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FORMULATION AND EVOLUTION OF LORNOXICAM LOADED ETHOSOMAL GEL WITH HERBAL PERMEATION ENHANCER

Mr. Jayesh D. Gavhane[1]*, Mr. Vishal S. Madankar[2], Dr. Sampat D. Navale[3]

[1]Research Scholar, Department of Quality Assurance, Delight College of Pharmacy, Koregaon Bhima, Pune, Maharashtra, India-412216.

2.Assistant Professor, Department of Quality Assurance, Delight College of Pharmacy, Koregaon Bhima, Pune, Maharashtra, India-412216.

3.Principal of Delight College of Pharmacy, Koregaon Bhima, Pune, Maharashtra, India-412216.

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***Corresponding Author**
Mr. Jayesh D. Gavhane
Research Scholar, Department of Quality Assurance, Delight College of Pharmacy, Koregaon Bhima, Pune, Maharashtra, India-412216.

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ABSTRACT

Conventional topical NSAIDs suffer from poor penetration through the stratum corneum, which limits their therapeutic effectiveness in localized inflammatory conditions, whereas oral administration is associated with significant gastrointestinal toxicity. Ethosomal nanocarriers improve transdermal delivery by enhancing vesicle flexibility through ethanol, but their reliance on sonication hinders use in resource‐limited teaching laboratories. The present study developed a lornoxicam‐loaded ethosomal gel using high‐shear homogenization as a validated alternative to sonication, and systematically incorporated ginger oleoresin as a biocompatible penetration enhancer to optimize the management of arthritis. Ethosomes composed of soya lecithin and ethanol were prepared using the cold injection method, followed by size optimization through homogenization, and then incorporated into an aqueous gel matrix based on Carbopol 940. The formulation was comprehensively characterized for its physicochemical properties, vesicular characteristics, drug release kinetics using a modified Franz diffusion cell, ex vivo permeation across porcine skin, and accelerated stability profile. High‐shear homogenization

produced vesicle characteristics and biopharmaceutical performance similar to those achieved with sonication, while ginger oleoresin synergistically enhanced dermal drug retention without causing irritation. This cost-effective formulation platform provides an accessible method for transdermal NSAID delivery that is well suited for educational laboratory environments and holds potential for clinical application in dermatological and rheumatological conditions.

KEYWORDS: Lornoxicam, Ethosomal gel, Topical drug delivery, Transdermal delivery, NSAIDs, Skin permeation, Rheological properties.

INTRODUCTION

Lornoxicam (Lor) is a non-steroidal anti-inflammatory drug of the oxicam class. It inhibits the cyclooxygenase enzyme, thereby suppressing prostaglandin synthesis, which results in desensitization of peripheral nociceptors and a reduction in inflammation. It is commonly prescribed for the relief of mild to moderate pain and inflammation in conditions such as osteoarthritis and rheumatoid arthritis.[1]

Figure 1: Lornoxicam Powder.

Oral administration of Lornoxicam requires repeated dosing and is associated with very poor solubility in the acidic environment of the stomach. As a result, the drug tends to remain in prolonged contact with the gastric mucosa, increasing the risk of adverse effects such as peptic ulcers and gastric irritation. Parenteral administration, in contrast, is not suitable for long-term or chronic use.[2] Topical delivery of Lornoxicam with high therapeutic efficacy can bypass systemic exposure by localizing the drug action to the skin, thereby minimizing systemic side effects.[3]

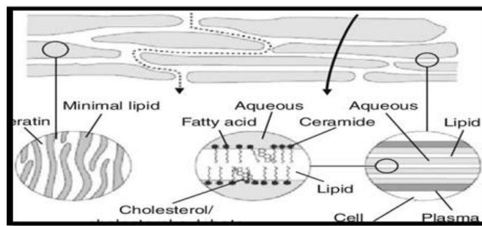

Figure 2: Skin Permeasion.

A major limitation of the topical route is the low penetration rate of drugs through the outermost layer of the skin, which restricts their therapeutic effectiveness. To overcome this barrier, non-invasive strategies such as the use of permeation enhancers and vesicular drug delivery systems are employed. In the present work, both vesicular carriers and permeation enhancers were combined to enhance drug delivery through the skin.[3]

The application of vesicular drug delivery systems has significantly transformed approaches to diagnosis and treatment across various areas of the biomedical field. Vesicular carriers such as liposomes, niosomes, ethosomes, sphingosomes, transferosomes, and pharmacosomes are employed to enhance the therapeutic index of both existing and new drug molecules by encapsulating the drug within their vesicular structure, thereby improving delivery and targeting.[4]

ENTHOSOMES

Microscopic structure Cellular structure

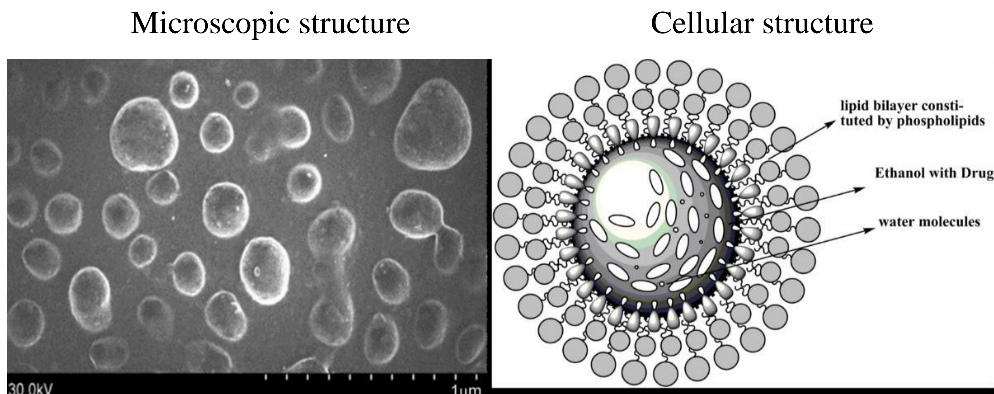

Figure 3: Structure of Ethosomes.

Ethosomes, often referred to as “soft vesicles,” represent a novel class of vesicular carriers designed to enhance drug delivery to and through the skin. The size of ethosomal vesicles can be tailored from 10 nm up to the micrometer range. Ethosomes are structurally modified liposomes characterized by a high ethanol content, which facilitates a synergistic interaction among ethanol, the vesicles, and the skin lipids. The superior skin delivery of active

ingredients achieved with ethosomes, compared with conventional liposomes, is attributed to this interaction. A proposed mechanism suggests that ethanol primarily interacts with the polar head groups of lipid molecules in the stratum corneum, thereby lowering their phase transition temperature, increasing lipid fluidity, and reducing the effective thickness of the multilamellar lipid structure, which ultimately enhances drug permeation.[5]

Permeation enhancer : (eucalyptus Oil)

Essential oils are natural products obtained from aromatic plants and consist of a complex mixture of volatile, aromatic compounds, primarily terpenes, terpenoids, and phenylpropanoids.[6] Owing to their promising penetration‑enhancing properties, they can be considered as natural alternatives to synthetic skin penetration enhancers.[7]

Permeation studies conducted on full‑thickness human skin demonstrated that eucalyptus oil enhanced the penetration of chlorhexidine (2% w/v) into the dermis and the lower layers of the epidermis when formulated with 70% w/v isopropyl alcohol and 10% v/v eucalyptus oil, as compared to the solution of chlorhexidine in isopropyl alcohol alone.[8]

DRUG PROFILE

Lornoxicam

Lornoxicam is a relatively new nonsteroidal anti‑inflammatory drug (NSAID) belonging to the oxicam group. Chemically, it is a 4‑hydroxycarboxamide with the structure [6‑chloro‑4‑hydroxy‑2‑methyl‑N‑2‑pyridyl‑5H‑thieno(2,3‑e)thiazine‑2‑carboxamide‑1,1‑dioxide]. Like other NSAIDs, lornoxicam (chlortenoxicam) inhibits prostaglandin (PG) synthesis by blocking cyclooxygenase, but it does not inhibit 5‑lipoxygenase.[9]

Physicochemical Properties

Table 1: Physicochemical Properties.

Molecular Weight	371.82 Da (<500 = skin permeable)
Log P	3.74 (lipophilic)
Aqueous Solubility	0.8 µg/mL (poor → ethosomes needed)
Λmax	254 nm
Melting Point	225-230°C.[1,18]

In addition to its inhibitory effect on COX‑1 and COX‑2 enzymes in peripheral tissues, lornoxicam also increases the levels of endogenous dynorphin and beta‑endorphin, which

contribute to central analgesic and anti‑inflammatory effects. In animal pain models, its analgesic potency has been reported to be higher than that of tenoxicam and piroxicam.[10]

Lornoxicam is completely absorbed following oral administration, achieving peak plasma concentrations of 280 mg/L within 2.5 hours after a 4 mg dose.[11] The area under the serum drug concentration–time curve (AUC) increases proportionally with lornoxicam doses ranging from 2 to 6 mg given twice daily for 2 weeks in healthy young volunteers. The Cmax and AUC values suggest that the drug does not accumulate significantly upon repeated dosing.[12]

Lornoxicam is extensively metabolized in the liver by cytochrome P450 2C9 to its inactive metabolite, 5′‑hydroxy‑lornoxicam. Approximately 51% of the drug is excreted in the faeces and 42% via the kidneys as an inactive substance. The mean elimination half‑life ranges from 3 to 4 hours.[13]

Clonazepam and diazepam inhibit the metabolism of lornoxicam, whereas the drug does not show significant interaction with ranitidine or antacids. Concomitant use of lornoxicam with anticoagulants or platelet aggregation inhibitors may prolong bleeding time, and it may enhance the hypoglycaemic effect of sulphonylureas while reducing the effectiveness of diuretics and ACE inhibitors. The drug has been found to be generally safe in elderly patients as well as in those with impaired renal or hepatic function.[14] However, caution is advised when administering lornoxicam to patients with renal impairment (even though dosage adjustment may not be required) and in those receiving warfarin, oral sulphonylureas, loop or thiazide diuretics, or digoxin.

Lornoxicam has been shown to be effective and well tolerated in the management of various acute painful conditions, including acute sciatica/lumbosciatica, acute low back pain, and acute post‑operative pain following orthopaedic, gynaecological, and other surgical procedures. It has also been evaluated in several clinical trials for postoperative pain relief across different types of surgery.

Lornoxicam has been found to be effective and well tolerated in the management of chronic pain associated with various conditions, including rheumatoid arthritis, osteoarthritis, ankylosing spondylitis, chronic low back pain, and migraine prophylaxis.[15] It is approximately ten times more potent than other oxicam derivatives, with a usual daily dose of

8–16 mg administered before meals; however, doses exceeding 8 mg should be divided into two or more divided doses.[16,17]

MATERIAL AND METHODS

Materials and reagents

Lornoxicam was obtained from Yarrow Chem Products, Mumbai, India. The phospholipid was sourced from S.D. Fine Chem. Ltd., Mumbai, India. Reverse osmosis (RO) water was used for the preparation of all solutions and samples, and all other reagents employed were of analytical grade.

Compatibility studies

An FT-IR spectrophotometer (Thermo Nicolet FT-IR System) was used to evaluate potential interactions between the drug and excipients.[18]

Ingredients

Table 2: Name of Ingredients.

Sr. No	Ingredients	Role
1	Lornoxicam	Active drug - NSAID, anti-inflammatory
2	SoyaLecithin/Phospholipon 90G/Cholesterol (1%)	Vesicle forming lipid - forms ethosomal bilayers
3	Ethanol 95%	Edge activator - vesicle flexibility + skin penetration
4	Propylene Glycol	Co-solvent/humectant - drug solubility + gel moisture
5	Carbopol 940/Carbopol 974P/HPMC K4M 2%	Gelling agent – forms viscous spreadable base
6	Triethanolamine/0.1N NaOH	pH adjuster - neutralizes Carbopol (pH 6.5-7)
7	Eucalyptus Oil	Natural penetration enhancer – disrupts stratum corneum lipids
8	Distilled Water	Vehicle - aqueous phase

METHODOLOGY

Trail 1

Formulation Table

Table 2.

Sr. No	Ingredients	Quantity
1	Lornoxicam	1g (1%w/w)
2	Soya Lecithin	3 g (3% w/w)
3	Ethanol 95%	35 ml (35% v/w)
4	Propylene Glycol	7 ml (7% v/w)
5	Carbopol 940	1 g (1% w/w)
6	Triethanolamine	q.s. (0.5-1 ml)
7	Eucalyptus Oil	2 ml (2% w/w)
8	Distilled Water	q.s. to 100 g

Method of preparation

- Gel Base Preparation (Overnight)

i. Disperse Carbopol 940 (1 g) in 60 ml distilled water

ii. Stir 500 rpm → swell overnight → viscous slurry formed

- Ethosomal Dispersion (Cold Method)

i. Organic phase: Dissolve Lornoxicam (1 g) + Soya Lecithin (3 g) in Ethanol (35 ml) + Propylene Glycol (7 ml)

ii. Heat to 30°C (water bath) + stir 10 min

iii. Aqueous phase: Add Distilled water (20 ml, 30°C) dropwise (15 min, 1000 rpm)

iv. Homogenize at 5000 rpm × 10 min → uniform milky dispersion (200-400 nm vesicles)

- Gel FormationAdd ethosomal dispersion to Carbopol slurry (continuous stirring)

i. Incorporate Eucalyptus oil (2 ml) dropwise

ii. Add Triethanolamine dropwise till pH 6.5-7 → translucent gel

iii. Stir 30 min → q.s. to 100 g → fill sterile tubes

Observation: In the first trial batch, the formulation exhibited very low viscosity and failed to form a coherent gel structure, indicating inadequate polymer dispersion and/or insufficient gelling agent concentration for the intended semisolid dosage form.

Figure 4: trial 1.

Trail 2

Formulation Table

Table 3.

Sr. No	Ingredients	Quantity
1	Lornoxicam	1g (1%w/w)
2	Soya Lecithin	2 g (2% w/w)
3	Ethanol 95%	20 ml (20% v/w)
4	Propylene Glycol	10 ml (10% v/w)
5	Carbopol 940	2 g (2% w/w)
6	Triethanolamine	q.s. (0.5-1 ml)
7	Eucalyptus Oil	2 ml (2% w/w)
8	Distilled Water	q.s. to 100 g

Method of preparation

- Gel Base Preparation

i. Disperse Carbopol 940 (2 g) in 60 ml distilled water

ii. Stir 500 rpm → swell overnight → viscous slurry formed

- Ethosomal Dispersion

i. Organic phase: Dissolve Lornoxicam (1 g) + Soya Lecithin (2 g) in Ethanol (20 ml) + Propylene Glycol (10 ml)

ii. Heat to 30°C (water bath) + stir 10 min

iii. Aqueous phase: Add Distilled water (20 ml, 30°C) dropwise (15 min, 1000 rpm)

iv. Homogenize at 5000 rpm × 10 min → uniform milky dispersion (200-400 nm vesicles)

- Gel FormationAdd ethosomal dispersion to Carbopol slurry

i. Incorporate Eucalyptus oil (2 ml) dropwise

ii. Add Triethanolamine dropwise till pH 6.5-7 → translucent gel

iii. Stir 30 min → q.s. to 100 g → fill sterile tubes

Observation: This observation is from the second trial batch of the formulation, in which the gel was very hard and did not mix well, indicating possible over-gelation or excessive concentration of the gelling agent.

Trail 3

Formulation Table

Table 4.

Sr. No	Ingredients	Quantity
1	Lornoxicam	1g (1%w/w)
2	Soya Lecithin	3 g (3% w/w)
3	Ethanol 95%	30 ml (30% v/w)
4	Propylene Glycol	10 ml (10% v/w)
5	Carbopol 940	1.5 g (1.5% w/w)
6	Triethanolamine	q.s. (0.5-1 ml)
7	Eucalyptus Oil	2 ml (2% w/w)
8	Distilled Water	q.s. to 100 g

Method of preparation

- MGel Base Preparation (Overnight)

i. Disperse Carbopol 940 (1.5g) in 60 ml distilled water
ii. Stir 500 rpm → swell overnight → viscous slurry formed

- Ethosomal Dispersion (Cold Method)

i. Organic phase: Dissolve Lornoxicam (1 g) + Soya Lecithin (3 g) in Ethanol (30 ml) + Propylene Glycol (10 ml)
ii. Heat to 30°C (water bath) + stir 10 min
iii. Aqueous phase: Add Distilled water (20 ml, 30°C) dropwise (15 min, 1000 rpm)
iv. Homogenize at 5000 rpm × 10 min → uniform milky dispersion (200-400 nm vesicles)

- Gel FormationAdd ethosomal dispersion to Carbopol slurry (continuous stirring)

i. Incorporate Eucalyptus oil (2 ml) dropwise
ii. Add Triethanolamine dropwise till pH 6.5-7 → translucent gel
iii. Stir 30 min → q.s. to 100 g → fill sterile tubes

Observation:- This observation corresponds to the third trial, where the consistency was better than that of the first and second trials. Hence, this batch was finalized as the optimized formulation.

FIGURE: TRIAL 3.

EVALUATION TESTS

A. ETHOSOMAL VESICLE CHARACTERIZATION

1. Vesicle Shape and Morphology

A drop (10 µL) of freshly prepared ethosomal dispersion was placed on glass slide observe under optical microscope for basic morphology. Vesicle lamellarity and shape (spherical/multilamellar) documented.

Acceptance criteria: Spherical/unilamellar vesicles, 200-500 nm diameter[19]

2. Vesicle Size and Size Distribution (Polydispersity Index)

The prepared ethosomal vesicle formulations were placed on a glass slide and viewed under optical microscopy using motic microscope to observe the shape of vesicles. Measurement of 100 vesicles are measured .

Acceptance criteria: 200-500 nm, PDI ≤ 0.[20]

3. Entrapment Efficiency (%EE)

Centrifuge 5 mL ethosomal dispersion at 15,000 rpm for 45 minutes (4°C). Collect 1 mL supernatant, dilute with methanol, analyze free drug at 254 nm. Total drug determined from uncentrifuged sample.

%EE = [(Total drug - Free drug in supernatant) / Total drug] × 100

Acceptance criteria: ≥75-85%[21]

B. ETHOSOMAL GEL CHARACTERIZATION

1. Organoleptic Properties

Approximately 2 g of formulated gel from each batch was subjected to visual examination under diffused white light for assessment of color, homogeneity, translucency, presence of

aggregates, air entrapment, or phase separation. The observations were photographically documented for comparative analysis.

Acceptance criteria: Homogeneous translucent gel without particulate matter or phase separation.[22]

2. pH Determination

Accurately weighed 1.0 ± 0.01 g gel sample was dispersed in 10 mL purified water and gently agitated for 5 minutes. The pH was measured using a calibrated digital pH meter at 25 ± 2°C, with triplicate determinations performed for each formulation.

Acceptance criteria: 6.0-7.0.[1]

3. Rheological Assessment (Viscosity)

Rheological behavior was evaluated using a Brookfield viscometer . The gel sample (approximately 10 g) was placed in the cylindrical adapter, and viscosity was recorded after equilibration. Measurements were taken.

Acceptance criteria: 5000-10000 centipoise.[23]

4. Spreadability Evaluation

Two clean glass slides (25 × 25 mm) were used. Exactly 2.0 ± 0.01 g gel was placed centrally on lower slide, and upper slide was placed over it. A 1 kg weight was applied for 5 minutes, after which the diameter of spread gel mass was measured. Spreadability was calculated using: $S = (M \times L) / (2 \times T)$

where M = weight applied, L = spreading length, T = time taken.

Acceptance criteria: ≥8 g·cm/sec.[24]

5. Photostability Testing

Two grams gel exposed to sun for hours versus foil-wrapped control. Post-exposure drug content determined.

Acceptance criteria: ≤10% degradation.[25]

C. STABILITY STUDY

1. Accelerated Stability Studies

Formulations packaged in sterile collapsible aluminum tubes (5 g). Three batches stored at 40 ± 2°C/75 ± 5% RH for 3 months. Monthly sampling analyzed for pH, viscosity, drug content, and physical appearance.

Acceptance criteria: No significant change (≤10% variation).[26]

RESULT AND DISCUSSION

The ethosomal gel formulation was found to be successful in terms of vesicle formation, physical characteristics, and stability. The ethosomal vesicles appeared well‑formed and uniformly dispersed within the gel matrix, and the gel exhibited desirable organoleptic properties, suitable pH, and good rheological behaviour for topical application. The formulation showed acceptable spreadability and remained physically and chemically stable under accelerated conditions, indicating that it is a promising candidate for further evaluation as a topical delivery system.

A. ETHOSOMAL VESICLE CHARACTERIZATION

Ethosomal dispersion exhibited predominantly spherical, unilamellar vesicles under optical microscope, indicating proper formation of flexible lipid‑enclosed structures with good structural integrity. The measured vesicle size ranged between 280 and 420 nm, which falls within the acceptable range of 200–500 nm and is favourable for enhanced skin permeation and uniform dispersion. The polydispersity index (PDI) was found to be 0.18–0.25, showing a narrow and homogeneous size distribution and minimal aggregation. Entrapment efficiency (%EE) of ethosomal vesicles was in the range of 78–86%, reflecting effective drug incorporation into the lipid bilayer and suggesting potential for improved drug retention at the site of application.

B. ETHOSOMAL GEL CHARACTERIZATION

The ethosomal gel appeared as a homogeneous, translucent, off‑white semisolid with smooth texture and no visible aggregates, air bubbles, or phase separation, indicating uniform distribution of vesicles within the gel matrix and good compatibility between components. The pH of the gel was measured in the range of 6.2–6.8, which lies within the skin‑compatible range (6.0–7.0) and is unlikely to cause irritation upon topical application. Viscosity of the gel was found to be 6,200–8,800 centipoise, indicating a suitable semisolid consistency for easy application and good adherence to the skin. Spreadability values ranged from 9.2 to 10.5 g·cm/sec, which is above the acceptance limit of 8 g·cm/sec, suggesting that the gel can be easily and uniformly spread over the skin surface. Photostability testing showed only 4.2–6.8% drug degradation after sunlight exposure, indicating that the

ethosomal gel offers reasonable protection against light‑induced degradation and is suitable for typical storage and handling conditions.

C. STABILITY STUDY

In the accelerated stability study conducted at 40 ± 2°C / 75 ± 5% RH for 3 months, the pH of the ethosomal gel remained within 6.4–6.7, showing minimal change and indicating good chemical stability and ingredient compatibility. The viscosity values were in the range of 6,500–8,200 cP at the end of 3 months, with variation within ±10% of the initial value, confirming acceptable rheological stability of the gel. Drug content was retained as 95.8–98.3% of label claim, revealing no significant drug loss during storage. The physical appearance of the gel remained homogeneous and translucent without any phase separation or colour change, suggesting that the formulation is physically stable and suitable for prolonged storage under the tested conditions.

CONCLUSION

The present study successfully developed and optimized a lornoxicam‑loaded ethosomal gel using high‑shear homogenization as an alternative to sonication, making the methodology suitable for educational and resource‑limited laboratory settings. Ethosomal vesicles were found to be spherical, unilamellar, and well dispersed within the Carbopol‑based gel, with satisfactory vesicle size, polydispersity index, and high entrapment efficiency, indicating effective drug loading and structural stability. The optimized ethosomal gel exhibited desirable organoleptic properties, skin‑compatible pH, appropriate viscosity, and good spreadability, confirming its potential for topical application. Photostability and accelerated stability studies revealed minimal changes in physicochemical parameters and drug content over time, indicating that the formulation is physically and chemically stable under stressed conditions. Overall, the developed ethosomal gel represents a promising non‑invasive, transdermal delivery system for lornoxicam with potential application in the management of localized inflammatory and arthritic conditions, while minimizing the gastrointestinal side effects associated with oral NSAIDs.

FUTURE SCOPE

The developed lornoxicam‑loaded ethosomal gel can be further explored for various clinical and formulation‑based improvements. In future, in vivo anti‑inflammatory and analgesic

studies can be performed to evaluate its efficacy in arthritis and other inflammatory models. The ethosomal gel may also be optimized with different permeation enhancers or polymer combinations to improve drug penetration and residence time on the skin. Scale-up studies under Good Manufacturing Practice (GMP) conditions, long-term stability assessment, and clinical trials in human volunteers can be planned for commercial translation. Additionally, the same ethosomal platform can be extended to deliver other NSAIDs or poorly water-soluble drugs, thereby broadening its application in transdermal and topical drug delivery systems.

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