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Research Article

PROTECTIVE EFFECT OF CLEOME VISCOSA LEAVES ON ACETIC ACID INDUCED COLITIS IN RATS

Atifa Ahmed[1] and MangalaLahkar[2]

[1]Department of Pharmacology, Tezpur Medical College and Hospital, Tezpur: 784010, Assam, India
[2]Department of Pharmacology, Gauhati Medical College and Hospital, Guwahati: 781032, Assam, India

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ABSTRACT

Objective: To evaluate the effect of ethanolic extract of Cleome viscosa leaves on acetic induced colitis in rats.

Materials and methods: Animals were divided into six groups with six animals in each group. Group I was taken as normal control, Group II-VI , were administered 0.2ml of 10% acetic acid intrarectally, following which Group III received sulfasalazine (360mg/kg) and GroupIV, Group V, Group VI received ethanolic extract of Cleome viscosa in the doses of 100, 200, 400mg/kg respectively for 14 days. Then the animals were sacrificed and the distal colon of the different groups was dissected out and the mucosal damage was evaluated based on their weight (gms), macroscopic score and histopathological score.

Results: The results showed that there was statistically significant decrease in mucosal damage in all the three groups receiving ethanolic extract of Cleome viscosa leaves as compared to Group II, the disease control. However the mucosal damage was least in Group III, which received sulfasalazine as a standard drug for IBD.

Conclusion: Ethanolic extract of Cleome viscosa leaves has been found to reduce both microscopic as well as macroscopic damage of the colonic mucosa following acetic acid induced colitis in rats.

INTRODUCTION

Inflammatory bowel disease (IBD) is a spectrum of chronic, idiopathic, inflammatory intestinal conditions that causes significant gastrointestinal (GI) symptoms that include diarrhoea, abdominal pain, bleeding, anaemia, and weight loss. IBD also is associated with a variety of extraintestinal manifestations, including arthritis, ankylosing spondylitis, sclerosing cholangitis, uveitis, iritis, pyodermagangrenosum, and erythema nodosum.[1]

Inflammatory bowel diseases, ulcerative colitis and crohn's disease, represents chronic alteration of the gastrointestinal tract of unknown etiology perhaps involving immunological events. The immunological parameters have been described as secondary but may possibly be attributed to the chronicity of the disease.[2] A consensus hypothesis is that each of these three major host compartments that function together as an integrated "supraorganism" (microbiota, IECs, and immune cells) are affected by specific environmental (e.g., smoking, antibiotics, enteropathogens) and genetic factors that, in a susceptible host, cumulatively and interactively disrupt homeostasis, which in so doing culminates in a chronic state of dysregulated inflammation; that is IBD. [3]

When fully developed, crohn's disease is characterized by (1) sharply delimited and typically transmural involvement of the bowel by an inflammatory process with mucosal damage,(2) the presence of noncaseating granulomas in 40% to 60% of cases, and (3) fissuring with formation of fistulae. A classical feature of crohn's disease is the sharp demarcation of diseased bowel segments from adjacent uninvolved bowel. [4]Ulcerative colitis involves the rectum and sigmoid and may involve the entire colon. Presentation with an even higher proximal extension (pancolitis) occurs much less frequently. Colonic involvement is continuous from the distal colon, so that skip lesions are not encountered. [4] Current drug treatment is aimed to induce and then maintain the patient in remission and ameliorate the disease's secondary effects rather than modify or reverse the underlying pathogenic mechanism. However, the management of inflammatory bowel disease with the use of conventional treatment is expensive and also associated with number of side effects. [5] *CleomeviscosaLinn*. (Capparidaceae), commonly known as "wild or dog mustard," is an annual, sticky herb found as a common weed all over the plains of India and throughout the tropics of the world.Following the various traditional claims for the use of *C.Viscosa*(CV) as a cure of numerous diseases ,considerable efforts have been made by researchers to verify its utility through scientific pharmacological screenings.The pharmacological studies have shown that CV possess various notable biological activities such as antihelminthic, antimicrobial, analgesic, antiinflammatory, immunomodulatory, antipyretic, psychopharmacological, antidiarrhoeal, and hepatoprotective activities. A wide variety

of phytoprinciples have been isolated from the plant.[6] Its seeds are occasionally used as remedy for fever and diarrhoea, and powdered roots are put on the lips by Santhal tribes to restore consciousness in people who fainted. Leaves and its juice are applied externally on wounds and ulcer. The smoke from its leaves is used by the locals to repel mosquitos at night.Its extract exhibited larvicidal activity against the 2nd and 4th instar larvae of *Anophelesstephensi*, a vector of malaria in India.[7]In this study, we tried to evaluate the anti-inflammatory effect of *Cleomeviscosa* leaves in experimentally induced IBD in experimental animals.

MATERIALS AND METHODS

The present study was conducted in the Department of Pharmacology, Gauhati Medical College and Hospital, Guwahtai. The anti-inflammatory effect of ethanolic extract of the leaves of *Cleome viscosa* was studied in experimentally induced inflammatory bowel disease in albino rats.

Ethical review

Experimental protocol was approved by the Institutional Animal Ethics Committee (IAEC) of Gauhati Medical College and Hospital, Guwahati (MC/05/2015/19). The study was performed in accordance to the CPCSEA guidelines.

Plant material

The leaves of ***Cleome viscosa*** were collected from Narakasur hill top, Guwahati, Assam during the month of June, 2015. Authentication of the plant was done by Department of Botany, Gauhati University and a voucher specimen was preserved for further reference.

Extraction of plant material

The leaves were thoroughly washed, shade dried and then chopped to coarse powder using a mixer grinder. Powder (200 gram) was tightly packed in Soxhlet apparatus and subjected for continuous hot percolation employing ethanol as solvent for 5 days at a temperature of 40-60°C using a heating mantle.

Experimental animals

Adult wister rats of either sex weighing between 150-250 gm were procured from the animal house of Department of Pharmacology, Gauhati Medical College and Hospital. The animals were housed in standard laboratory conditions at 25°C and 12 hours light and dark cycle. Animals were given free access to rat chow diet and water *ad libitum.* The experimental animals were acclamatized to laboratory conditions for seven days before starting the experiment.

Acute toxicity tests

The acute toxicity study was carried out as per OECD guidelines 425.

Drugs and chemicals

Drugs and chemicals needed for the present study are sulfasalzine (Cadila), Glacial acetic acid obtained from Fisher Scientific Ltd.

Experimental design

Total 36 animals were included in the study. Animals were divided into six groups I, II, III, IV, V and VI.
Groups are:

GroupI Normal Control group, received normal saline at the dose of 10mg/kg
GroupII Disease control, 0.2ml of 10% acetic acid
GroupIII Standard group, 0.2ml of 10% acetic acid + Sulfasalazine 360mg/kg[8]
GroupIV 0.2ml of 10% acetic acid + Ethanolic extract of *C.viscosa* 100mg/kg
GroupV 0.2ml of 10% acetic acid + Ethanolic extract of *C.viscosa* 200mg/kg
GroupVI 0.2ml of 10% acetic acid + Ethanolic extract of *C.viscosa* 400mg/kg

Induction of colitis

A model of diffuse colitis in rats induced by intraluminal colonic instillation or serosal application of dilute acetic acid was described by MacPharson and Pfeiffer (1976, 1978) [9,10] Fitzpatrick *et al.* (1990) [11] tested the anti-inflammatory effects of various drugs on acetic acid induced colitis in the rat.

Briefly, rats were anaesthetized with ketamine (5-10mg/kg i.p) [12] following 24hr fast, a soft 6F paediatric catheter lubricated with lignocaine jelly was inserted rectally into the colon through anus such that tip is 8cm proximal to anus, approximately at the spenic flexure.Then 0.2ml of 10% acetic acid was introduced into the colon. After 10s contact *in situ*, the remaining acid is withdrawn and the lumen washed with three successive 0.5ml volumes of isotonic saline. Test drugs are administered daily during the following days. Diffuse colonic lesions appear after 3 days in control animals and the animals experience bloody diarrhoea. An initial mucosal inflammation develops into submucosaledema; petechial haemorrhages become enlarged with subsequent neutrophil invasion, and pseudopolyps become evident. [2]

Colon weight change

After induction of colitis and drug treatment, colon weight changes were calculated.

Assessment of colonic damage

After 14 days of administration of extract/drug, animals were euthanized by ether and 10cm of distal colon was removed from surrounding tissues, opened longitudinally along its mesenteric border, rinsed, and processed for histology. After washing the mucosa with saline solution, mucosal injury (macroscopically) was assessed using the grading scale of Morris *et al.* (1989). [13]

Score 0 - No damage.
Score 1 - localized hyperaemia but no ulcer
Score 2 - linear ulcers with no significant inflammation
Score 3 - linear ulcers with inflammation at one side
Score 4 - two or more sites of ulceration and inflammation
Score 5 - two or more sites of ulceration and inflammation or one major site of inflammation and ulceration extending > 1cm along the length of the colon.

Histopathological examination[14]

The histopathological examination of the thesis work was done in the Department of Pathology, Gauhati Medical College and Hospital.

The histopathological grading was done using modified model of Wei *et al.* [15] which is as follows:

1 the infiltration of acute inflammatory cells:
0-no,
1-mild increasing,
2-severe increasing;
2 the infiltration of chronic inflammatory cells:
0-no,
1-mild increasing,
2-severe increasing;
3 the deposition of fibrin protein:
0-negative,
1-positive;
4 the submucosaedema:
0-no,
1-patchy-like,
2 fusion-like;
5 the epithelium necrosis:
0-no,
1-limiting,
2-widening;
6 the epithelium ulcer:
0-negative,
1-positive.

Statistical analysis

Data were expressed as mean ± SEM. Results were analyzed by one way analysis of variance (ANOVA), followed by Dunnet's multiple comparison test. P value < 0.05 was considered statistically significant. All the data were analyzed using Graph pad Prism Version 5.

RESULTS AND OBSERVATIONS

The degree of colonic damage has been assessed by three parameters: colon weight (gms), macroscopic score and histopathological score. Results are presented as mean ± standard error of mean (SEM) of 6 animals in each group. Results obtained are summarized in Table 1-4.

Table 1 Colon weight change (gms)

Groups	Treatment	Mean±SEM
I	Normal saline	4.83±0.1498*
II	Acetic acid treated	9.81±0.1536
III	Sulfasalazine (360mg/kg)	5.13±0.1201*
IV	EECV (100mg/kg)	8.05±0.1648*
V	EECV (200mg/kg)	7.15±0.1204*
VI	EECV (400mg/kg)	6.15±0.1176*

*p<0.05 when compared with acetic acid group.

Table 2 Macroscopic grading

Groups	Treatment	Mean±SEM
I	Normal saline	0±0*
II	Acetic acid treated	4.83±0.1666
III	Sulfasalazine (360mg/kg)	1.66±0.3333*
IV	EECV (100mg/kg)	3.83±0.1666*
V	EECV (200mg/kg)	3.16±0.1666*
VI	EECV (400mg/kg)	2.16±0.1666*

* p<0.05 when compared with acetic acid group.

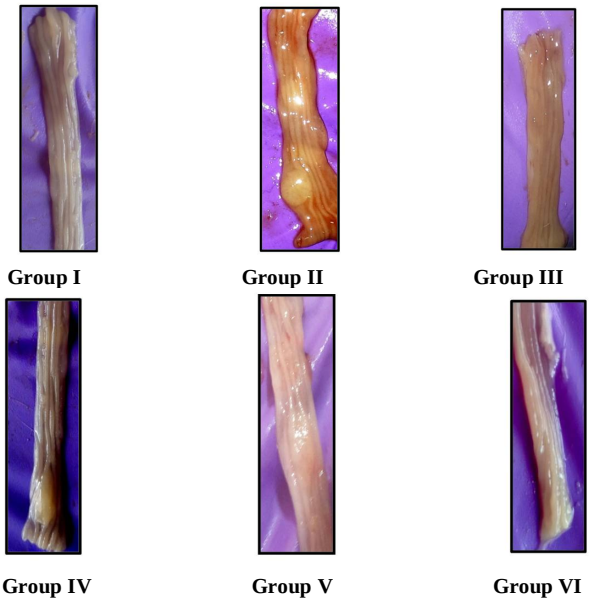

Fig 1 Gross picture of rat colon in different groups [It shows that the severe inflammation in Group II has significantly reduced with increasing doses of EECV (Group IV-VI)]

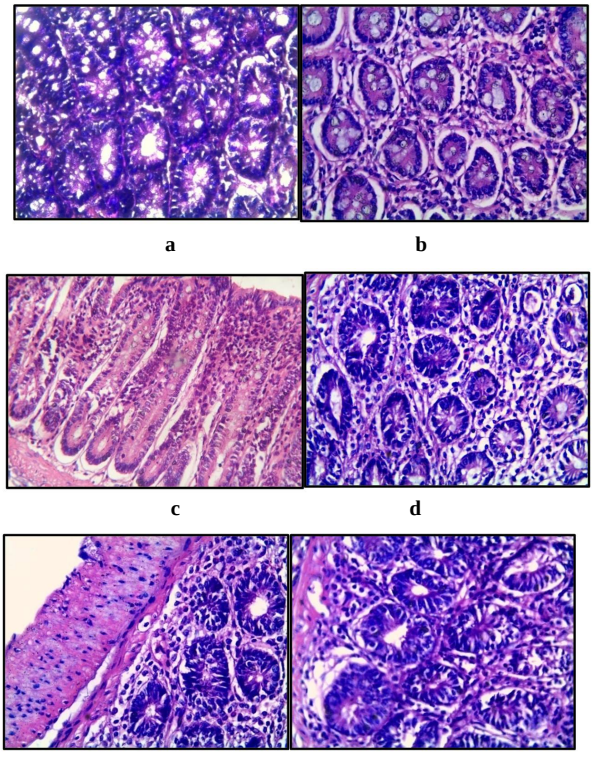

Fig 2 Histological sections of rat ileum stained with H & E [(a): Normal intact mucosa, (b): Disease control showing massive necrosis and inflammatory infiltrate, (c): Sulfasalazine treated sections with much improved histology, (d), (e), (f): EECV 100,200,400 mg/kg, treatment with the plant extract have attenuated the extent and severity of damage]

Table 3 Histopathological score

	Acute inflammatory cell infiltration	Chronic inflammatory cell infiltration	Fibrin deposition	Submucosaledema	Epithelial necrosis	Epithelial ulcer
Group I	0±0*	0±0*	0±0*	0±0*	0±0*	0±0*
Group II	1.66±0.2107	2±0	1±0	2±0	1.83±0.1666	1±0
Group III	0.166±0.1666*	0.33±0.2107*	0±0*	0.16±0.1666*	0.16±0.1666*	0±0*
Group IV	0.66±0.2107*	0.83±0.3072*	0.33±0.2107*	0.83±0.3072*	0.83±0.1666*	0.33±0.2107*
Group V	0.5±0.2236*	0.66±0.2107*	0.16±0.1666*	0.66±0.2107*	0.5±0.2236*	0.16±0.1666*
Group VI	0.33±0.2107*	0.5±0.2236*	0±0*	0.33±0.2107*	0.33±0.2107*	0±0*
d.f	5,30	5,30	5,30	5,30	5,30	5,30
One way ANOVA (F)	9.954	12.21	12.71	14.90	14.87	12.71

*p<0.05 when compared with acetic acid group.

Table: 1 shows the mean colon weight (gms).

Table: 2shows the mean macroscopic score.

Table: 3 shows mean scores for the six histopathological parameters (acute inflammatory cell infiltration, chronic inflammatory cell infiltration, fibrin deposition, submucosaledema, epithelial necrosis and epithelial ulcer).

Table: 4 shows the mean total histopathological scores (i.e the sum of the six histopathological parameters) in all the six group of animals.

Table 4 Histopathological score

Groups	Treatment	Mean±SEM
I	Normal saline	0±0*
II	Acetic acid treated	9.5±0.2236
III	Sulfasalazine (360mg/kg)	0.83±0.1666*
IV	EECV (100mg/kg)	3.83±0.4014*
V	EECV (200mg/kg)	2.66±0.3333*
VI	EECV (400mg/kg)	1.5±0.2236*

*p<0.05 when compared with acetic acid group.

DISCUSSION

Plant based traditional medicine system continues to play an essential role in healthcare with about 80% of world's inhabitants relying mainly on it for their primary healthcare.[16] The indigenous knowledge of many traditional communities has been formulated, been documented, and eventually become organized systems of medicine, such as Ayurveda, Siddha, Unani, and other systems outside India.[6] Because of potential adverse effects and lack of effectiveness of standard therapies, the use of complementary and alternative medicines, particularly of herbal therapies, for chronic illness such as diabetes, osteoporosis, cancer and liver diseases is widespread and increasing, and inflammatory bowel disease is not excluded from this trend.[17]

Cleome viscosa Linn. (Cleomaceae) is a weed distributed throughout the tropical regions of the world and plains of India. The plant is an annual sticky herb with a strong penetrating odour, yellow flower and strong slender pods containing seeds. In Ayurvedic system of medicine the plant is used for the treatment of fever, inflammations, liver diseases, bronchitis and diarrhoea. The rural people used the fresh juice of the crushed seeds for the treatment of infantile convulsions and mental disorder. The juice of the plant diluted with water is given internally in small quantities in fever and the leaves are useful in healing wounds and ulcer.[18] The present study has been designed to evaluate the effect of ethanolic extract of *Cleome viscosa* leaves in the treatment of acetic acid induced inflammatory bowel disease in albino rats. In the present study we have taken Group I as normal control, Group II as the disease control, Group III received sulfasalazine (360mg/kg), which is the standard drug , Group IV, V, VI were administered three different doses of plant extract 100mg/kg, 200mg/kg, 400mg/kg. All the animals of Group II-VI , were administered 0.2ml of 10% acetic acid intrarectally, following which Group III received sulfasalazine (360mg/kg) and GroupIV, Group V, Group VI received ethanolic extract of *Cleomeviscosa* in the doses of 100, 200, 400mg/kg respectively for 14 days. Then the animals were sacrificed and the distal colon of the different groups was dissected out and the mucosal damage was evaluated based on colon weight (gms), macroscopic score and histopathological score. The results showed that there was statistically significant decrease in mucosal damage in all the three groups receiving ethanolic extract of *Cleome viscosa* as compared to Group II, the disease control, which did not receive any drug following intrarectal administration of acetic acid. However the mucosal damage was least in Group III, which received sulfasalazine as a standard drug for IBD. Thus the study showed that ethanolic extract of *Cleome viscosa* has potential efficacy in the treatment of inflammatory bowel disease, even though it is lesser than sulfasalazine.

Parimala et al. (2003) [19] evaluated *Cleomeviscosa* for its anti-inflammatory potential against carageenin, histamine, and dextran-induced rat paw edema. Diclofenac sodium (20 mg/kg), a non-steroidal anti-inflammatory agent, was included as a standard for comparison. The results of the study demonstrated significant activity of the extract compared to the reference standard used.

Ravi Kant Upadhyay (2015) also mentioned that the flavonoid glycoside isolated from *C. Viscosa*showed significant antiinflammatory activity oncarrageenan induced rat paw edema (*in vivo*). This antiinflammatory effect of the flavonoid glycoside may bedue to the inhibition of prostaglandin synthesis. Similarly, querection 3-0-(2″-acetyl)-glucoside obtained from ethylacetate (EA) fraction of *C. viscose* showed anti-inflammatoryactivity against carrageenan induced rat pawedema (*in vivo*). Oral administration of coumarinolignoids inhibited the pro inflammatory mediators and enhancesthe production of antiinflammatory mediator in dose dependent manner. [20]*In vivo* and/or *invitro* studies indicate that one of the probable mechanisms of action of anti-IBD effect exerted by natural products is inhibition of pro-inflammatory mediators. [21] Hence, this property exhibited by the phyto principles in *Cleome viscosa* might be responsible for its beneficial role in acetic acid induced IBD in rats as found in the present study.

Gupta et al. (2011) compared the antioxidant activity of extracts of leaf and stem parts of the medicinal plant, *Cleome viscosa*, with respect to its phenols and flavonoids content. The study revealed that leaves of *Cleome viscosa* have high phenolic and flavonoid content, inhibition of β carotene bleaching (antioxidant activity), reducing power and free radical scavenging activity in comparison to stem.[22] It is now well recognised that reactive oxygen species (ROS) are produced in excess by the inflamed mucosa in inflammatory bowel disease (IBD) and may be pathogenic. The predominant sources of ROS in the inflamed mucosa are probably activated mucosal phagocytic leucocytes and episodes of ischaemia reperfusion. Trials have suggested that specific antioxidant treatment may be therapeutically effective in IBD. [23] Thus, the antioxidant activity exhibited by the phyto principles present in *Cleome viscosa* might have also resulted in healing of mucosal damage in acetic acid induced IBD in rats.

Tiwari et al. (2004) [24] demonstrated the immunomodulatory effect of aqueous and ethanolic extracts of the aerial parts of Cleome viscosa Linn (Capparidacea) in mice. The assessment of immunomodulatory activity was carried out by various haematological and serological tests. Extract of the plant showed significant immunosuppressant activity and administration of extract remarkably decreased the number of WBC and spleenic lymphocytes. It also decreased phagocytic

index and both cellular and humoral antibody response. Since there is defective response in both the innate and adaptive immune systems in IBD, hence the immunomodulatory property of *Cleome viscosa* may also contribute to its beneficial effect in acetic acid induced IBD in rats as found in the present study.

In recent years, ethnomedicinal studies have received much attention, bringing to light the numerous little known and unknown medicinal uses especially of plant origin. They obviously deserve evaluation by modern scientific methods such as phytochemical analysis, biological screenings, and clinical trials. Almost all the parts of *Cleome viscosa* are documented to possess medicinal benefits in ethnobotanical surveys conducted by researchers. [14]

Thus the anti-inflammatory as well as antioxidant and immunomodulatory properties of *Cleome viscosa* may be responsible for its beneficial role in inflammatory bowel disease, as demonstrated in the present study. As there is no scientific data available to substantiate the traditional use of this plant for its use in the treatment of inflammatory bowel disease, the present study was undertaken to investigate the effect of ethanolic extract of *Cleome viscosa*leaves in experimental animal models of IBD.

Further characterization of the plant extract could lead to interesting findings which can provide more accurate details about the mechanisms by which *Cleome viscosa* acts as a protective agent. Thus *Cleome viscosa* may prove to be an important and effective indigenous drug in the future treatment of inflammatory bowel disease.

CONCLUSION

Thus from the above study it can be concluded that ethanolic extract of ***Cleome viscosa***leaves acts by reducing microscopic and macroscopic damage score in colonic mucosa following acetic acid induced inflammatory bowel disease in albino rats. Sulfasalzine, which was taken as the standard drug is however more effective than the different doses of ethanolic extract of ***Cleome viscosa***leaves in reducing the mucosal damage as indicated by decrease in both macroscopic and microscopic score.

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