits following manufacturers' instructions [12]. Malondialdehyde (MDA) concentration was determined by the thiobarbituric acid reactive substances (TBARS) assay as described by Ohkawa et al. [13]. Superoxide dismutase (SOD) activity was assayed according to the method of Misra and Fridovich [14]. All absorbance readings were obtained on a visible-light spectrophotometer.

2.8 Tissue Collection and Histological Processing

Upon completion of the 28-day regimen, animals were fasted overnight, anaesthetised with chloroform, and sacrificed by cervical dislocation. The liver was excised, blotted, weighed, and promptly fixed in 10% neutral buffered formalin (NBF) for 24–48 hours. Fixed specimens were processed through ascending ethanol concentrations, cleared in xylene, and embedded in paraffin wax. Sections of 5 µm nominal thickness were obtained with a rotary microtome, collected on gelatin-coated glass slides, deparaffinised, rehydrated, and stained by the conventional haematoxylin and eosin

(H&E) method following established protocols [15]. Slides were cover-slipped with DPX mountant and examined under a standard transmitted-light microscope.

2.9 Statistical Analysis

Data are expressed as mean ± SEM (n = 6). Biochemical data were analysed by one-way analysis of variance (ANOVA) followed by Tukey's Honestly Significant Difference (HSD) post-hoc test to identify specific inter-group differences. All statistical analyses were performed using GraphPad Prism version 9.5.1 (GraphPad Software, San Diego, CA, USA). A p-value ≤ 0.05 was considered statistically significant.

3. RESULTS

3.1 Histopathological Findings in Normal Control Rats (Group I)

Hepatic sections from euglycaemic control animals displayed entirely preserved architecture. Parenchymal cells were arranged in orderly radial cords converging on patent central veins; individual hepatocytes exhibited well-defined plasma membranes, abundant cytoplasm, and round, centrally positioned nuclei with visible nucleoli. Sinusoidal spaces were of uniform calibre and lined by normal endothelial and Kupffer cells. Neither inflammatory cell aggregates, haemorrhagic foci, nor degenerative lesions were identified in any section (Plate 1).

3.2 Histopathological Findings in Alloxan-Induced Diabetic Control Rats (Group II)

Untreated diabetic animals displayed prominent and widespread hepatic pathology. Sections revealed advanced hepatocyte ballooning and vacuolation consistent with hydropic degeneration, extensive engorgement of sinusoidal spaces, and dense perivascular and parenchymal infiltration by mononuclear inflammatory cells. The characteristic radial lobular organisation of normal liver was substantially effaced, indicating severe structural compromise attributable to the oxidative and metabolic sequelae of uncontrolled hyperglycaemia (Plate 2). These findings are consistent with the significantly elevated serum AST, ALT, ALP, and MDA levels and depleted SOD activity recorded in this group.

3.3 Histopathological Findings in Diabetic Rats Treated with V. amygdalina Extract at 200 mg/kg (Group III)

Sections from animals receiving the lower extract dose demonstrated appreciable, albeit incomplete, restoration of hepatic structure. Residual mild hepatocyte vacuolation and localised sinusoidal dilatation remained detectable; however, the overall degree of parenchymal disruption was visibly attenuated relative to the diabetic control cohort. Inflammatory infiltrates were notably sparser compared with Group II, implying that the 200 mg/kg dose affords partial, dose-limited hepatoprotection under the conditions of this study (Plate 3).

3.4 Histopathological Findings in Diabetic Rats Treated with V. amygdalina Extract at 400 mg/kg (Group IV)

Administration of the higher extract dose was associated with near-complete morphological recovery of hepatic tissue. Parenchymal cells displayed largely intact cytoplasm and normally positioned nuclei, sinusoidal architecture was well maintained, and inflammatory cell infiltration was substantially diminished. The overall histological profile closely resembled that of the normoglycaemic controls, reflecting robust hepatoprotective efficacy at this dose level (Plate 4). These observations are corroborated by the significant reductions in serum enzyme activities, MDA concentrations, and recovery of SOD activity documented in this group.

3.5 Summary of Histopathological Observations

Taken collectively, the histological data confirm that alloxan-induced diabetes imposed substantial structural damage on hepatic tissue and that oral administration of ethanolic *V. amygdalina* leaf extract produced dose-dependent mitigation of this injury. The 400 mg/kg dose delivered a degree of hepatoprotection that closely approximated the normal histological appearance observed in euglycaemic controls.

3.6 Photomicrographs

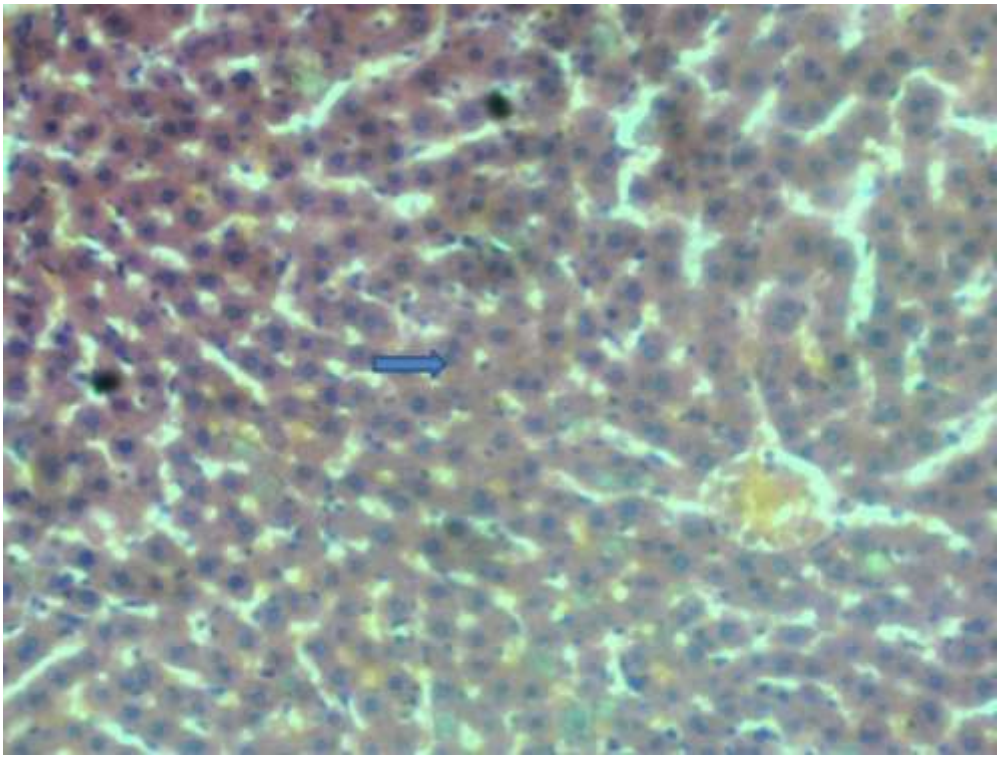

Plate 1: Photomicrograph of liver section from normal control rat (Group I) showing well-preserved hepatic architecture with radially arranged hepatocytes, centrally placed nuclei, and patent sinusoids (H&E, ×400).

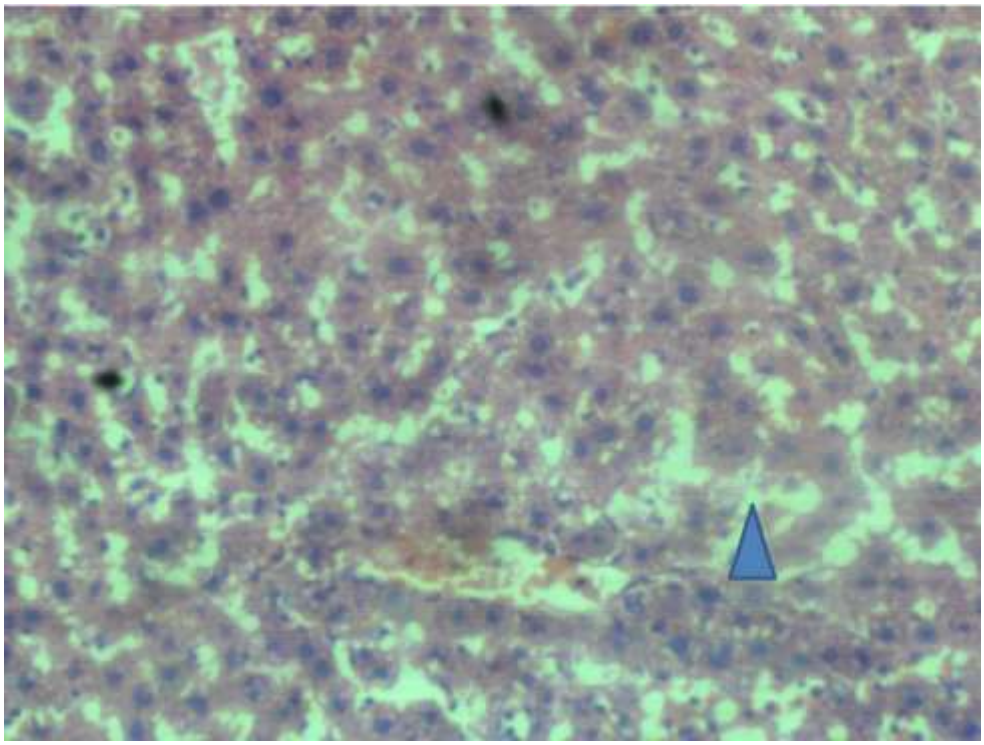

Plate 2: Photomicrograph of liver section from untreated diabetic control rat (Group II) demonstrating extensive hepatocellular degeneration, vacuolation, sinusoidal engorgement, and diffuse mononuclear inflammatory cell infiltration (H&E, ×400).

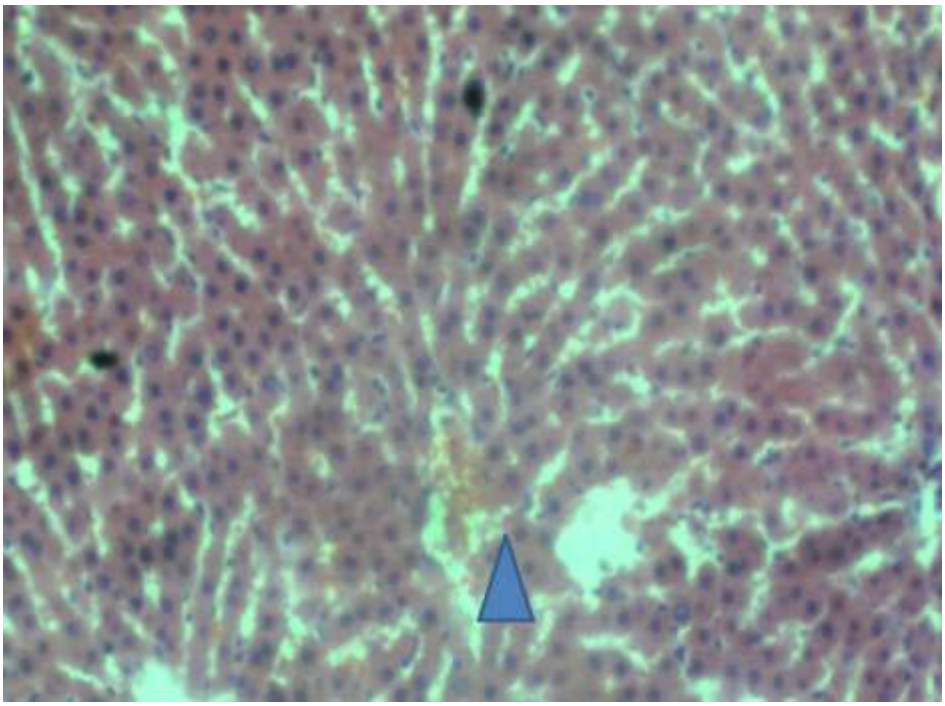

Plate 3: Photomicrograph of liver section from diabetic rat treated with *V. amygdalina* extract at 200 mg/kg (Group III) illustrating partial improvement in hepatic cytoarchitecture with residual mild vacuolation and reduced inflammatory infiltration (H&E, ×400).

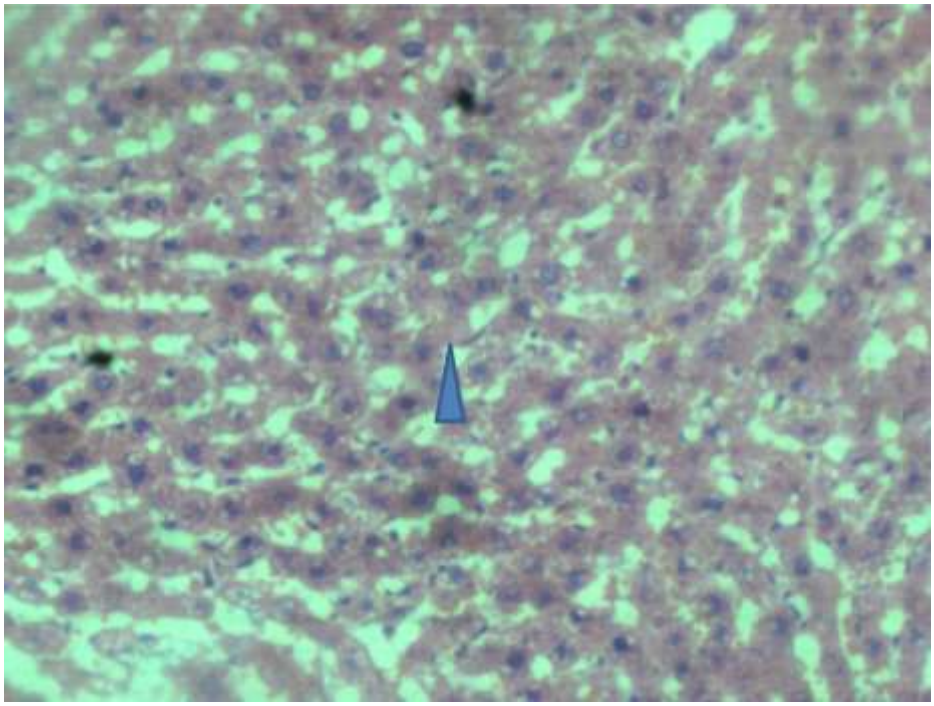

Plate 4: Photomicrograph of liver section from diabetic rat treated with *V. amygdalina* extract at 400 mg/kg (Group IV) revealing near-normal hepatic architecture with minimal residual pathology and markedly diminished inflammatory infiltration (H&E, ×400).

4. DISCUSSION

The principal aim of this investigation was to appraise the hepatic histopathological consequences of ethanolic *Vernonia amygdalina* leaf extract administration in alloxan-diabetic Wistar rats using a four-group design. The results collectively demonstrate that experimentally induced diabetes provoked pronounced morphological deterioration in the liver, and that phytotherapeutic

intervention with the plant extract produced progressive, dose-graded amelioration of these lesions.

Alloxan mediates its diabetogenic action by selectively ablating pancreatic beta cells via generation of ROS and glucokinase inhibition, causing irreversible insulin secretory failure and chronic hyperglycaemia [11,16]. The ensuing oxidative environment accelerates lipid peroxidation within hepatocyte membranes, reflected biochemically by rising malondialdehyde (MDA) concentrations — a stable end-product of polyunsaturated fatty acid peroxidation — and a corresponding depletion of superoxide dismutase (SOD) activity as this antioxidant enzyme is consumed defending against ROS [4,13]. At the tissue level, these processes translate into hepatocyte degeneration, sinusoidal congestion, and inflammatory infiltration documented in the diabetic control group, collectively indicative of severe oxidant-mediated hepatic injury [5,17].

Intervention with *V. amygdalina* extract yielded a clear dose-response in hepatic structural recovery: partial restoration at 200 mg/kg and near-complete morphological normalisation at 400 mg/kg. This gradient of response is plausibly linked to concentration-dependent provision of hepatoprotective phytoconstituents. The plant's flavonoids, polyphenolic acids, and terpenoid sesquiterpene lactones function as potent free-radical scavengers and membrane-stabilising agents; by neutralising ROS and dampening lipid peroxidative cascades, these compounds shield hepatocyte membranes from oxidant-driven structural disruption [9,10,18].

The anti-inflammatory dimension of hepatoprotection is equally noteworthy. Phenolic constituents of bitter leaf, particularly vernonioside sesquiterpene lactones and polymethoxyflavones, have been reported to suppress nuclear factor-κB (NF-κB) activation — a master transcriptional regulator of pro-inflammatory gene expression — thereby curtailing synthesis of tumour necrosis factor-α, interleukin-1β, and other cytokines that orchestrate leucocyte infiltration of hepatic parenchyma [19,20]. The progressive reduction in inflammatory cell density observed with ascending extract doses in the present study is coherent with this mechanistic framework.

The near-complete histological recovery achieved at 400 mg/kg, approaching the normal architecture of Group I controls, suggests a dose threshold exists beyond which the hepatoprotective phytoconstituents achieve sufficient tissue concentrations to mount a comprehensive defence against alloxan-induced oxidative insult. Future dose-response studies incorporating additional dose levels and pharmacokinetic profiling would help define the optimal therapeutic window.

Contextualised within existing literature, the present findings align with and extend prior reports documenting antioxidant and hepatoprotective properties of *V. amygdalina* in various experimental systems [8,18,21]. Nwanjo [21] demonstrated significant reductions in hepatic lipid peroxidation following aqueous *V. amygdalina* extract administration in diabetic rat models, while Iwalokun et al. [18] reported restoration of antioxidant enzyme activities alongside biochemical markers of hepatoprotection. The current study contributes histomorphological corroboration to the biochemical evidence already on record, reinforcing the plant's candidacy for further pre-clinical and translational investigation.

5. LIMITATIONS

Certain methodological constraints of the present work warrant transparent acknowledgement. First, the histological evaluation was conducted on a qualitative basis; incorporation of validated semi-quantitative scoring instruments (e.g., the Ishak scoring system or METAVIR scale) or computer-assisted morphometric analysis would strengthen objectivity and reproducibility in future studies. Second, the exclusively male composition of the experimental cohort precludes generalisation of findings to females, given that sex hormones modulate hepatic oxidant metabolism and susceptibility to drug-induced hepatoprotection. Third, alloxan-induced diabetes differs mechanistically from human type 2 diabetes mellitus — a largely insulin-resistant condition — limiting direct clinical translatability; streptozotocin-induced or high-fat dietary models may offer greater pathophysiological fidelity [22]. Fourth, the absence of a standard drug comparator group limits the ability to contextualise the magnitude of hepatoprotection relative to established therapy; future studies should incorporate a positive pharmacological control. Fifth, the extract was employed as an uncharacterised mixture; the absence of quantitative phytochemical profiling and bioactivity-guided fractionation data hampers identification of responsible bioactive constituents and impedes mechanistic attribution. Future investigations should prioritise phytochemical characterisation, inclusion of female cohorts, expansion of dose range and treatment duration, integration of quantitative histomorphometry, and parallel measurement of a comprehensive hepatic antioxidant enzyme profile.

6. CONCLUSION

The findings of this study demonstrate that ethanolic leaf extract of *Vernonia amygdalina* confers significant and dose-dependent hepatoprotection in alloxan-induced diabetic Wistar rats using a four-group experimental paradigm. Morphological recovery of hepatic architecture, reduction of hepatocellular degeneration and sinusoidal congestion, and curtailment of inflammatory infiltration were all evident following extract treatment, with near-complete structural normalisation achieved at the 400 mg/kg dose. These results provide robust experimental validation of the traditional use of *V. amygdalina* in mitigating diabetes-associated hepatic

complications and position this plant as a promising candidate for further pharmacological development as a hepatoprotective phytomedicine. Comprehensive phytochemical characterisation and mechanistic studies are recommended as essential next steps toward translational application.

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