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Bridging Ethnomedicine and Biomedicine: Ethnomedicinal and Anti-Inflammatory Evaluation of *Aspidium Cicutarium* Rhizome

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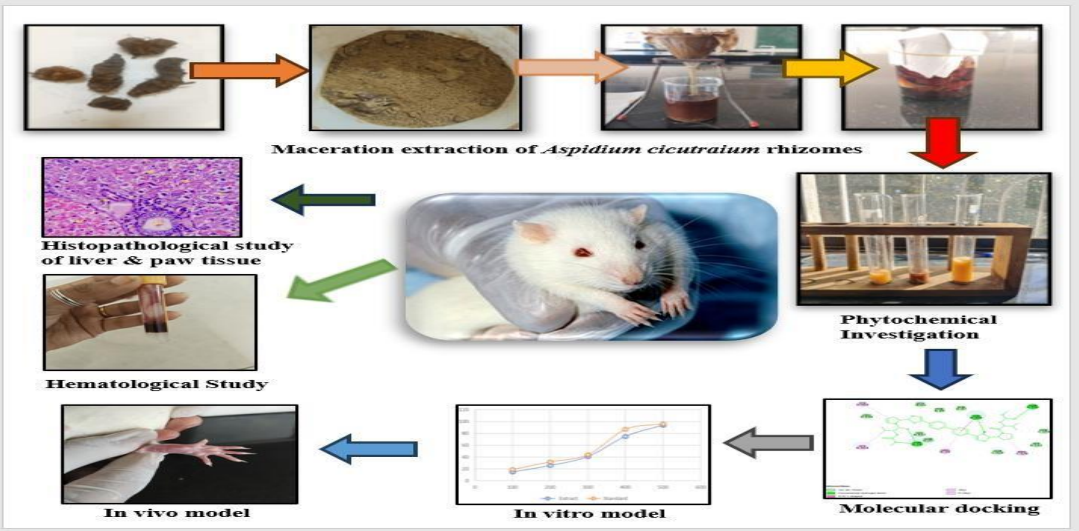
ABSTRACT

The current research explores the anti-inflammatory potential of Aspidium cicutarium rhizome using phytochemical, pharmacological, and molecular docking methods. Inflammation is a major response linked to many chronic diseases, and existing treatments like NSAIDs often come with serious side effects. Considering the traditional medicinal use of Aspidium cicutarium, this study sought to confirm its effectiveness through scientific means. It is traditionally used in the Konkan region of India to treat inflammatory conditions. The methanolic extract of the rhizome by the maceration method was evaluated for phytochemical study and showed the presence of active components such as alkaloids, phenols, and tannins. The methanolic extract was assessed for in vitro anti-inflammatory activity using a protein denaturation test, and in vivo activity was measured with a carrageenan-induced paw edema model in Sprague Dawley rats. The results showed a significant reduction of inflammation in both tests, with higher doses providing more effectiveness. Molecular docking studies also indicated strong binding between key phytochemicals and the COX receptor, suggesting a possible mechanism to reduce inflammation. Blood samples were withdrawn from all the animals by retro-orbital plexus puncture. Blood was collected for hematology analysis for biomarker investigation (C-reactive protein). Overall, these findings support the traditional use of Aspidium cicutarium and highlight its potential as a natural option for creating safer anti-inflammatory treatments.

Keywords: *Anti-inflammatory; Aspidium cicutarium; NSAID alternatives; Natural therapeutic leads; Biomarkers; Integrative pharmacology.*

GRAPHICAL ABSTRACT



INTRODUCTION

According to the World Health Organization (WHO), diseases with an inflammatory origin pose the most significant threat to human existence. They stand as the primary cause behind global mortality rates and are projected to escalate as leading causes of death in the United States over the next three decades. Research conducted by the Rand Corporation in 2014 revealed that approximately 60% of the American population is afflicted by at least one chronic inflammatory ailment [1,2].

Inflammation is a physiological defence mechanism characterized by a coordinated series of cellular and molecular responses aimed at limiting tissue injury and eliminating pathogenic agents. This complex biological process involves vasodilation of arterioles, venules, and capillaries, accompanied by increased vascular permeability. These vascular changes facilitate the exudation of plasma proteins and the directed migration of leukocytes to the site of inflammation. During the inflammatory response, there is a sequential infiltration of the affected tissue by polymorphonuclear leukocytes (PMNs), followed by monocytes and lymphocytes, contributing to the resolution or progression of the inflammatory reaction [3–5].

Inflammation involves a localized rise in leukocyte number and many complex mediators. Prostaglandins are universal substances that indicate and modulate tissue responses during inflammation. Biosynthesis of prostaglandins has been implicated in the pathology of Alzheimer's disease, cardiovascular diseases, colonic adenomas, and cancer [6].

Non-steroidal anti-inflammatory drugs (NSAIDs) are widely used for the symptomatic treatment of rheumatic disorders. Since the early seventies, it has been widely accepted that the main mechanism of action—also responsible for the main side effect of gastric mucosal damage—is inhibition of cyclooxygenase (COX), a key enzyme in prostaglandin synthesis [7,8].

Therapeutically, NSAIDs act by inhibiting cyclooxygenase (COX-1 and COX-2) and thereby the formation of prostaglandins (PGs). COX-1 generates thromboxane A2 and prostaglandins that regulate the gastrointestinal mucosal barrier, renal balance, and platelet aggregation, whereas COX-2 is induced in inflammatory cells to produce PGs linked to fever, pain, and inflammation [9].

While one mode relies on NSAIDs regulating tissue inflammation via prostanoid synthesis inhibition, a second mode is linked to mitochondrial toxicity. Before organ-specific effects are discussed, it is essential to outline the basic mechanisms behind NSAID-related organ damage [10].

With the discovery of aspirin's mechanism (John Vane; Nobel Prize 1982), understanding of NSAIDs increased, enabling development of novel anti-inflammatory therapeutics. Because of their analgesic, antipyretic, and anti-inflammatory activities, these drugs are used widely for pain, fever, and inflammation in rheumatic disorders, osteoarthritis, and dysmenorrhea [11].

Medicinal plants remain vital for drug development and primary healthcare due to affordability and generally favorable safety. Globally, over 20,000 medicinal plant species have been cataloged, with India contributing significantly due to floristic diversity in regions such as the Eastern Himalayas and Western Ghats. Approximately 70% of rural India's population still depends on traditional medicine, with growing global interest in herbal therapeutics [12–21].

Aspidium cicutarium has been reported to contain several key phytoconstituents, including alkaloids and glycosides, and various parts of the plant have shown pharmacological properties including wound healing, antidiabetic, and antimicrobial effects. The present study investigates the anti-inflammatory potential of *Aspidium cicutarium* [22].

MATERIALS AND METHODS

Extraction of *Aspidium cicutarium*

Rhizomes of *Aspidium cicutarium* were collected and authenticated at ADT's, Department of Botany, Shardabai Pawar Mahila Arts, Commerce and Science College, Shardanagar, Baramati, Dist-Pune (Ref. No.

PASR 261). Fresh rhizomes were shade-dried for 3–4 days, powdered, and sieved to obtain a fine powder. The powder was macerated with methanol at 38°C for 120 hours. The macerate was filtered and concentrated via natural evaporation for 48 hours and then by gentle stirring at 40°C. The extract was stored in a labelled airtight container. Overall percent yield: 10.3%.

Phytochemical screening

Alkaloids: Dragendorff's (orange-red ppt), Mayer's (yellow ppt), Wagner's (reddish-brown ppt), Hager's (yellow/orange ppt). Cardiac glycosides: Keller–Kiliani (blue color in lower layer), Legal test (pink to purple), Borntrager's (pink to red in ammoniacal layer) [23].

Thin Layer Chromatography (TLC)

Normal-phase TLC of the methanolic extract was performed on silica gel G60F254. Solvent systems: (A) n-butanol:acetic acid:water (4:2:1) and (B) ethyl acetate:methanol:water (8:4:1). Spots were visualized under UV at 254 and 354 nm [24].

FT-IR Analysis

Semi-solid extract was ground with KBr to form pellets. Spectra were recorded and characteristic bands used to assign functional groups.

Molecular Docking

Phytochemicals (e.g., berberine) were retrieved from PubChem (SMILES) and converted to PDB/SDF. COX receptor (PDB ID: 3LN1, Mus musculus) was downloaded from the Protein Data Bank. Ligands were prepared by adding polar hydrogens; water/heteroatoms were removed. Structures were converted to PDBQT. Docking was performed with AutoDock Vina using 3LN1 as the receptor. Binding energies were recorded; best poses were saved for analysis in Discovery Studio [25].

In vitro model – Protein denaturation assay

A 5 mL reaction mixture contained 0.2 mL egg albumin (1–2%), 2 mL test extract or diclofenac sodium at various concentrations, and 2.8 mL PBS (pH 7.4). Control used 2 mL water. Mixtures were incubated at 37 ± 2°C for 30 min and then heated at 70 ± 2°C for 15 min. After cooling, absorbance was read at 280 nm [26].

In vivo model – Carrageenan-induced paw edema

Sprague Dawley rats (100–150 g) were housed at 22 ± 2°C, 45–55% RH, 12 h light/dark with food and water ad libitum. IAEC approval: SGRS/IAEC/08/2024-25 (as per CCSEA). Thirty-six males were randomized into six groups. Extract was administered orally; diclofenac (standard) was given 1 h prior to carrageenan. Edema was induced by subplantar injection of 0.1 mL carrageenan in the right hind paw. Paw volume was measured at baseline and at 30, 60, and 240 min using a plethysmometer (UGO-BASILE: 7140). Hematology and histopathology were performed on day 8 [27].

Statistical analysis

Results are mean ± SEM. One-way ANOVA followed by Dunnett's test; $p < 0.05$ deemed significant.

RESULTS

The methanolic extraction of dried *Aspidium cicutarium* rhizomes yielded 10.3% w/w based on initial dry weight.

Phytochemical screening

Alkaloids and glycosides were indicated by classical color tests as described in Methods.

Thin layer chromatography

For alkaloids (mobile phase n-butanol:acetic acid:water 4:2:1), the extract showed Rf 0.66 (cf. atropine sulfate Rf 0.70). For glycosides (ethyl acetate:methanol:water 8:4:1), characteristic spots were observed.

FT-IR

Characteristic bands supported the presence of amine/amide (N–H), aliphatic C–H, and carbonyl (C=O) functionalities.

Table No.1 : FT-IR values with standard range

Functional Group	Obtained Value (cm^{-1})	Standard Range (cm^{-1})	Interpretation
N–H (amine or amide)	3448	3300–3500 (sharp)	Within range
C=C (alkene)	1404	1620–1680 (medium)	Below expected
Aromatic C–H	3140	3000–3100 (weak to medium)	Slightly above
Aliphatic C–H	2924	2850–2960 (strong)	Within range
C=O (carbonyl)	1720	1700–1750 (strong)	Within range

Molecular Docking

Docking against COX (PDB: 3LN1) indicated strong binding affinity of the selected ligand with a best pose energy of −10.4 kcal/mol (RMSD = 0).

Ligand	Binding Affinity (kcal/mol)	rmsd/ub	rmsd/lb
3ln1	-10.4	0	0

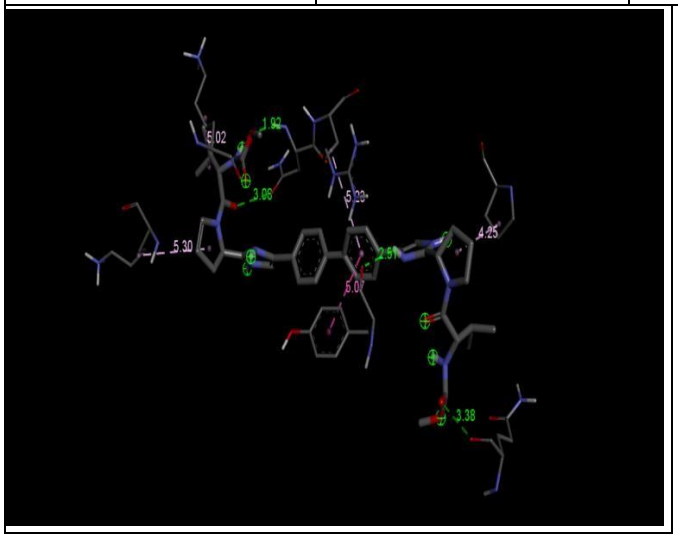


Figure No.1: Ligand Interaction which displace the hydrogen bonding

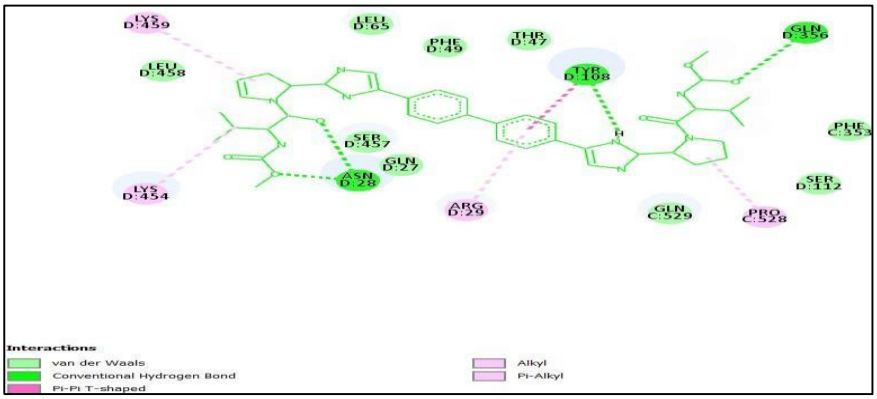


Figure No.2: Ligand Interaction with Length and type of bond.

In vitro model – Protein denaturation assay

Percentage inhibition increased with concentration for both extract and standard, with near-comparable activity at 500 µg/mL. IC50 for the extract ≈ 326.5 µg/mL.

Table No.2 : The percentage inhibition by protein denaturation assay

Sr No	Conc (µg/mL)	Extract (% inhibition)	Standard (% inhibition)
1	100	15	19
2	200	26	32
3	300	41	44
4	400	75	87
5	500	94	96

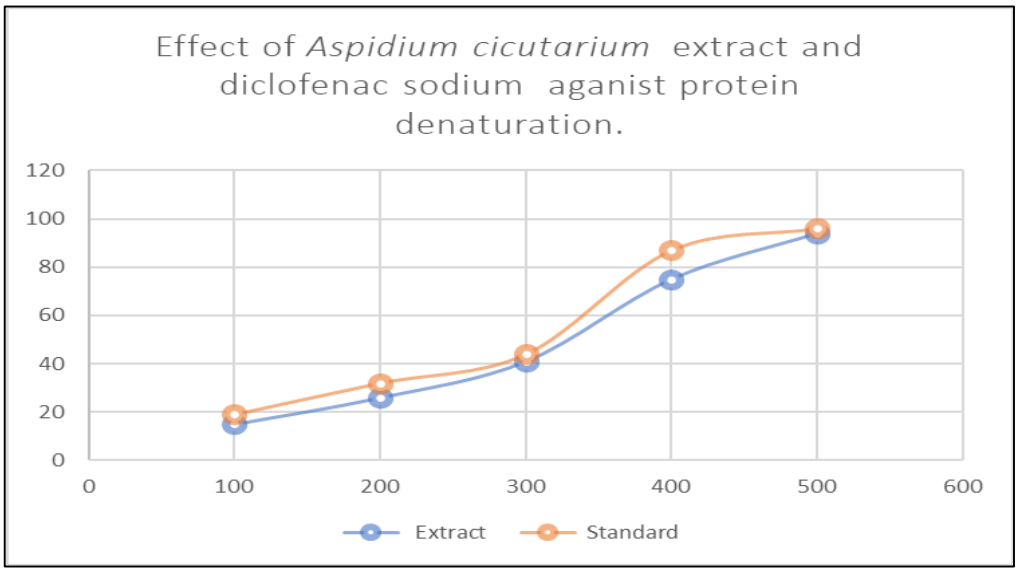


Figure No 3 : Effcet of *Aspidium cicutarium* extract and Diclofenac Sodium against protein denaturation

In vivo model – Carrageenan-induced paw edema in rats

Table No. 3 : Effect of extract on paw volume of Carrageenan induced paw edema in rats

Sr No.	Groups	Dose (mg/kg)	0 min	30 min	60 min	240 min	Notes
1	NC	Vehicle	0.86 #	0.90 ###	1.01 #	1.05 #	
2	STD	10	0.66±0.5**	0.87±0.04***	0.82±0.04***	0.71±0.5***	
3	DC	0.1 mL	0.88±0.02	1.42±0.03	1.45±0.02	1.68±0.84	
4	T1	100	0.93±0.01 ns	1.25±0.02*	1.24±0.02*	1.18±0.3*	
5	T2	200	1.01±0.03 ns	1.20±0.02**	1.19±0.02**	1.10±0.02	
6	T3	400	0.98±0.01**	1.18±0.02***	1.12±0.02***	1.05±0.02***	

Paw volume values are mean ± SEM (n=4). One-way ANOVA followed by Dunnett's test; $p < 0.05$ considered significant. ns $p>0.05$; ** $p<0.01$; *** $p<0.001$.

Percentage of edema inhibition in rats

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Table No. 4: Effect of extract on the % inhibition of paw volume in Carrageenan-induced paw edema in rats.

Groups	30 min	60 min	240 min
Normal Control (NC)	–	–	–
Control (DC)	0%	0%	0%
Diclofenac (STD)	64%	77.5%	93%
T1	42%	45.75%	68.5%
T2	61.75%	67.5%	87.75%
T3	63.5%	74.5%	90%

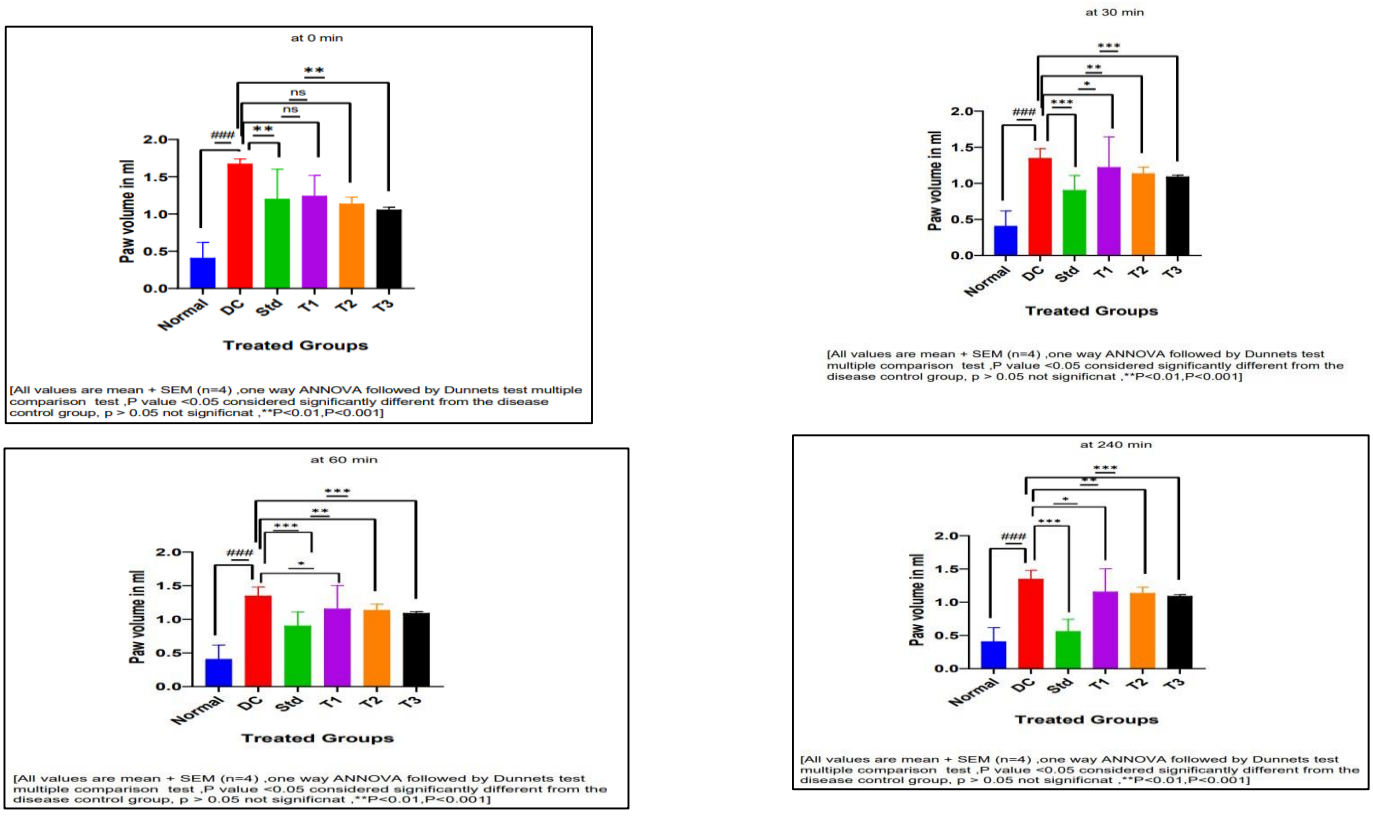


Figure No. 4: Effect of extract on the % inhibition of paw volume in Carrageenan-induced paw edema in rats.

Table No 5 : Effect of extract on the C-reactive protein (CRP) levels

Groups	CRP (mg/L)
NC	11.464 ± 0.542
DC	9.906 ± 0.032
STD	10.496 ± 0.149
T1	3.230 ± 0.195
T2	1.542 ± 0.133
T3	4.814 ± 0.045

Data are mean ± SEM. One-way ANOVA with Dunnett's test. P > 0.05 non-significant; *** p < 0.001; **** p < 0.0001.

The table displays C-reactive protein (CRP) levels (in mg/L) for different treatment groups, reflecting the degree of inflammation. The Normal Control (NC) group shows the highest CRP level (15.714 mg/L), indicating a normal baseline without inflammation induction. The Disease Control (DC) group shows a reduced CRP level (9.821 mg/L), reflecting carrageenan-induced inflammation. Diclofenac (STD) moderately reduces CRP (10.804 mg/L), while the test groups T1, T2, and T3 show a dose-dependent reduction, , particularly T1, T2, and T3, are effective in significantly reducing CRP levels in a dose-dependent manner (T2 showing the greatest effect), whereas the standard treatment did not show a significant reduction compared to the disease control

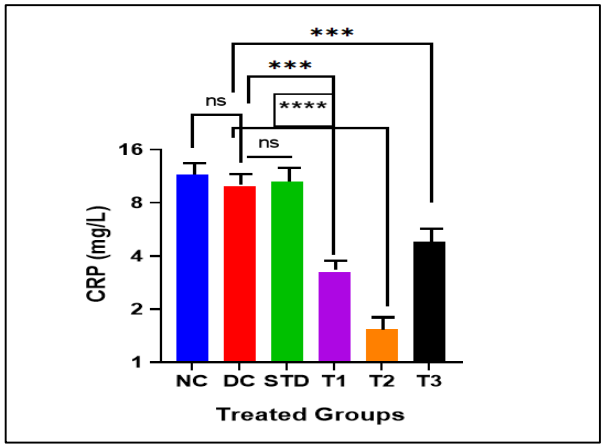


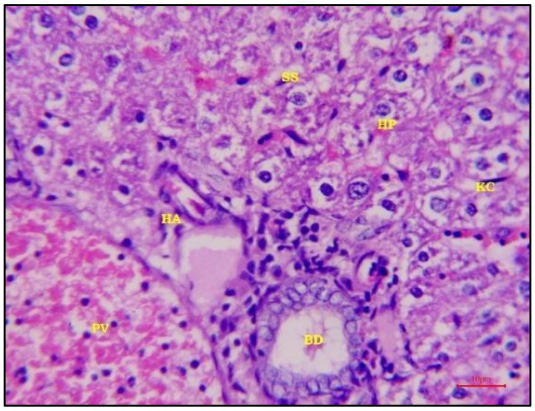
Figure No 5 : Effect of extract on the C-reactive protein (CRP) levels
The data are presented as means ± SEM .One way ANOVA test by Dunnet's test. P > 0.05 non- significant,P > 0.05 non- significant,*** p < 0.001 Highly significant ,**** p < 0.0001 Extremely significant

Histopathological study

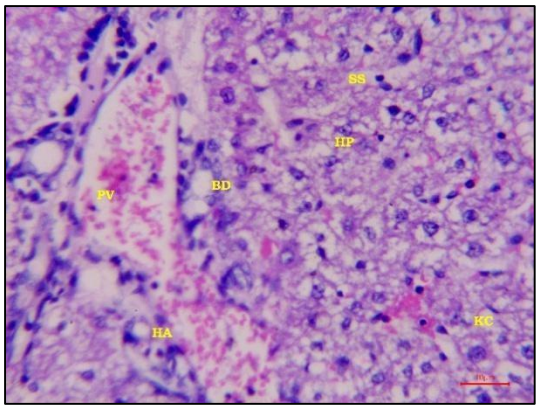
Liver and paw tissues were examined. Representative micrographs to be inserted for groups: Normal Control, Standard, Disease Control, T1 (100 mg/kg), T2 (200 mg/kg), T3 (400 mg/kg).

Histopathological study of Liver:–

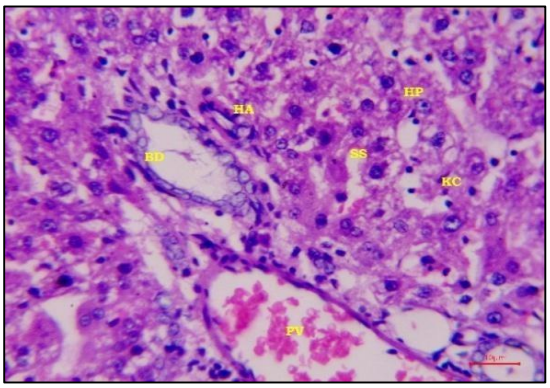
1. Liver



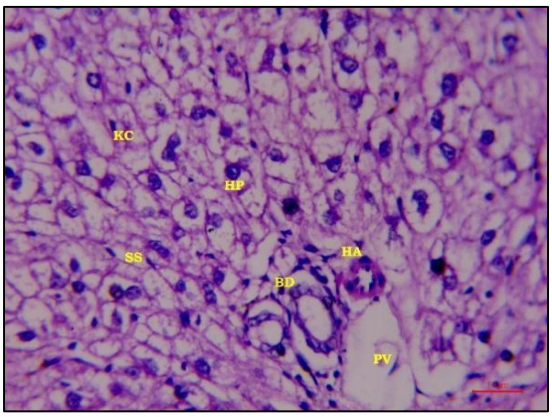
Group Ⅰ -Normal Control



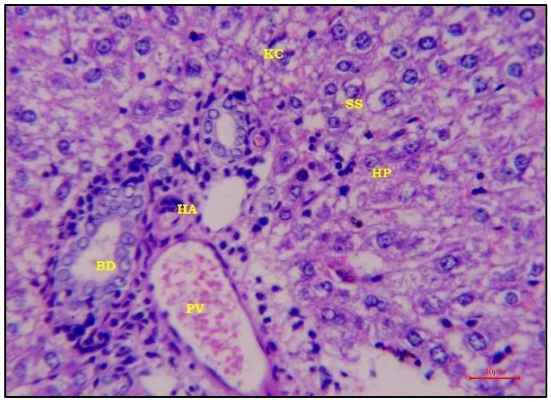
Group Ⅱ - Standard



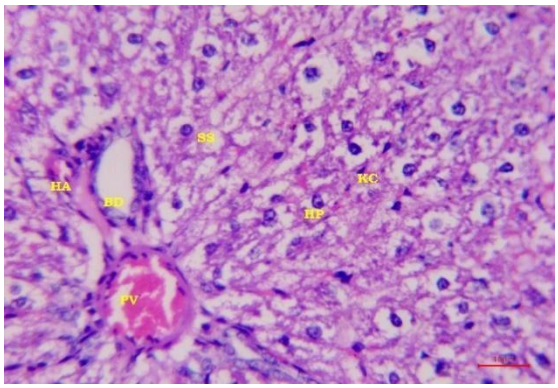
Group Ⅲ -Disease control



Group Ⅳ -T1 100 mg/kg



Group Ⅴ -T2 200 mg /kg



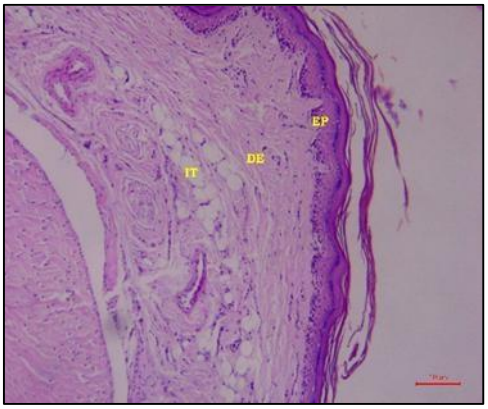
Group Ⅵ -T3 400 mg/kg

Table No 6 **Histopathological Evaluation of Liver Tissue**

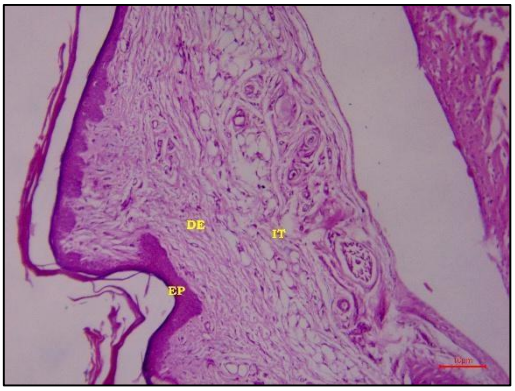
Group	Histopathological Microscopic Observations	Pathological Grade

NC (Normal Control)	Normal histoarchitecture with intact central/portal veins, hepatic artery, bile duct, and canaliculi; normal hepatocytes, Kupffer and stellate cells; minimal congestion.	Minimal
DC (Disease Control)	Normal histoarchitecture with minimal hemorrhages and congestion around central and portal veins.	Minimal
STD (Diclofenac)	Normal histoarchitecture; minimal congestion.	Minimal
Test-1 (100 mg/kg)	Normal histoarchitecture; minimal congestion.	Minimal
Test-2 (200 mg/kg)	Normal histoarchitecture; minimal congestion.	Minimal
Test-3 (400 mg/kg)	Normal histoarchitecture; minimal congestion.	Minimal

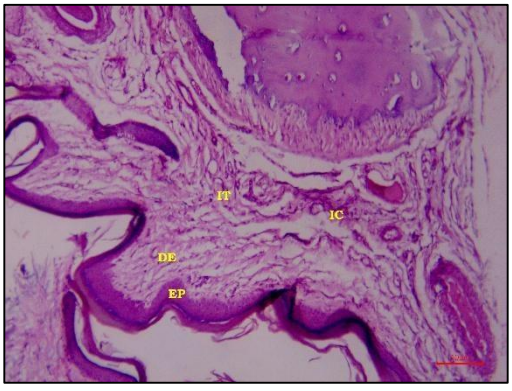
2. Histopathology of Paw Tissue



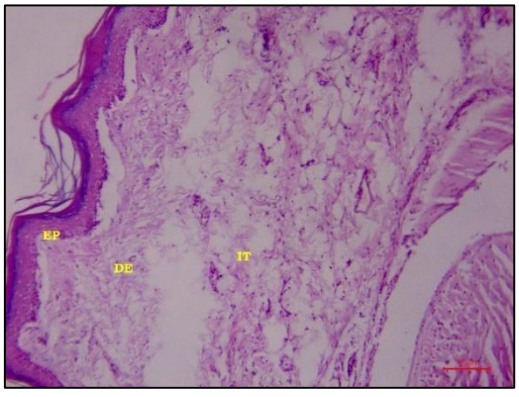
Group Ⅰ – Normal Control



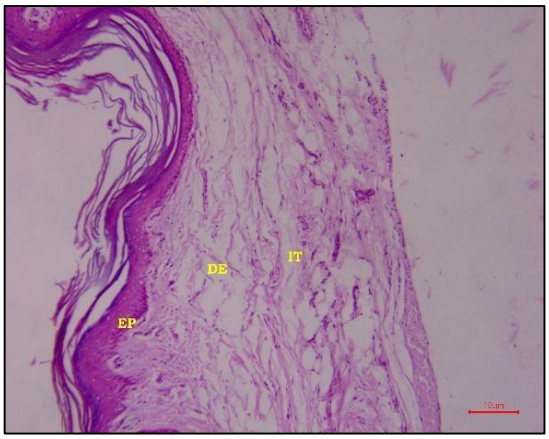
Group Ⅱ - Standard

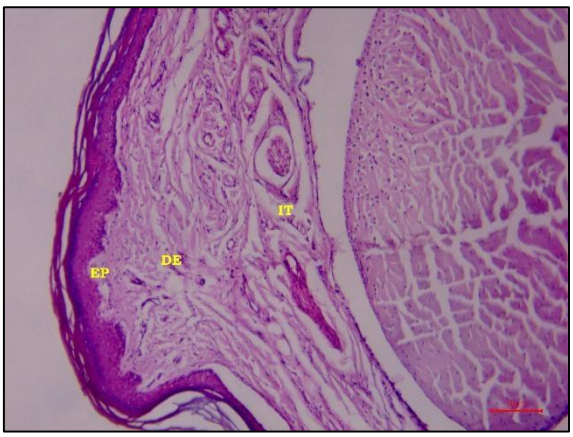


Group Ⅲ- Disease Control



Group Ⅳ -T1 100 mg/kg





Group **V** -T2 200 mg /kg Group **VI** -T3 400 mg/kg

Histopathological examination of paw tissue provides crucial insight into the **cellular and tissue-level effects** of inflammatory stimuli and the protective effects of test compounds. In the present study, carrageenan-induced paw edema in rats led to marked tissue damage in the **disease control group (Group III)**, evidenced by **severe edema, inflammatory cell infiltration, and disruption of normal dermal architecture**. These findings are consistent with previous reports indicating that carrageenan triggers an acute inflammatory response via **histamine, serotonin, bradykinin release, and prostaglandin synthesis**, resulting in vasodilation and leukocyte migration.

Treatment with ***Aspidium cicutarium*** **rhizome extract** exhibited a **dose-dependent protective effect** on paw tissue histology:

- **T1 (100 mg/kg)** showed moderate reduction in edema and inflammatory infiltration, suggesting partial mitigation of acute inflammation.
- **T2 (200 mg/kg)** demonstrated **substantial preservation of tissue architecture**, with mild edema and limited leukocyte infiltration.
- **T3 (400 mg/kg)** exhibited **near-normal histology**, comparable to the standard drug (Diclofenac), indicating **potent anti-inflammatory efficacy at the highest dose**.

The observed histological improvements correlate with the **in vivo paw edema measurements** and **CRP biomarker analysis**, highlighting the extract's ability to reduce inflammatory mediators and tissue damage. The reduction of neutrophilic infiltration and preservation of dermal structure suggest that the extract may **modulate early-phase inflammatory responses**, potentially through inhibition of **COX-mediated prostaglandin synthesis**, as supported by molecular docking studies.

Overall, the histopathological findings provide **cellular-level evidence** for the anti-inflammatory potential of *Aspidium cicutarium* and support its traditional use in managing inflammatory disorders. The data indicate that the extract not only reduces **edema** but also protects tissues from **structural damage**, enhancing its pharmacological relevance.

DISCUSSION

The present study comprehensively evaluated the anti-inflammatory potential of *Aspidium cicutarium* rhizome extract through phytochemical analysis, in vitro and in vivo pharmacological assessments, molecular docking, and histopathological examination. Phytochemical screening and Thin Layer Chromatography confirmed the presence of alkaloids and glycosides, both of which are well-documented for their anti-inflammatory properties. FTIR spectral analysis further supported the presence of key functional groups, including N–H

stretches indicative of amines and C=O stretches characteristic of glycosides, which may facilitate interactions with inflammatory mediators.

Molecular docking studies revealed that phytochemicals from *Aspidium cicutarium* demonstrated strong binding affinities with the cyclooxygenase (COX) receptor, exhibiting hydrogen bonding, π–π stacking, and hydrophobic interactions analogous to classical NSAIDs. These interactions suggest potential inhibition of prostaglandin synthesis, which is central to the anti-inflammatory effect.

In vitro protein denaturation assays demonstrated a dose-dependent inhibition of protein denaturation, with maximum inhibition (94%) observed at 500 µg/mL, comparable to the standard diclofenac (96%). This result underscores the extract's potential to stabilize inflammatory proteins, thereby preventing tissue damage associated with inflammatory responses.

In vivo, the carrageenan-induced paw edema model demonstrated significant anti-inflammatory activity. The extract at 400 mg/kg produced maximal inhibition of paw edema, closely matching the standard treatment, confirming its efficacy in acute inflammation. Histopathological examination of paw tissues provided structural corroboration of these functional effects. The disease control group exhibited marked edema, inflammatory cell infiltration, and disruption of dermal and subcutaneous architecture. In contrast, extract-treated groups showed a **dose-dependent restoration of tissue architecture**, with minimal inflammatory infiltration and preserved connective tissue observed in the highest dose group (400 mg/kg). These findings indicate that the extract not only reduces edema but also mitigates cellular and tissue-level inflammatory damage.

Biochemical assessment of C-reactive protein (CRP), a sensitive marker of inflammation, revealed a significant dose-dependent decline in treated groups, with the 200 mg/kg group showing the greatest reduction. This biochemical outcome aligns with the histopathological observations and the functional reduction in paw edema, collectively demonstrating the systemic anti-inflammatory effects of the extract. Liver histology confirmed normal histoarchitecture across all treatment groups, indicating that the extract is safe at the tested doses and does not induce hepatotoxicity.

Taken together, these findings provide strong pharmacological evidence supporting the traditional use of *Aspidium cicutarium* for the management of inflammatory conditions. The extract exhibits a multi-modal anti-inflammatory effect, encompassing inhibition of protein denaturation, reduction of acute edema, restoration of tissue architecture, and modulation of inflammatory biomarkers. This comprehensive activity profile highlights its potential as a safe and effective plant-based therapeutic for inflammation.

CONCLUSION

Aspidium cicutarium methanolic rhizome extract exhibits significant anti-inflammatory activity across in vitro and in vivo models, with supportive docking and histopathology. The data provide pharmacological validation for its ethnomedicinal use and justify further isolation and characterization of active constituents.

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FUTURE PERSPECTIVES

Future work should isolate and characterize individual phytochemicals, quantify their abundance, explore geographic/environmental variability, and assess mechanisms in-depth. Synergy studies and standardized extract development with comprehensive toxicity profiling are recommended. Screening of related Tectaria species may broaden discovery.

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