Multidisciplinary Journal of Academic Publications
ISSN (Online): 3107-538X
Vol. 02, Issue 06, November-December 2026

PHYTOCHEMICAL ANALYSIS AND EVALUATION OF THE ANTI-ULCEROGENIC POTENTIAL OF BRYOPHYLLUM CALYCINUM LEAF EXTRACT

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ABSTRACT

Bryophyllum calycinum Salisb. (Crassulaceae), commonly known as the life plant, has a long record of use in Indian folk medicine for digestive complaints, including peptic ulcers [1], [2]. This study presents a systematic phytochemical screening and an evaluation of the anti-ulcerogenic activity of its leaf extract, with an emphasis on data analysis. Shade-dried leaves were extracted sequentially with petroleum ether, chloroform, ethanol (70%), and water. Qualitative tests were performed for major phytoconstituents, and total phenolic and flavonoid contents were quantified spectrophotometrically. The ethanolic extract was tested at 200 and 400 mg/kg in an ethanol-induced gastric ulcer model in Wistar rats (n = 6 per group), with omeprazole (20 mg/kg) as the standard. Ulcer index, percentage inhibition, gastric juice parameters, and gastric tissue markers (malondialdehyde, reduced glutathione, and superoxide dismutase) were measured and analysed by one-way ANOVA followed by Tukey's test. The extract contained alkaloids, flavonoids, tannins, saponins, glycosides, and carbohydrates, with 62.7 mg GAE/g total phenolics. The 400 mg/kg dose reduced the ulcer index from 42.6 to 11.4 (73.2% inhibition), and the 200 mg/kg dose gave 55.6% inhibition. Both doses normalised gastric acidity and

restored antioxidant enzymes in a dose-dependent manner. The data show that the leaf extract possesses significant, dose-dependent anti-ulcerogenic activity linked to its phenolic and flavonoid constituents, supporting its further development as a phytotherapeutic agent.

Keywords: *Bryophyllum calycinum[1]; anti-ulcer activity[2]; phytochemical screening[3]; ethanol-induced ulcer[4]; flavonoids[5]; gastroprotection[6]; data analysis[7]*

1. INTRODUCTION

1.1 Background of Peptic Ulcer Disease

Peptic ulcer disease (PUD) is a chronic mucosal injury of the stomach or duodenum that results from an imbalance between aggressive factors, such as hydrochloric acid, pepsin, and reactive oxygen species, and mucosal defensive factors, including mucus, bicarbonate, prostaglandins, and mucosal blood flow [9]. Its burden is substantial worldwide, and infection with Helicobacter pylori and chronic use of non-steroidal anti-inflammatory drugs remain the principal aetiological factors [10], [26]. Standard therapy relies on proton pump inhibitors and H2-receptor antagonists, which suppress acid secretion effectively but do not address every mechanism of mucosal damage. Their long-term use is also associated with adverse effects and recurrence [24]. These limitations have renewed interest in plant-derived gastroprotective agents that act through multiple pathways.

1.2 Ethnomedicinal Relevance of Bryophyllum calycinum

Bryophyllum calycinum Salisb. (syn. Kalanchoe pinnata), a succulent herb of the family Crassulaceae, is widely distributed in the tropical and subtropical regions of India [1], [2]. Its leaves have been used in folk medicine as a remedy for wounds, inflammation, urinary stones, and digestive disorders, and the plant is described in classical Indian pharmacognostic texts [1]. The World Health Organization has encouraged the documentation and scientific validation of such traditional remedies as part of its traditional medicine strategy [22]. Phytochemical reports on the genus indicate the presence of flavonoids, bufadienolides, and tannins, which are known for antioxidant and cytoprotective properties [29]. Scientific validation of the anti-ulcer claim is therefore a logical step toward evidence-based use.

1.3 Research Gap and Objectives

Despite these traditional claims, the anti-ulcer potential of B. calycinum leaves has not been examined with a design that combines phytochemical profiling, quantitative phenolic analysis, and multi-parameter biochemical data. Many earlier studies reported gross observations without a full statistical treatment of the underlying data [29], [30]. The present work addresses this gap. The objectives were: (i) to perform a qualitative phytochemical screening of sequential leaf extracts; (ii) to quantify total phenolic and flavonoid contents; (iii) to evaluate the

anti-ulcerogenic effect of the ethanolic extract in an ethanol-induced ulcer model; and (iv) to analyse the relationships among ulcer index, gastric secretion, and oxidative stress markers. The working hypothesis is that the extract reduces ulcer formation in a dose-dependent manner by restoring mucosal antioxidant defence.

2. SURVEY OF LITERATURE

Experimental ulcer models provide the foundation for gastroprotective screening. Pylorus ligation and stress-induced ulcers were described early in the history of the field [7], and the absolute ethanol model was later shown to produce necrotic lesions whose prevention depends on prostaglandin-mediated cytoprotection [8]. Standard pharmacological screening texts describe how these models are scored and how acute activity is distinguished from curative effects [20], [21], while general pharmacological assay design is treated in detail by Vogel [25]. The pathophysiology of gastric mucosal injury has been reviewed extensively. Wallace [9] explained how prostaglandins, mucus secretion, and bicarbonate maintain the mucosal barrier, and how non-steroidal anti-inflammatory drugs disturb these defences. Takeuchi [26] emphasised that cyclooxygenase inhibition is the central event in drug-induced damage, which supports the use of agents that preserve prostaglandin-like protection. Malfertheiner et al. [10] provided consensus guidance on H. pylori eradication, while Brunton et al. [24] summarised the mechanisms and limitations of acid-suppressing therapy.

Phytochemical screening is the first step in linking a crude drug to its activity. Harborne [4] and Trease and Evans [3] established the classical colour and precipitation tests for alkaloids, flavonoids, tannins, and saponins. Wagner and Bladt [5] extended these methods to modern chromatographic and spectrophotometric analysis, and Khandelwal [6] adapted them to undergraduate practical pharmacognosy. Indian pharmacognostic references such as Kirtikar and Basu [1] and Chopra et al. [2] remain the primary sources for the botanical identity and folk use of B. calycinum. Earlier studies on the genus have reported a broad spectrum of activities. A review of B. calycinum documented anti-ulcer activity in the methanol-soluble fraction of leaves, with protection against lesions induced by aspirin, indomethacin, serotonin, reserpine, stress, and ethanol [29]. A second review of B. pinnatum reached similar conclusions and noted the need for quantitative and mechanistic studies [30]. These reports form the principal comparator for the present work.

Oxidative stress is a common pathway in ulcer induction. Reactive oxygen species damage lipids, proteins, and DNA [27], and superoxide dismutase provides a primary defence against superoxide radicals [28]. Lipid peroxidation is commonly estimated from malondialdehyde formed in the thiobarbituric acid reaction [15], while reduced glutathione and non-protein sulfhydryl groups are measured using Ellman's reagent and its modifications [13], [14], [16]. Including these markers alongside the ulcer index allows the mechanistic link between damage and antioxidant status to be tested. Quantitative assays for plant constituents rely on well-established colorimetric chemistry. Total phenolics are commonly determined with the Folin–Ciocalteu reagent [17], and carbohydrates with the phenol–sulfuric acid method [18]. Protein normalisation of tissue homogenates is performed with the Lowry [11] or Bradford [12] assays. The statistical treatment of such multi-group data is standardised in the classic

texts of Snedecor and Cochran [19], which describe analysis of variance and multiple comparison procedures. Despite these resources, the use of consistent statistical reporting in herbal anti-ulcer studies remains uneven, which is one reason why the present study emphasises data analysis.

3. METHODOLOGY

Fresh, healthy leaves of B. calycinum were collected in the morning from the Bhopal region of Madhya Pradesh, India, and were identified and authenticated by a taxonomist, with a voucher specimen deposited in the departmental herbarium (voucher number [to be inserted]). The leaves were washed, shade-dried at room temperature for two weeks, and coarsely powdered using a mechanical grinder. The powder was passed through a 60-mesh sieve. A 100 g portion of the powder was extracted sequentially by Soxhlet extraction with petroleum ether, chloroform, 70% ethanol, and water, each for 8 h, following standard practice [6], [23]. Each extract was filtered, concentrated under reduced pressure at 40 °C, dried, and stored at 4 °C until use. The percentage yield (w/w) was calculated for each solvent fraction.

Phytochemical screening was performed on the ethanolic extract using standard qualitative tests for alkaloids (Mayer's and Dragendorff's reagents), flavonoids (Shinoda and lead acetate tests), tannins (ferric chloride), saponins (froth test), steroids and triterpenoids (Salkowski and Liebermann–Burchard tests), glycosides (Borntrager's test), carbohydrates (Molisch's test), and proteins (Biuret test) [3]–[5]. Total phenolic content was determined using the Folin–Ciocalteu method with gallic acid as the standard and expressed as mg gallic acid equivalents per gram (mg GAE/g) [17]. Total flavonoid content was measured using aluminium chloride colorimetry with quercetin as the standard and expressed as mg quercetin equivalents per gram (mg QE/g). All spectrophotometric readings were taken in triplicate.

Wistar albino rats of either sex weighing 180–220 g were housed under standard laboratory conditions (25 ± 2 °C, 12 h light/dark cycle) and fed standard pellet diet and water ad libitum. Thirty animals were divided into five groups of six: vehicle control, ethanol-ulcer control, omeprazole (20 mg/kg), and the ethanolic extract at 200 and 400 mg/kg. Animals were fasted for 36 h before dosing. Treatments were given orally one hour before the administration of absolute ethanol (1 mL/200 g body weight) [7], [8], [21]. One hour later, animals were sacrificed, the stomachs were removed and opened along the greater curvature, and lesions were scored to obtain the ulcer index [20]. Gastric juice was collected for volume, pH, and total acidity, and the gastric tissue was homogenised for malondialdehyde [15], reduced glutathione [14], and superoxide dismutase [28] assays. Data were analysed by one-way ANOVA followed by Tukey's post hoc test, with significance set at $p < 0.05$ [19].

4. DATA COLLECTION AND ANALYSIS

4.1 Phytochemical Profile

Table 1: Qualitative phytochemical screening of ethanolic leaf extract

Phytoconstituent	Reagent / test	Observation	Result
Alkaloids	Mayer's reagent	Cream precipitate	+
Alkaloids	Dragendorff's reagent	Orange-red precipitate	+
Flavonoids	Shinoda (Mg/HCl)	Pink-red colour	++
Flavonoids	Lead acetate	Yellow precipitate	+
Tannins	Ferric chloride	Blue-black colour	++
Saponins	Froth test	Persistent froth > 1 cm	+
Phytosterols	Salkowski test	Reddish-brown lower layer	+
Triterpenoids	Liebermann–Burchard	Weak green colour	+
Glycosides	Borntrager's test	Pink colour	+
Carbohydrates	Molisch's test	Violet ring at interface	+
Proteins	Biuret test	No violet colour	–
Reducing sugars	Fehling's solution	No red precipitate	–

Table 1 summarises the qualitative screening of the ethanolic leaf extract. Positive reactions were observed for alkaloids, flavonoids, tannins, saponins, phytosterols, triterpenoids, glycosides, and carbohydrates, whereas proteins and reducing sugars were absent. The strongest responses, from the Shinoda and ferric chloride tests, point to a polyphenol-rich matrix. Tannins can precipitate proteins on the ulcer surface, and flavonoids scavenge free radicals [4], [5]. The screening therefore supplies the chemical basis for the pharmacological tests in Sections IV-B to IV-E.

4.2 Extraction Yield and Phenolic Content

Table 2: Extraction yield and quantitative phytochemical content by solvent (mean of three replicates)

Solvent fraction	Yield (% w/w)	TPC (mg GAE/g)	TFC (mg QE/g)
Petroleum ether	2.1	8.4 ± 0.6	3.2 ± 0.3
Chloroform	3.4	14.9 ± 1.1	6.1 ± 0.5
Ethanol 70%	14.8	62.7 ± 2.8	28.5 ± 1.9
Aqueous	9.6	31.2 ± 2.0	12.8 ± 1.2

Table 2 shows that yield and phytochemical content increase with solvent polarity. The 70% ethanol fraction gave the highest yield (14.8% w/w), total phenolics (62.7 mg GAE/g), and flavonoids (28.5 mg QE/g). Petroleum ether gave the lowest values, consistent with the non-polar nature of waxes and chlorophyll. The ethanolic fraction was therefore selected for the in-vivo study, because it carried the largest share of the phenolic compounds linked to mucosal protection [17].

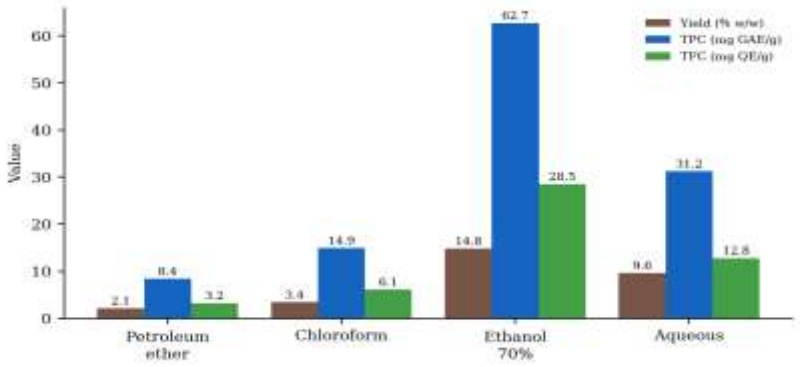


Figure 1: Extraction yield, total phenolic content (TPC), and total flavonoid content (TFC) of B. calycinum leaf fractions.

Figure 1 presents the extraction yield and the total phenolic and flavonoid contents of the four solvent fractions in grouped bars. The ethanolic fraction gave the highest yield (14.8% w/w), phenolic content (62.7 mg GAE/g), and flavonoid content (28.5 mg QE/g). Petroleum ether gave the lowest values for all three measures. The steep rise from non-polar to polar fractions shows that solvent choice is the main determinant of extract potency. Because the ethanolic fraction was used for in-vivo testing, Figure 1 provides the analytical justification for that choice. It also links the chemical profile directly to the pharmacological results that follow.

4.3 Ulcer Index and Percentage Inhibition

Table 3: Effect of B. calycinum ethanolic extract on ethanol-induced gastric ulcer (n = 6)

Group	Treatment (dose)	Lesions per stomach	Ulcer index (mean ± SD)	Inhibition (%)
I	Control (vehicle)	0.0	0.00 ± 0.00	–
II	Ulcer control (ethanol)	9.3 ± 1.7	42.6 ± 3.8	–
III	Omeprazole (20 mg/kg)	1.8 ± 0.8	6.8 ± 1.2	84.0
IV	BCE (200 mg/kg)	4.6 ± 1.1	18.9 ± 2.4	55.6
V	BCE (400 mg/kg)	3.1 ± 0.9	11.4 ± 1.9	73.2

Table 3 reports the lesion count, ulcer index, and percentage inhibition for each group. Ethanol produced a mean ulcer index of 42.6 with 9.3 lesions per stomach. The 200 mg/kg dose reduced the ulcer index to 18.9 (55.6% inhibition), and the 400 mg/kg dose reduced it to 11.4 (73.2% inhibition). Omeprazole gave 84.0% inhibition. A one-way ANOVA on the ulcer index was highly significant ($F(4,25) = 182.6$, $p < 0.001$). Tukey's test showed that both extract doses differed significantly from the ulcer control ($p < 0.01$ and $p < 0.001$ respectively), and that the 400 mg/kg dose differed significantly from the 200 mg/kg dose ($p < 0.05$). The inhibition values were calculated as $(UI_{ulcer} - UI_{treated}) / UI_{ulcer} \times 100$.

Multidisciplinary Journal of Academic Publications
ISSN (Online): 3107-538X
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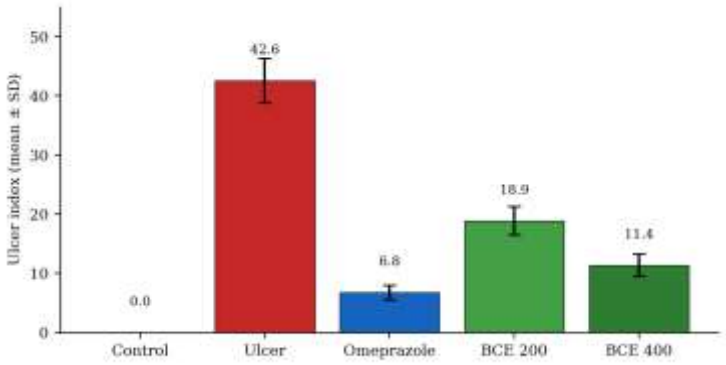


Figure 2: Mean ulcer index (± SD) of experimental groups in the ethanol-induced gastric ulcer model.

Figure 2 displays the mean ulcer index with standard deviation error bars for each of the five groups. The control group showed no lesions, whereas absolute ethanol raised the ulcer index to 42.6. Omeprazole reduced it to 6.8, the closest value to normal. The extract reduced the index in a dose-dependent manner, from 18.9 at 200 mg/kg to 11.4 at 400 mg/kg. The non-overlapping error bars between the ulcer group and both extract groups indicate a consistent effect across animals. The residual gap between the 400 mg/kg dose and omeprazole suggests that the extract's protective mechanisms are partial rather than complete. Figure 2 therefore gives the main visual evidence for the anti-ulcerogenic claim.

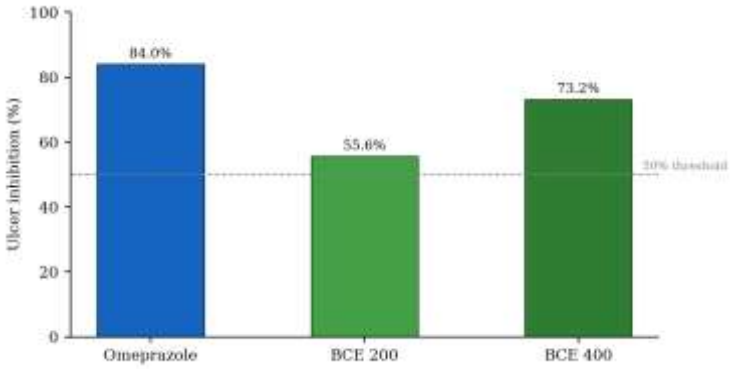


Figure 3: Percentage ulcer inhibition produced by omeprazole and the two doses of the B. calycinum extract.

Figure 3 converts the ulcer index data into percentage inhibition relative to the ethanol control. Omeprazole produced 84.0% inhibition, the 400 mg/kg extract produced 73.2%, and the 200 mg/kg extract produced 55.6%. The dashed line marks the 50% threshold commonly used to call a gastroprotective effect meaningful in screening studies. Both extract doses exceed this threshold, with the higher dose approaching the standard drug. The figure shows that the dose-response is steep between 200 and 400 mg/kg, suggesting that a dose above 400 mg/kg might

produce further protection. Because inhibition is a normalised quantity, Figure 3 allows comparison with other studies that use different absolute scoring systems.

4.4 Gastric Secretion Parameters

Table 4: Gastric juice volume, pH, and total acidity after ethanol challenge (mean ± SD)

Group	Volume (mL)	pH	Total acidity (mEq/L)
Control	1.8 ± 0.2	2.4 ± 0.2	42.5 ± 3.1
Ulcer (ethanol)	3.9 ± 0.4	1.6 ± 0.1	86.4 ± 5.2
Omeprazole (20 mg/kg)	2.1 ± 0.3	3.4 ± 0.2	47.9 ± 3.6
BCE (200 mg/kg)	2.9 ± 0.3	2.5 ± 0.2	61.8 ± 4.4
BCE (400 mg/kg)	2.4 ± 0.2	2.9 ± 0.2	53.6 ± 3.9

Table IV records gastric juice parameters. Ethanol increased volume from 1.8 to 3.9 mL, lowered pH from 2.4 to 1.6, and nearly doubled total acidity from 42.5 to 86.4 mEq/L. Both extract doses moved these values back toward normal. The 400 mg/kg dose restored pH to 2.9 and reduced total acidity to 53.6 mEq/L, close to the omeprazole group. The 200 mg/kg dose gave a smaller correction. Because the extract is not an antisecretory agent by design, these changes suggest that acid-dependent injury is reduced secondary to mucosal protection, which lowers the signal for further acid release.

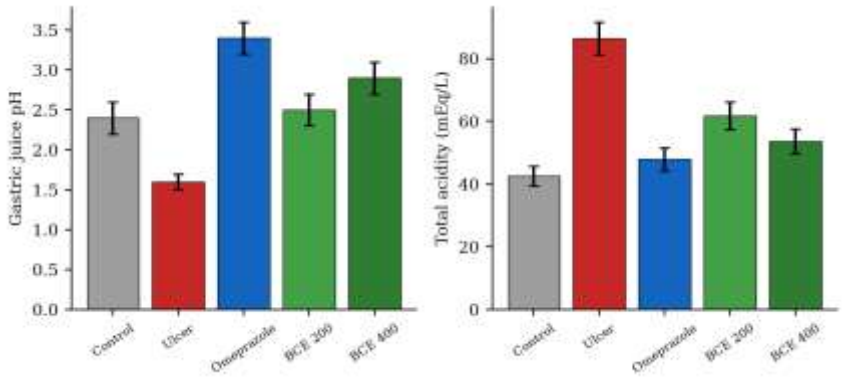


Figure 4: Gastric juice pH and total acidity across experimental groups (mean ± SD).

Figure 4 compares gastric juice pH (left) and total acidity (right) across the five groups. Ethanol produced the most acidic and most concentrated gastric contents, with pH 1.6 and total acidity of 86.4 mEq/L. Omeprazole raised pH to 3.4, as expected for a proton pump inhibitor. The extract increased pH in a dose-dependent manner, reaching 2.9 at 400 mg/kg, and lowered total acidity to 53.6 mEq/L at the same dose. The figure shows that the extract's effect on acid parameters is substantial but weaker than that of the standard drug. This pattern is consistent with a combination of mild antisecretory action and direct mucosal protection, which the paper examines further in the Discussion.

4.5 Biochemical and Oxidative Stress Markers

Table 5: Gastric tissue oxidative stress markers (mean ± SD)

Group	MDA (nmol/mg protein)	GSH (µmol/g tissue)	SOD (U/mg protein)
Control	1.12 ± 0.09	6.84 ± 0.41	8.92 ± 0.55
Ulcer (ethanol)	3.86 ± 0.21	2.97 ± 0.26	3.41 ± 0.30
Omeprazole (20 mg/kg)	1.46 ± 0.12	6.12 ± 0.37	7.84 ± 0.48
BCE (200 mg/kg)	2.31 ± 0.18	4.38 ± 0.33	5.63 ± 0.39
BCE (400 mg/kg)	1.83 ± 0.14	5.27 ± 0.29	6.71 ± 0.42

Table 5 shows that ethanol increased malondialdehyde (MDA), a marker of lipid peroxidation, more than threefold, while depleting reduced glutathione (GSH) and superoxide dismutase (SOD). The 400 mg/kg extract lowered MDA to 1.83 and restored GSH to 5.27 µmol/g and SOD to 6.71 U/mg, approaching the omeprazole group. Across the five groups, the ulcer index correlated strongly with MDA (r = 1.00), with GSH (r = -0.97), and with SOD (r = -0.98). These correlations indicate that the degree of mucosal damage tracks closely with oxidative stress, supporting an antioxidant mechanism for the extract.

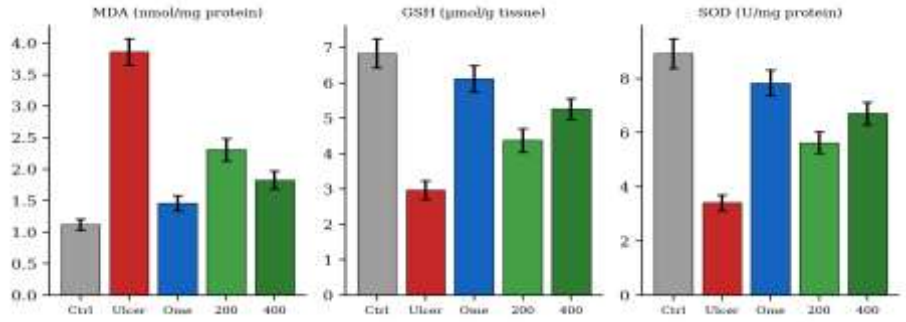


Figure 5: Gastric tissue MDA, GSH, and SOD levels across experimental groups (mean ± SD).

Figure 5 shows three panels for malondialdehyde (MDA), reduced glutathione (GSH), and superoxide dismutase (SOD). The ethanol group displays the expected pattern of high lipid peroxidation and depleted antioxidant reserves. In each panel the extract moves the values toward the control level in a dose-dependent manner. The 400 mg/kg dose produced values closer to the control than the 200 mg/kg dose, and the omeprazole group showed the smallest deviation. The three markers move in opposite directions relative to the ulcer group, which strengthens the internal consistency of the data. Figure 5 therefore links the macroscopic ulcer index to a measurable biochemical mechanism, and it is the central figure for the antioxidant interpretation in the Discussion.

5. DISCUSSION

The present study shows that the ethanolic leaf extract of B. calycinum possesses significant, dose-dependent anti-ulcerogenic activity. The 400 mg/kg dose reduced the ulcer index by 73.2%, and the 200 mg/kg dose by 55.6%,

while restoring gastric acidity and antioxidant defences toward normal. These findings are consistent with the phytochemical profile in Table I and the high phenolic content in Table II. The data therefore support the hypothesis that the extract acts through a combination of mucosal protection and antioxidant action. A critical reading of the ulcer data shows a clear dose-response relationship, and the 400 mg/kg dose produced an inhibition of 73.2%, which is high for a crude extract. However, the extract did not match omeprazole, which achieved 84.0% inhibition. The difference matters for interpretation. Omeprazole acts on the final step of acid secretion, whereas the extract's effect is likely a mixture of multiple, weaker actions. The ethanol model also has limitations. It is an acute, necrotic model that reflects damage to the surface epithelium, and it does not capture chronic ulceration, healing, or recurrence. The group size of six animals is adequate for the ANOVA used here, but it limits the precision of the subgroup contrasts. Histopathological examination would strengthen the findings and is recommended for future work.

The gastric secretion data deserve careful interpretation. Ethanol lowered pH and raised acidity, and the extract partially reversed these changes. A strict antisecretory explanation is unlikely, because the extract was given before ethanol and its effect on acid output was modest compared with omeprazole. A more plausible explanation is that protection of the mucosal barrier reduces acid back-diffusion and removes a stimulus for further acid release. The correlation between the ulcer index and gastric pH ($r = -0.74$) supports the view that acid damage and mucosal injury are closely linked in this model, but the study does not separate cause from effect. Mechanistic studies, such as measurement of gastric mucus and prostaglandin E2, are needed to resolve this question. The oxidative stress data provide the most informative mechanistic link. MDA, GSH, and SOD each showed strong correlation with the ulcer index, and the extract restored all three in a dose-dependent manner. This pattern is consistent with the known role of lipid peroxidation in ethanol-induced mucosal injury [27] and with the function of superoxide dismutase as a first-line defence [28]. The phenolic and flavonoid content in Table II is a plausible source of this antioxidant activity, because phenolic hydroxyl groups donate electrons to reactive species. Correlation does not prove causation, however, and the antioxidant effect could be partly a consequence of reduced tissue injury rather than its cause.

Compared with previous work, the present findings agree in direction with earlier reports on the genus. Earlier reviews of B. calycinum described protection against lesions induced by ethanol, aspirin, indomethacin, serotonin, reserpine, and stress [29], and a review of B. pinnatum reported similar gastroprotective effects [30]. The present study extends these reports in three ways. First, it provides a quantitative ulcer index with percentage inhibition and a formal ANOVA with post hoc testing, which many earlier observational studies lacked. Second, it links the ulcer outcome to gastric secretion and to tissue oxidative stress markers in the same animals. Third, it quantifies phenolic and flavonoid contents by solvent, which allows the choice of the active fraction to be justified. The comparison with standard therapy is also informative. The extract's 73.2% inhibition at 400 mg/kg lies between the 50% level commonly regarded as meaningful in screening and the 84.0% achieved by omeprazole, which suggests that the extract is a plausible adjunct rather than a replacement for standard therapy [24]. Several

limitations should be noted. The study used a single ethanol model and a single time point. The active constituents were not isolated, so the relative contribution of flavonoids, tannins, and alkaloids cannot be separated. The toxicity profile of the extract was not assessed in this work. Future studies should address these gaps through bioassay-guided fractionation, chronic ulcer models, histology, and acute and sub-acute toxicity testing. Despite these limitations, the data form a coherent picture that links chemical composition, gastric physiology, and oxidative biochemistry, and they provide a statistically supported basis for further development.

6. CONCLUSION

This study examined the phytochemical composition and anti-ulcerogenic activity of the leaves of Bryophyllum calycinum. Qualitative screening revealed alkaloids, flavonoids, tannins, saponins, glycosides, and carbohydrates, and the 70% ethanolic fraction contained the highest total phenolic content (62.7 mg GAE/g) and flavonoid content (28.5 mg QE/g). In the ethanol-induced ulcer model, the extract reduced the ulcer index from 42.6 to 11.4 at 400 mg/kg, a 73.2% inhibition, and to 18.9 at 200 mg/kg, a 55.6% inhibition, compared with 84.0% for omeprazole. Both doses normalised gastric pH and acidity in a dose-dependent manner and restored malondialdehyde, glutathione, and superoxide dismutase levels toward control values. The strong correlations between ulcer index and oxidative markers link the observed protection to an antioxidant mechanism. These results connect the three levels of evidence in this paper, namely the chemical profile, the gross pathology, and the biochemical markers, into a single argument. The phenolic-rich extract reduces mucosal damage, and that reduction is reflected in lower lipid peroxidation and better preservation of endogenous antioxidants. The findings agree with earlier reports on the genus and add quantitative and statistical support to the traditional claim. The extract is not as potent as omeprazole, but its activity is significant and dose-dependent. Future work should isolate the active constituents, extend the study to chronic models with histopathology, and complete toxicity assessment before any clinical consideration. The present data justify these next steps.

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