

**A COMPARATIVE STUDY OF THERAPEUTIC
RESPONSES OF CHEMICAL PEELING, CHEMICAL
PEELING AND MICRODERMABRASION IN
PATIENTS WITH ACNE VULGARIS**

*Dissertation Submitted In Partial
Fulfillment of University Regulation For*

**MD DEGREE IN
DERMATOLOGY, VENEREOLOGY AND LEPROLOGY
(BRANCH XII A)**



THE TAMILNADU DR. M. G. R. MEDICAL UNIVERSITY

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CERTIFICATE

Certified that this dissertation entitled “A comparative study of therapeutic responses of chemical peeling, chemical peeling and microdermabrasion in patients with acne vulgaris” is a bonafide work done by DR. R.SINDHUJA, Postgraduate student of the Department of Dermatology and Leprology and Institute of Venereology, Madras Medical College, Chennai- 3, during the academic year 2004 – 2007. This work has not previously formed the basis for the award of any degree or diploma.

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Declaration

I, Dr. R.SINDHUJA, solemnly declare that dissertation titled, “**A comparative study of therapeutic responses of chemical peeling, chemical peeling and microdermabrasion in patients with acne vulgaris**” is a bonafide work done by me at Madras Medical College during 2004-2007 under the guidance and supervision of Prof. Dr. B. PARVEEN, M.D.,D.D., Professor and Head, Department of Dermatology, Madras Medical College, Chennai-600 003.

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INTRODUCTION

Chemical peeling is a technique used to improve the appearance of the skin. A chemical solution is applied to the skin, which causes it to separate, peel off, and allows new skin to regenerate. The new skin is smoother and less wrinkled than the old skin, and may also be more even in color

Dermatologists have used various peeling agents for decades and are experts in performing all types of this chemical surgery. This concept dates back to Roman times. Superficial and medium depth skin peeling with trichloroacetic acid has been a well documented therapy in United states since atleast 1960. Dr. Baker and Gordon pioneered chemical peeling with phenol in the year 1961.¹ Chemical peeling one of the latest treatments available for the about disease in modern era. Superficial and medium depth skin peels can create dramatic improvement in the skin but the results were not long lasting as those with phenol peels. The use of retinoic acid alpha hydroxyl acids, broad-spectrum sunscreens and skin bleaches as part of a post-peel maintenance programme has allowed patients maintain the improvement in skin for far longer.²

Microdermarasion is a non- chemical, non- invasive modality for treating superficial skin blemishes resulting from trauma, aging, prolonged

exposure to skin or mild acne. Microdermabrasion has been used successfully since at least 1992 to treat acne, fine lines and wrinkles, unwanted pigmentation and other superficial skin damage.³ Even though various modalities of treatment are available for acne, it still poses a therapeutic problem to the attending dermatologist. The advent of new and potent topical therapeutic agents, chemical peeling and dermabrasion has resulted in significant improvement for us to treat many forms of acne.

This study attempts to find the therapeutic response to various chemical peels like salicylic and glycolic acid on acne along with microdermabrasion.

REVIEW OF LITERATURE

Acne vulgaris is a chronic inflammatory, self-limited disease of pilosebaceous unit, seen primarily in adolescents clinically characterized by formation of comedones papules pustules nodules or pseudo cysts and in some cases accompanied by scarring.⁴ Acne vulgaris is a very common physiological malady of adolescents but it is better regarded as a disease due to its inflammatory component and the disfigurements it produces on the socially and psychologically most important body region.

The term 'comedone' was suggested by Hoeflt (1846).¹ Samuel plumbe (1795-1837) recognized these primary acne lesions which in some patient evolve into papules, pustules and nodules. The importance of propionibacterium acnes and sebum in the pathogenesis of acne was emphasized by Thibierge (1900). This condition usually starts in adolescence⁵ although some patients present in the first year of life with neo-natal or infantile acne and frequently resolves by mid twenties. Some degree of acne affects 95% and 83% of 16-year boys & girls respectively.⁶ In about 20%, the disease needs the help of physician. A peak in the incidence and severity occurs between 14 and 17 years in females, 16, and 19 years in male.

The highest concentration of acne lesions are found in the sebaceous rich anatomic areas of face, shoulders, mid-chest, and upper back. The disease is usually self-limited but cases appearing *denovo* in the second and third decade are not uncommon.

A ETIOLOGY AND PATHOGENESIS

Although the basic cause of acne is unknown, there is considerable information on the various factor concerned in its pathogenesis. Acne is a multifactorial disease, developing in the sebaceous follicles.⁵

FOUR PRINCIPLE PATHOLOGIC EVENTS IN ACNE ARE:

1. Seborrhoea
2. Abnormal follicular keratinisation leading to plugging of the follicle
3. proliferation of propionibacterium acnes in the sebum
- 4 .mediation of inflammation.

SEBORRHOEA:

Patients with increased sebum production complain of \square olonizat . Sebaceous activity is predominantly dependent on androgenic sex hormones of gonadal or adrenal origin. High level of sebum secretion results from high overall androgen production or increased bioavailability

of free androgen due to deficiency of sex hormone binding globulin (SHBG)⁷

Sebum secretion varies from follicle to follicle and certain follicles may be prone to acne. An enhanced peripheral response to androgen stimulation or even the circulating androgens⁸ are converted to a more potent androgen 5 α reductase type 1.⁹ Patients with acne and polycystic ovarian disease have high level of LH , prolactin ,testosterone ,androstenedione and LH/FSH ratio more than three.¹⁰

Sebum in acne patients have significant decrease in the level of linoleic acid. This results in follicular hyperkeratosis and decreased epithelial barrier function. Late onset adrenal hyperplasia due to a partial deficiency α 21 hydroxylase can be present in patient with persistent problems with their acne.

ABNORMAL FOLLICULAR KERATINISATION:

Kinetic studies have demonstrated that there is an increase in cellular turnover¹¹ in comedones and their increased adhesion due to persistence of desmosome leads to retention hyperkeratosis. The stimulus for hypercornification may be androgen mediated or the result from the irritant effect of sebaceous free fatty acids. Androgen mediated hypercornification is due to its receptor present in the outer root sheath of infra infundibular

region of follicles.¹² Sebum initiate the infundibular keratinocyte leading to release of interleukin 1 alpha. This induces the follicular hyperkeratosis.^{13,14}

Impaired water barrier function is caused by reduced amounts of ceramides which are responsible for comedone formation, since barrier dysfunction is accompanied by hyperkeratosis of the follicular epithelium. Local follicular deficiency of epidermal lipids (free sterol, ceramides) and increased sebum glycerides may induce abnormal follicular keratinisation.¹⁵

BACTERIAL COLONISATION:

Bacterial colonization of sebaceous follicles is another important contributing factor in the production of acne. Propionibacterium acnes, staphylococcus epidermidis and pityrosporum ovale are the primary organisms found in the acne patients but propionibacterium acnes is the most abundant organism present.¹⁶ Ideally for the over production of propionibacterium acnes, the anaerobic atmosphere of the blocked sebaceous follicle with its lipids substrate is useful for the production of the free fatty acids ,which in turn may be a factor causing retention hyperkeratosis.

Hydrolysis of serum triglycerides by lipases produced by Propionibacterium takes place.

The chemotactic factor released by propionibacterium acnes attracts to the follicle. These leucocytes ingest propionibacterium acnes with the resultant release of hydrolytic enzyme that damage the follicular wall causing it to rupture. The content of the follicle provoke inflammatory reaction. The severity of the inflammation in acne¹⁷ is determined also by host response to propionibacterium acnes. Antibodies IgG1, IgG2, IgG3 to propionibacterium may be involved in the pathogenesis of acne.¹⁸

MEDIATION OF INFLAMMATION:

Propionibacterium acnes produce proteases like lipases phosphatases, hyaluronate lyase which mediate inflammation. This produce biologically active substances that diffuses into the dermis and causes inflammation by activating complement and chemotactic neutrophils.

EVOLUTION OF ACNE LESION

Microcomedone mature and become epithelial lined follicular cyst containing keratinous material, lipid, hair and bacteria.¹¹ Two types of mature comedones are produced. The open comedone (black head) orifice is widely dilated by a cornified impaction continuous with the deeper keratinized lamella. Black colour is due to melanin and oxidized lipids in

the follicle. The closed comedone (white head) is a small usually flesh coloured papules that has a microscopic opening which keeps its contents from escaping.

PRECIPITATING FACTORS:

1.DRUGS:

HORMONES & STEROIDS:	Androgen Anabolic Steroids
Anti epileptic drugs :	Phenytoin Phenobarbitone
Anti-tuberculous drugs:	Iso-niacid Rifampicin
Halagens:	Bromides Iodide Halothane
Miscellanaeous:	Lithium, puva. Sulphur Chloral hydrate

OCCUPATION:

Exposure to cutting oils, coal tar oils and pitch may induce acne.

STRESS:

Emotional factors presumably affect acne by altering the adrenal-pituitary axis.¹¹

CLINICAL MANIFESTATION:

The pathognomonic lesion of acne is the comedones, either open or closed. As the disease progress papules, nodules, and cyst may appear as a single lesion or as a combination of all types. As acne lesion, resolve a post inflammatory erythema and even pigmentation can last several months before they resolve.

The deeper inflammatory process the more likely it will tend to produce permanent scarring which vary from small pits to deep fissures and even hypertrophic or keloidal scar. The primary site of scar is the face and to a lesser degree the back, chest and shoulder. The courses of acne tend to wax and wane. Seasonal variation may be seen.

GRADING OF ACNE:

The severity of acne can be graded on clinical grounds as¹⁹

Grade 1 (Mild) - Comedones, Occasional, papule

Grade 2 (Moderate) - papules, comedones, few pustules

Grade 3 (severe) - predominant pustules, nodules & abcesses

Grade 4(cystic) - mainly cysts, abcesses and widespread scarring.

Various grading and scoring system in acne .²⁰

MODIFIED COOKS METHOD:

Reliable method since photographic reference standard is required. Used only for facial lesion. Grading ranging from 0 -9 is used for one group that includes comedones papules and macules.²¹

Overall severity is graded on another scale of 0-8 that also includes pustules, nodules and cysts.

LEEDS TECHNIQUE:

Complex scores but also includes assessment of lesions over face as well as over back and chest. No photo graphic reference is required. Face is divided into right and left halves and counting is done on both sides.²²

SEVERITY INDEX MICHAELSON:

Simple score, by counting number of open or closed comedones, papules, pustles and infiltrated lesions. Severity index. .5 for comedones, 1 for papule, 2 for pustle, 3 for infiltrated lesions, 4 for cystic lesions²³.

In Grimes study²⁴ the total number of lesions including the comedones, papules and pustules were counted before the start of treatment and the response noted by the reduction of the number of total lesions and graded.

ASSOCIATIONS WITH ACNE:

FOLLICULAR OCCLUSION TRIAD: Dissecting folliculitis of scalp, hidradenitis suppurativa and acne conglobata

POLYCYSTIC OVARIAN DISEASE : Acne vulgaris, hirsutism, infertility or irregular menstruation.²⁵

APERT SYNDROME: Acneiform eruptions and pre-mature epiphyseal fusion of long bones and skull

SAPHO SYNDROME : consists of synovitis, acne, pustulosis, hyperostosis and osteitis.

VARIANTS OF ACNE :

Externally induced :-

- | | |
|----------------------|-------------------|
| 1. Cosmetic acne | 5. Detergent acne |
| 2. Mechanical acne | 6. Chlor acne |
| 3. Pomade acne | 7. Tropical acne |
| 4. Occupational acne | |

Severe forms :-

Pyoderma faciale

Acne conglobata

Acne fulminans

Others :

Acne excoricea

Drug induced acne

Endocrine acne

Infantile & Juvenile acne

TOPICAL TREATMENT OF ACNE :

GENERAL MEASURES:

1.CLEANSING:

Cleansing agents like soaps or topical anti bacterial agents used.⁵

OTHER TOPICAL THERAPIES:

COMEDOLYTIC AGENTS

ANTI MICROBIALS

ANTI INFLAMMATORY AGENTS

COMBINATION PREPARATION

PHYSICAL MODALITIES

1.CHEMICAL PEELING

2.MICRO DERMABRATION

3.PHOTO DYNAMIC THERAPY

4.LASER THERAPY WITH N LITE LASER

5.CRYOTHERAPY

6.SUPER FICIAL X-RAY THERAPY

CHEMICAL PEELING:

Definition:

Chemical peeling is basically an accelerated form of exfoliation induced by the use of a chemical cauterant or escharotic agent. Very light peeling agents induce a faster sloughing of the cells in the stratum corneum where as deeper peeling agents create necrosis and inflammation in the epidermis, papillary dermis or reticular dermis.²⁶

History of chemical peels:

The first breakthrough in histological assessment of chemical peeling was perhaps by Brown and Kaplan, who demonstrated thickening of dermis in rabbit ears treated with phenol, a phenomenon they termed Fibrosis. In 1985, Kligman modified Baker's technique and undertook a long term evaluation of chemical peels. He noted a white band of thin compact collagen bundles arranged horizontally parallel to skin surface, usually 2-3 mm wide. Numerous elastic fibers parallel to collagen bundles were seen. Telangiectasia was noted in the deeper dermis where the peel had not reached.

Mechanism of action:

Stimulation of epidermal growth through removal of stratum corneum²⁶

Destruction of specific layers of damaged skin.

Induction of an inflammatory reaction deeper in the tissue than the necrosis induced by the peeling agent. It is able to induce production of new collagen and ground substance in the dermis. Epidermal wounds are capable of inducing deposition of collagen and glycosaminoglycans in the dermis.

Wound Healing Stages in chemical peeling:

Coagulation inflammation.

Re-epithelialization.

Granulation tissue formation.

Angiogenesis.

Collagen remodeling.

1. Coagulation inflammation.

These are the initial phases of wound healing in chemical peeling.. Soluble factors are elaborated in clotting that activate kinins and complement inflammatory pathways, which function as chemoattractant for neutrophils, macrophages and lymphocytes. Macrophages direct the subsequent development of granulation tissue and lymphocyte augment fibroblast accumulation and proliferation.²⁷

2. Re-epithelialization.

This begins within 24 hours of wound-healing. Certain mediators released during inflammation such as fibronectin, laminin and platelet

derived growth factor may stimulate keratinocyte cell movement. Fibronectin stimulates adhesion of fibrin, collagen and a variety of cells. The water content of wound-bed is a major factor in the epithelial cell migration rate. Occluded wounds reepithelialize faster than open dry wound.^{28,29}

3. Granulation tissue formation.

Granulation tissue is a loose collection of cellular components including fibroblast, fibronectin, inflammatory cells, glycosaminoglycans and collagens. Granulation tissue formation begins on second or third day after peeling.^{30,31} The chief cell is the fibroblast which produces fibrillar collagen, elastin, fibronectin and proteases such as collagenases.

4. Angiogenesis.

After chemical peeling the resumption of blood flow is essential for supplying oxygen and nutrients to the healing wound.^{32, 33} Angiogenic growth factors may be important which are released from fibroblasts, macrophages and endothelial cells.

5. Collagen remodeling.

Collagen and matrix remodeling begins when granulation tissue formation begins and continues for months after re-epithelialization.^{34,27} As collagen is laid down, fibronectin gradually disappears. Fibers of collagen type 1 and 3 close together as water is resorbed and reorient in a parallel

fashion to the skin surface. This remodeling is responsible for the texture of the skin after peeling.

Fitzpatrick's Classification of skin types :

Skin Type	Color	Reaction to sun
I.	Very white or freckled	Always burn
II.	White	Usually burn
III.	White to olive	Sometimes burn
IV.	Brown	Rarely burn
V.	Dark brown	Very rarely burn
VI.	Black	Never burn, always tan

Classification of peels: ³⁵

Very superficial

Superficial

Medium depth

Deep

1. Very Superficial

It is more of an exfoliation. The most superficial layers of the stratum corneum (at the top of the epidermis)³⁶ is removed or thinned during exfoliation. Most chemical peels have pre-operative regimen of using

exfoliating agents. This helps the chemical peeling agents penetrate more deeply and evenly. They have the beneficial property of smoothing out thickened rough area. Chemicals used are:

30% Lactic acid

Salicylic acid 20% single coat.

TCA 10% single coat

Glycolic acid 35 % 1-2 mins.

2.Superficial

Extend up to the whole thickness of epidermis. They also help reducing the appearance of very mild blotchy skin discoloration, remnant acne discoloration and help cleanse the face³⁷. This is the most common form of skin peel and requires 2-3 days of exfoliation and healing. The peeling agents are:

1. TCA 15-20% two coats
2. Glycolic acid 35-50% 2-5 mins.
3. Jessner's Peel.

3.Medium Depth

This is up to the mid dermal level³⁸. These agents will cause destruction up to papillary dermis with or without inflammation thus improving superficial acne scars and rhytides. Chemical agents used are:

TCA 35-50 % till deep uniform frosting seen.

Glycolic Acid 65-75%

4. Deep Peels

This is up to reticular dermis level. These agents are not advised for Asian skins (type 4 and 5) as there are very high chances of scarring and other complications. Chemicals used are :

Phenol 88%

TCA 50%

Factors determining depth of peels:

Peeling agents.

Concentration of agent applied.

Number of coats applied.

Techniques of application.

The manner in which the skin was cleaned and degreased before the peel .

The method of priming of skin before the peel.

Duration of contact with the skin.

Peeling for Asian skins:

Deep peeling is not advisable as it causes scarring and hyper or hypo pigmentation in the Asian skin. Even medium depth peel should not exceed 15-20% of TCA and 35% GA. Combination peel or cryopeel can be tried for them. Superficial peels are very safe and cosmetic results are excellent for

Indian skin. cryopeel is very useful in treating active acne lesions and superficial scars of acne.³⁹

Various peeling agents used are:

Retinoic acid

Jessner's solution.

Resorcinol

Alpha hydroxyl acids.

Salicylic acid.

Trichloroacetic acid.

Phenols.

ALPHA HYDROXY ACIDS:

These are a group of organic acids that have recently become popular in the treatment of a variety of skin conditions, particularly those characterized by hyper keratinisation. Several of these acids are derived from fruits, so they are often referred to as fruitacids. Glycolic Acid is derived from sugarcane, citric acid from citrus fruit and malic acid from apples⁴⁰ the various alpha hydroxy acids are

Glycolic Acid , Malic Acid , Mandelic Acid ,Lactic Acid, Pyruvic Acid⁴² and Tartaric Acid

Points concerned on using AHA products:

All AHA products may create transient stinging when first applied to the skin. This is normal and not a cause for concern. Persistent stinging longer than 60 seconds implies too strong the product for the patient's skin.

It is best to start the patient on low level products and gradually increase the concentration of products.

Patients on AHA should use a broad spectrum sunscreen on the morning to protect the newly improved skin.

Patients on AHA's usually build up a tolerance to any irritation they may initially experience.

GLYCOLIC ACID PEELS:

Formula:

Highest cosmetic grade is a 70% solutions⁴¹ solutions are made using water or a combination of water alcohol and propylene glycol.

Stability:

Not light sensitive. So it does not need to be stored in a dark bottle.

Very stable(more than 2 years)

Deliquescent(absorbs moisture from the air).

Physical Characteristics:

Clear solution can be made to a gel with addition to a gelling agent.

For daily regimen – 8-15% concentration is used.

As Peeling Agent: Used in a concentration of 30% or greater.

Advantages:

Even very superficial glycolic acid peels may achieve significant effects.

Systemically safe, non-toxic acids.

They have few complications and well tolerated by patients.

It is an office procedure .

Disadvantages:

There is a tremendous variability from patient to patient in reactivity and efficacy.

It has to be neutralized. It has a tendency to penetrate unevenly.

Neutralization:

Glycolic acid peels need to be neutralized to terminate their action when they have achieved the desired depth of wound. If they are left un-neutralized, they may penetrate too deeply and over peel the patient.

Neutralization can be done using any product with an alkaline PH or by flushing the area with water and diluting the acid. Sodium bicarbonate solution can be used as the neutralizing agent.

Sodium Bicarbonate Solution:

Sodium bicarbonate solution creates carbon dioxide seen as bubbling or fizzing on the surface of the skin. The drawback to sodium bicarbonate solution is that it must be applied in copious amounts to the entire peeled area.

Strength of peel:

Starts with 30% and gradually increased to 50% and then 70% to produce a more aggressive peel .

Time of contact:

There is no definite endpoint to AHA peeling.⁴³ The stages of glycolic acid in increasing order of depth of peel are: pink, red, epidermolysis and vesiculation , frosting.

Erythema corresponds to intra epidermal wounding. Epidermolysis shows as Grey-White color in areas in which the epidermis is separated from dermis.

Vesiculation is due to epidermolysis. Frosting seems to be an indicator of dermal injury.

Light AHA peels: Endpoint is mild erythema.

Medium depth peels: Endpoint is epidermolysis.

Post Peel Care:

If the patient has any irritation, erythema or superficial crusting after the peel. The daily AHA products or retinoic acid should not be applied. If significant inflammation is present a mild topical steroids cream can be applied twice daily to speed up resolution. If there are areas of crusting it should be treated with a topical anti bacterial ointment. If the patient feels tight or sensitive bland emollient should be applied daily.

Areas that can be peeled with glycolic acid:

Face commonly, neck , chest and dorsa of hand.

Frequency of Peeling:

The superficial peels should be performed with atleast a two weeks interval and 3-4 weeks interval for deep peels.

SALICYLIC ACID:

Introduction:

Salicylic acid has been a main stay in dermatology for many centuries as a topical agent to treat skin disorders. It is also known as beta hydroxy acids. It is a white crystalline powder derived from Willow bark, winter green leaves and sweet birch. Kligman has described salicylic acid as beta hydroxyl acids

Formula:

Salicylic acid – USP 50%

Methyl salicylate – 16 drops.

Aquaphor – 112 grams.

Physical characteristics:

Lipid soluble. It interacts with lipids that surround keratinized cells there by producing the required keratolytic effect.⁴⁴ Salicylic acid must be formulated at a proper PH to allow enough free acids to be present. Thus effective formulations are those in which PH is closer to PKa of 2.98 .

Advantages:

Technically easy to perform.⁴⁵

Have a low incidence of significant complications.

Does not penetrate too deeply.

Effective for treatment hyper pigmented age spots on the hands and arms.

Comedolytic and lipophilic can be used in acne.

Currently a popular component of many in office peels.

Disadvantages:

Have a long healing time.⁴⁵

Salicylism is fairly common.

Areas that can be applied:

Face commonly, Arms and hands.

Frequency of peeling:

Repeeling should be done with an interval of 2-3 weeks to allow the skin to heal.

Concentration of peel used:

20% and 30% salicylic acid are the most common in office peels used.⁴⁶

Action of salicylic acid on acne:

BHA is lipophilic—so penetrates the sebaceous material in hair follicle and exfoliate the pores.

BHA has strong comedolytic activity.^{46,24}

Increases collagen synthesis.⁴⁶

Frosting in salicylic acid peel:

Frost seen in a BHA peeling represents precipitated salicylic acid, while the frost in TCA peel represents precipitated skin proteins. BHA peels frost in 2minutes. Retouching the frosted areas of TCA peel could result in burning and discouraged.⁴⁶ But retouching in BHA peel don't do any harm.

Neutralization:

No neutralization for salicylic acid is needed.

Timing of peel:

As soon as the frosting is seen application of salicylic acid is stopped. Then after 3-5 minutes patient is asked to wash the area.

Post peel care:

During healing the patient is instructed to drink 8 glasses of water a day and notify to the doctor on experiencing nausea, disorientation or tinnitus. Mild degree of burning or stinging may be experienced by the patient while doing the peel. Topical steroids can be applied on persistence of severe tingling or burning sensations.

Salicylism:

Salicylic acid toxicity is referred to as salicylism. Lesser level of toxicity are more common including rapid breathing and tinnitus decreased hearing, dizziness. Nausea , vomiting and abdomen cramps may also occur. Severe toxic reactions are marked by central nervous system reactions with mental disturbances that can simulate alcohol intoxications. Allergic reactions to salicylic acid are extremely rare, so patch testing is not required.

Comparison of AHA and BHA:⁴⁶

	AHA	BHA
Useful in photo-aging	Yes	Yes
Useful in acne ^{48,56}	Yes	Yes
Useful in Melasma	Yes	Yes
Useful in dry skin	Yes	Yes
Enhances exfoliations	Yes	Yes
Lipophilic	No	Yes
Inhibits arachidonic acid	No	Yes
Anaesthetic properties	May be	Yes
Must be neutralized	Yes	No
Visible frost	No	Yes
Risk of salicylism	No	Yes
Use in pregnancy/breast feeding	Unknown	No
FDA approved for home use	No	Yes
Shown to increase collagen synthesis	Yes	No

Other peeling agents:

Trichloro acetic acid:

Advantages:

No systemic toxicity.⁴⁸

Can be used to create superficial, medium or deep peels.

Inexpensive.

Stable.

No need to neutralize TCA peel.

Disadvantage:

Higher concentrations seem to create scarring.

Concentration used: Up to 20% for Indian skin as concentration more than this can cause pigmentary changes, hyper or hypo pigmentations.

Jessner's peel solution:

Formula:⁴⁹

Resorcinol 14 grams

Salicylic acid 14 grams

Lactic acid 14cc

Ethanol to add up to 100cc

Advantages:

Very difficult to over peel a patient and inadvertently create too deep wound.

Jessner's peel creates a good deal of exfoliation.

Disadvantages:

Greater chance of manufacturing variations.

Possible toxicity from salicylic acid and resorcinol.

BASIC CONCEPTS OF CHEMICAL PEELING:

Pharmaceutical considerations:⁵⁰

Peel should be standardized. Variation of factors such as skin type and biological response is inevitable in peels.

Legal standards:

When chemicals are used in preparations USP grade material should be used. For drugs not listed in USP, national formulary(NF), British Pharmacopoeia(BP) can be used.

Priming:

Priming the skin for a peel is one of the most important concepts in chemical rejuvenation.

The goals of skin preparation are as follows:

Reduced wound healing time.⁵⁰

Priming agents daily for atleast 2 weeks speed up re-epithelization by about 24 hours.

Thinning of the stratum corneum by the priming allows better penetration of the peeling agent.

Decrease the risk of post inflammatory hyper pigmentation by enhancing the dispersion of melanin granules throughout the epidermis.⁵⁰

Establish patient compliance and eliminate inappropriate patients.

Agents commonly used to prime the skin for peels include:

Retinoic acid, AHA's , Hydroquinone, Kojic acid and Broad spectrum sun screeners.

Bleaching agents can be used for priming in patients with dyschromias.

Indications for chemical peel:

Pigmentary dyschromias

- A. Melasma
- B. lentigenes
- C. Ephelides
- D. post inflammatory hyperpigmentation

Epidermal pigmentation responds better to treatment than with mixed or dermal pigmentation.

ACNE

Acne vulgaris

Acne excorie

Post acne scarring

Post acne hyper pigmentation

Superficial peels for acne work primarily through comedolysis and epidermolysis as pustules are unrooted.⁵¹

A series of superficial peels can give dramatic improvement in active acne over a very short period of time.⁵² Patients with acne tend to have skin very sensitive to peeling.⁵³

The best can be demonstrated in case of acne excorie.⁵⁴ Even in single sitting comedones can be effectively reduced. Post acne pigmentation and scarring can also respond to chemical peels but more number of sittings would be required for the same.

ACTINIC DAMAGE AND FINE LINES:

There was statistically significant improvement in rough texture, fine wrinkling and decreased number of actinic keratoses in a study with 50% glycolic acid gel performed by Newman et al.⁵⁵

MACULAR AMYLOIDOSIS:

Macular amyloidosis has been subjected to chemical peels along with cryotherapy to attain medium depth injury with good cosmetic results.

PERIORBITAL PIGMENTATION:

As the skin around the eyes is thinner and lax low concentration peels should be used and gradually increased. A lighter peel on the face can have a beneficial additive effect.

METHODOLOGY:

Chemical peeling procedure can be easily divided into 3 steps.

Prepeel : preparing the skin for peel

Peel : actual peel

Post peel: After care.

Pre peel:

Counseling is a very important part of chemical peeling. The patient must be informed about the problems encountered during post peel period, the number of peels to undergo to obtain maximum cosmetic results.

Consent form:

Any patient undergoing the procedure should sign a consent form. Written information about the type of peel they undergo. A consent form describing the risk and benefits of the procedure.

Priming:

Priming of the skin should be done before the peel as mentioned else where.

PEEL

All patients selected for chemical peel must be asked to come

- a. Without any makeup
- b. after 2 weeks of using the priming agent.

On arrival to the clinic, the patient must remove contact lens if used

Wash all the makeup with soap and water.

Wear loose cloths.

CLEANSING

The skin should be degreased with cleansing agent such as alcohol, acetone or another agent.

Actual procedure

Ask the patient to lie down for facial chemical peel at an angle of 45 degree to prevent pooling of the chemical into the eyes during procedure.

Put cotton plugs into the ears. Apply petrolatum to the sensitive areas . put cap on head covering the hair. Eyes closed throughout the procedure.

Syringe filled with normal saline in case of accidental spilling into eyes.

Application :

The cotton tipped applicator is dipped in chemical . Eyes closed and peel applied with smooth strokes unit wise and sequentially starting with a) forehead b) right cheek c) left cheek d)chin e) nasal bridge, nose , perioral area f) upper and lower eyelids (least sensitive to most sensitive area)

Feathering the solution in hairline, eyebrow, vermillion and submandibular border done.

Reaction :

Erythema , blanch – frost with TCA and salicylic acid.

Erythema , epidermolysis with glycolic acid.

Terminate the procedure by washing with water till the burning or stinging sensation stops. Don't cross over eyes during procedures.

Post Peel:**Salicylic Acid:**

Brownish black pigmentation seen and the skin starts peeling and separates from second day onwards. More the exfoliation better the results.

Glycolic Acid:

Erythema may persist for 24-48 hours. Patient looks near normal in the post peel period.

Advice for patients during the healing phase:

To avoid sun exposure.

To avoid exercise.

To avoid having the shower spray strike directly on the face.

Not to rub, pick and or unnecessarily touch the face.

To try to sleep on their back.

Complications:**Tears dripping on to the neck.⁵⁷**

Dripping of the tears on the face causes dilution of acids due to the aqueous solution in the tears.

Dripping on to the neck causes an area of peeling on the neck.

Premature peeling:

The layer of necrotic skin functions as a protective bandage and premature removal of this layer exposes layer of immature and fragile tissue. This can lead to infection, persistent erythema, post inflammatory hyper pigmentation and scarring.

Infections:

The evidence of infection increases with depth of peel. The organism that create infection with peels include

Common bacterial pathogens –Staphylococcus and streptococcus species.

Uncommon bacterial pathogens – pseudomonas and enterobacter species.

Herpes simplex

Candida species.

Acne form eruptions.

Appears as multiple tender erythematous follicular papules. They respond promptly to anti biotic therapy used to treat normal acne.

Ecchymosis.

Small number of patients develop ecchymosis in infra orbital area. These are self limited complications and discoloration resolves completely within 4-6 weeks.

Post inflammatory hyper pigmentation.

This is commonly associated with dark skin patients.

Will gradually improve with time with no therapy except sun avoidance.

Broad spectrum sun screeners is mandatory.

Hypo pigmentation.

Allergic reactions.

Persistent erythema.

.Scarring.

MICRO DERMABRASION:

Microdermabrasion is a popular cosmetic procedure in which the stratum corneum is partially or completely removed by light abrasion. It is a non chemical, non-invasive modality for treating superficial skin blemishes resulting from trauma, aging, or prolonged exposure to sun. Different methods include mechanical abrasion from jets of zinc or aluminium oxide crystals or a roughened surface.

History:

Ancient Egyptians are said to be the first to use Dermabrasion skin care techniques to improve their skin. They used pumice to remove the rough spots and blemishes and made their skin smooth and soft. In mid 90's a skin care technique using aluminium oxide powder for microdermabrasion

was developed in Italy. The process used vacuum and a small orifice to accelerate powder.³

Microdermabrasion became available in United States in 1998 under the Parsian peel brand and in Europe it has been used successfully since 1992 to treat acne, fine lines and wrinkles, unwanted pigmentation and other superficial skin damage.

The new peel system with diamond tome is an evolution in Microdermabrasion. This offers a 21st century skin care technology by eliminating the powders and crystals making the procedure clean and control.

Dr.Arnoldo Canella did an extensive study of controlled Microdermabrasion and concluded that instrumentation operates at 3 different potential depths.⁵⁸

Superficial Epidermis.

Superficial Dermis.

Deep Dermis.

He recommended 3 different hand piece moments.

Axial(in line with the axis of the lesion).

Perpendicular to the axis lesion.

Circular.

The main indication in his study was Cutaneous Striae and other indications include Hyper trophic scar, Melasma, Senile spots, face peeling, and burns scar.

Microdermabrasion represents an excellent treatment modality for hyper pigmentation and offers the prospect of a rapid exfoliation of stratum corneum to make the pigment reduce. It can be used in conjunction with chemical exfoliation to bring non-inflamed form of acne under control.^{59,63}

Mode of action:

It acts by

1. Reducing the thickness of stratum corneum through mechanical exfoliation of dead cells.
2. Stimulation of fibroblasts for collagen regeneration is achieved through vacuum action and mechanical exfoliation of stratum corneum.
3. Stimulation of blood flow aids in the skin nutrition and the regenerative process.

Microdermabrasion basis and treatment:

It is an extremely useful tool for enhancement of the techniques already in common use.

It is found that a single Microdermabrasion treatment may exfoliate as much stratum corneum as 3 consecutive 30% glycolic acid treatment or a single

70% glycolic acid treatment without the chemical irritation potential of acid based peels.⁶⁵

Recognition of the role that vacuum play as part of the Microdermabrasion treatment is vital. Besides being the vehicle by which the crystal is applied and removed, the vacuum provides lymphatic drainage and maintains proper lymphflow.

Comparing chemical exfoliants and Microdermabrasion:

Comparisons of the use of chemical exfoliants such as AHA's and BHA's to microdermabrasion offers some interesting insight into the value of microdermabrasion.⁶⁰ With the use of AHA's and BHA's only minor revision of early aging fine lines, overall rejuvenation of skin health will be seen but not necessarily any scar revision as in microdermabrasion.

Applications:

Application potential varies between the models and is related to the force at which crystals are dispensed on tissue and the vacuum used to draw the tissue into the treatment aperture.

Treatments are arranged as sessions as a part of a 4-8 visit strategy with a goal of accelerating the regeneration of cells to the outer surface of the skin. Optimum results with microdermabrasion are obtained after 4-8 visits at the rate of every 10-14 days. No healing time after dermabrasion,

although rebuilding of the stratum corneum is necessary but rapid, with an immediate return to normal lifetime and minimal discomfort.

Benefits of Microdermabrasion:

Reduction of minor to moderate early aging fine lines with stimulation of fibroblasts for collagen reproduction.⁶⁶

Scar revision.

Hyper pigmentation reduction, in combination with hydroquinone skin lightness and hyperpigmentation risk is minimal as no heat is generated during microdermabrasion.

Overall rejuvenation of skin health.

May be performed during pregnancy.

May be performed shortly after surgery with appropriate adjustment of vacuum/crystal flow. May assist and enhance healing due to increased blood flow.

Can be used in Fitzpatrick's type 4-6 skin types.

No peeling or flaking of skin present and any residual erythema from a moderate exfoliation may be camouflaged immediately.

Time of procedure:

To get the maximum results microdermabrasion procedure may be

performed in 45 minutes session depending upon the pre and post treatment strategies provided.

To get the maximum results microdermabrasion procedure may be performed in 45 minutes session depending upon the pre and post treatment strategies provided.

Indications of microdermabrasion:⁶¹

Sun induced pigmented problems, Melasma, age spots on the face and neck.

Reduction of whiteheads and blackheads formation due to regular exfoliation.

Blending of fine lines and minor wrinkles.

Facilitating ease of sebum and comedone extraction.

Reduction of dormant acne scars and blemishes.

Blending the texture of lazed to non-lazed areas after laser skin resurfacing.

Pretreatment for cosmetic surgery patients.

Reduction of well healed, raised scars and calluses.

Actinic keratosis.

Striae distensia.

Contra Indications:⁶¹

Active uncontrolled or brittle diabetes.

Raised moles, warts, skin tags.

Active acne or undiagnosed skin lesions, viral lesions(Herpes simplex, shingles)

Severe rosacea or telangiectasia

Tattoos.

Oral blood thinners.

Skin cancer and autoimmune diseases.

Vitiligo.

Sinus infection.

Long-term prednisolone or cortisone users.

Sun burned skin.

Vasodilatation disorders.

Precautions:

Patient who had collagen replacement therapy should be advised to wait atleast 10-14 days after the injection before having microdermabrasion treatments.

Botox: It is recommended that microdermabrasion treatment be performed before the injections.

Telangiectasia: Extremely low vacuum setting should be used.

Combination treatments:

These are clinical studies conducted under medical auspices regarding the

use of chemical peel, such as AHA's and BHA's in combination with microdermabrasion.⁶¹ Microdermabrasion can be combined with light chemical peel to increase the effect.^{62,64}

Using Salicylic Acid as pretreatment for Microdermabrasion:⁶¹

Salicylic acid can be applied to the skin prior to microdermabrasion to enhance the treatment for oily, thicker acne prone skins. The benefit includes its anti inflammatory and anti bacterial qualities as well as its keratolytic properties.

Microdermabrasion and active topicals :

Dry maturing skin - Applying products that include Hyaluronic acid to help epidermis retain moisture, anti oxidants to fight damage and transformation growth factors to stimulate collagen and elastin reproduction can produce an excellent result.

Use of Retinols/ Retinoids:

Retinols can be selectively applied in postdermabrasion for enhanced results. With microdermabrasion exfoliation, the retinols can penetrate better and begin to work more quickly because they don't have to fight through the upper layers of stratum corneum.

Vitamin –C Supplements:

Vitamin –C can help resolve erythema by strengthening the capillary walls.

It has anti- inflammatory activity to help the healing process.

Vitamin -E :

Provide protection from free radical damage.

Suggested protocols for Microdermabrasion techniques:

Pretreatment counseling and written consent.

To cleanse the face and remove makeup and pollutants.

Perform a test strip on the inner fore arm of the patient to observe how reactive the skin is.

Divide the face into anatomical sections as follows:

Hairline to Brows.

Brows to upper lip.

Lower lip to mandible.

Mandible to décolleté.

Forehead:

To avoid downward pressure and draw hand piece vertically up. Continue across the forehead from the brows to hairline. Proceed with horizontal passes. To avoid overlap strokes.

Nose:

Begin from mid glabellar area with strokes down the center of the nose and proceed along the sides of the nose around the tip of the nose and then along the nasolabial fold.

Mid face:

Proceed from one side of the face complete by going down. Then proceed with horizontal lines and similarly to treat next side

Chin area:

lower jaw and chin areas should be completed in vertical and then in horizontal passes

Neck:

Reduce the vacuum setting to avoid streaking and blood bruising. Should be performed from the centre outward.

Complications:

The greatest advantage of microdermabrasion is its lack of complications.

In early years, Redness of eyes, Photophobia, Conjunctival congestion, Crystals adherent to cornea and Superficial punctuate keratopathy were present. Now with the latest microdermabrasion the only side effect is Erythema which gradually resolves within hours after treatment.

AIM OF THE STUDY

The aim of the study is to compare the efficacy of glycolic acid and salicylic acid peels in the treatment of acne vulgaris with each other and as combination with microdermabrasion. The agents compared were the following:

Glycolic acid alone

Glycolic acid and microdermabrasion

Salicylic acid alone

Salicylic acid and microdermabrasion

MATERIALS AND METHODS

A total of 40 patients of either sex were enrolled and randomly assigned for the above study. The study was single blind randomized open prospective comparative clinical trial. It was carried out in the Department of Dermatology, Government General Hospital, Chennai during the period of January 2005 to May 2006 based on the following criteria.

Inclusion Criteria

Patients of either sex with moderate to severe acne vulgaris aged between 15 to 30 years.

Patients with facial lesions only.

Both new and already treated patients who have stopped therapy for 15 days.

Exclusion Criteria

Pregnant and lactating women

Women of child bearing age not practicing adequate contraception.

Having any systemic illness. (e.g.) Hypertension, Diabetes, Thyroid problem.

Taking any acne inducing drugs.

History of previous herpes simplex.

Treatment Protocol and Methodology

Patients selected were informed the nature of the study and written consent was obtained from the patients.

The Demographic datas such as age and sex of the selected patients, occupation, marital status and duration of the disease were taken. Other histories like family histories of acne , use of cosmetics and other precipitating factors were noted.

Patients were subjected for general and systemic examination. A thorough Dermatological examination was done and other existing Dermatological lesions apart from acne were recorded.

Dermatological examination for acne included the number of comedones, papules and pustules which were counted and graded as 1 to 4 as follows:

Grade 1	1 to 4 in number
Grade 2	5 to 9 in number
Grade 3	10 or more than 10
Grade 4	15 or more than 15

Clinical photographs of the lesions were taken before commencement of therapy and after completion of therapy. The patients were grouped into 4 batches. The 40 patients who fulfilled the inclusion criteria and selected randomly were grouped as 10 in each batch.

1st Batch: 35%Glycolic acid peel alone

2nd Batch 35%Glycolic acid peel for 3 sittings followed by microdermabrasion after six weeks alternatively with chemical peel with totally three sittings of microdermabrasion.

3rd Batch 20%Salicylic acid peel alone

4th Batch 20%Salicylic acid followed by microdermabrasion as described above.

The patients were advised to avoid cosmetics and were started on priming with retinoic acid at night for 2 weeks and then systemic antibiotics for severe inflammatory lesions.

Method of chemical peeling:

Patients were asked to wash the face with soap and then de-greasing with spirit has been done. The chemical peels selected (either glycolic acid or salicylic acid) was applied over the face starting from the forehead, cheeks, chin, nose and upper lips. After two minutes they were asked to wash the face and slight massage with ice cube pack done. Then sunscreen was applied and patients were advised to protect from sunlight by using sunscreen at day time. This procedure was repeated every two weeks for 6 times and the patients were followed every fortnight. During each visit any side effect attributed to the peel was recorded. The local tolerability of the

peel was evaluated by recording the degree of irritation, erythema, dryness, burning and peeling.

Method of microdermabrasion:

Patients were randomly selected for microdermabrasion who have already undergone chemical peel either with glycolic acid or salicylic acid. Only patients with non-inflammatory lesions like comedones and papules with scarring were selected for this procedure. After washing their face with soap, de-greasing of the skin was done and then microdermabrasion using aluminium oxide crystals was done over the face starting from the forehead, cheek, chin, nose and upper lips in three directions (vertical, horizontal and oblique) by using a pressure of 50 to 60mm Hg vacuum. This procedure was done for 30 minutes and was repeated every fortnight. The side effects were noted during each visit. Totally 3 sittings were done along with chemical peeling in the alternate week and it is as follows:²⁴

- | | |
|--------------|--------------------------------------|
| 1. Mild | 0 to 25 % reduction of acne lesions. |
| 2. Moderate | 26 to 50% reduction of acne lesions. |
| 3. Good | 51 to 75% reduction of acne lesions |
| 4. Excellent | 76 to 100% reduction of acne lesions |

The side/adverse effects were looked for and recorded.

OBSERVATIONS

The following observations were made in the present study. Of the 40 patients, 31 were males and nine were females. Their age ranged from 15 to 30 years with a mean of 21.

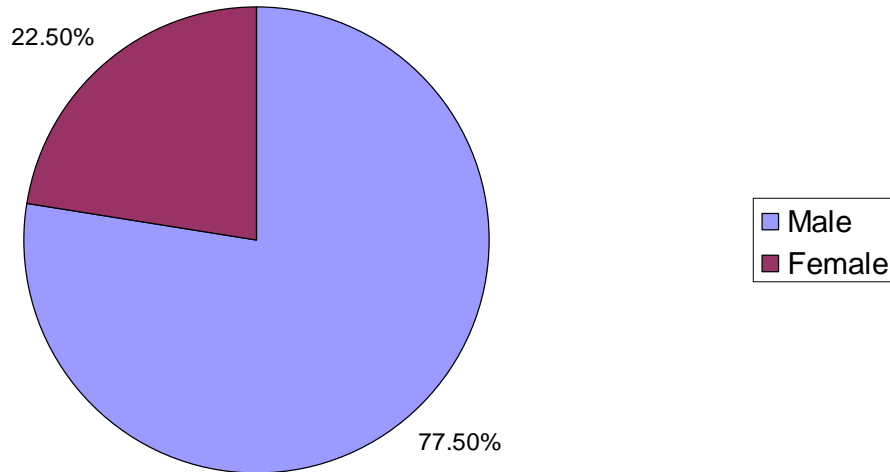
27 patients were students, others were employees in various sectors. None of them were employed in occupation involving substance, which are acnegenic. The duration of acne ranged from 6 months to 8 years with a mean of 17 months.

25% of patients had family history of acne. The following precipitating factors were recorded as per history from the patient. Premenstrual flare up was noted in three patients. Stress was noted in three patients and precipitating factors like diet and summer exacerbation in four patients 2 in each.

Of the 40 patients, 10 had Grade IV acne and 18 had Grade III acne and 12 had Grade II acne.

The dermatological conditions observed in associate with acne in this study were Tinea versicolor (2), Pityriasis Capitis (6), Hirsutism without PCOD (2) and Vitiligo(1).

Sex Distribution



The therapeutic response observed in patients who have been instituted Glycolic acid alone (Group I) were as follows:

There was a moderate reduction of both non-inflammatory and inflammatory acne. After about 4 weeks the total number of lesions came down with few new lesions appearing. At the end of 8 weeks 42% of the lesions subsided with no new lesions. At the end of 12 weeks the overall reduction rate was 51.1%

Table I – Observation of responses with Glycolic acid peels in Group I patients with acne (N -10)

	Average number of lesions (Weeks)							Average number of lesions resolved	Percentage reduction
	0	2	4	6	8	10	12		
Comedones	5.0	4.5	3.9	3.4	2.9	2.7	2.7	2.3	46%
Papules	3.1	2.6	2.3	2.0	1.9	1.3	1.4	1.7	55%
Pustules	0.3	0.3	0.2	0.1	0.1	0	0	0.3	100%
Total lesions	8.4	7.4	6.4	5.5	4.9	4.0	4.0	4.3	51.2%

COMPLICATIONS

In this group, 5 patients had mild irritation, 2 patients had mild erosion of skin and 1 had Hyper pigmentation. With application of Steroids and mild Emollients the side effects subsided during the course of therapy.

Glycolic acid peel alone



Fig no 1 Patient with Grade 2 acne before therapy



After 12 weeks of therapy



Fig no 2 Patient with Grade3 acne Before therapy



After 12 weeks of therapy



Fig no 3 Patient with Grade 3 acne Before therapy



After 12 weeks of therapy

The therapeutic response observed in patients who have been taken Glycolic acid peel and microdermabrasion (Group 2) were as follows:

These patients showed a rapid and sustained reduction in the inflammatory and non-inflammatory lesions. After 6 weeks of treatment, there was a good reduction of lesions and no emergence of new lesions due to the combination of microdermabrasion. At the end of 12 weeks 72.3% of lesions had subsided.

Table 2: Observation of responses with Glycolic acid peel and Microdermabrasion in Group 2 Patients with acne (N-10)

	Average number of lesions (Weeks)							Average number of lesions resolved	Percentage reduction
	0	2	4	6	8	10	12		
Comedones	8.7	7.4	6.9	6.0	5.0	3.9	2.7	6.0	69%
Papules	4.9	4.2	3.6	2.9	2.2	1.6	1.2	3.7	76%
Pustules	0.5	0.4	0.2	0.1	0	0	0	0.5	100%
Total lesions	14.1	12.0	10.7	9.0	7.2	5.5	3.9	10.2	72.3%

COMPLICATIONS

Two patients had Skin irritations and two patients had Hyper pigmentation which subsided on continuation of Microdermabrasion. In this group patients

Glycolic acid with microdermabrasion



Fig no 4 Patient with Grade 4 acne before therapy



After 12 weeks of therapy

Glycolic acid with microdermabrasion



Fig no 5 Patient with Grade 4 acne before therapy



After 12 weeks of therapy



Fig no 6 Patient with Grade 3 acne before therapy



After 12 weeks of therapy

with mild acne scar were more satisfied. Mild erythema was present after microdermabrasion which subsided by itself in few hours.

The therapeutic response observed in the patients who have been taken salicylic acid peel alone (Group 3) were as follows:

Response to therapy occurred after 2 to 4 weeks of treatment. There was a sustained reduction of lesions and at the end of 12 weeks 63.8% of lesions had subsided.

Table 3: Observation of responses with salicylic acid peel alone in Group 3 patients with acne (N -10)

	Average number of lesions (Weeks)							Average number of lesions resolved	Percentage reduction
	0	2	4	6	8	10	12		
Comedones	5.7	5.0	4.5	3.9	3.3	2.7	2.2	3.5	61.4%
Papules	4.1	3.4	3.0	2.5	2.1	1.7	1.6	2.5	61%
Pustules	0.7	0.7	0.3	0.2	0.1	0.1	0	0.7	100%
Total lesions	10.5	9.1	7.8	6.6	5.5	4.5	3.8	6.7	63.8%

Salicylic acid peel alone



Fig no 7 Patient with Grade 4 acne before therapy



After 12 weeks of therapy



Fig no 8 Patient with Grade III before therapy



After 12 weeks of therapy.

COMPLICATIONS: In this Group 3 patients had mild irritation, 2 patients had Post inflammatory hyper pigmentation and 1 had Photo sensitivity. All the patients had peeling of skin. After applying mild emollients and steroids along with peeling, the side effect subsided.

The therapeutic response observed in patients who had been instituted combination of salicylic acid peel and microdermabrasion were as follows (Group 4):

After the salicylic acid peel for 6 weeks (Once every 2 weeks) the patients were started on microdermabrasion combined with salicylic acid peels. There was a rapid and sustained reduction of both inflammatory and non-inflammatory acne lesions. At the end of 12 weeks most of the lesions subsided with an overall reduction of 80.3%.

Table 4: Observation of response with salicylic acid peel and microdermabrasion in Group 4 patients with acne (N-10):

	Average number of lesions (Weeks)							Average number of lesions resolved	Percentage reduction
	0	2	4	6	8	10	12		
Comedones	8	7.1	6.3	5.1	3.8	3.0	2.2	5.8	72.5%
Papules	5.7	4.9	4.6	3.5	2.2	1.2	0.8	4.9	86%
Pustules	1.6	1	0.6	0.2	0	0	0	1.6	100%
Total lesions	15.3	13.0	11.5	8.8	6.0	4.2	3.0	12.3	80.3%

Salicylic acid with Microdermabrasion



Fig no 9 Patient with Grade III Acne before therapy



After 12 weeks of therapy

Salicylic acid with Microdermabrasion



Fig no 10 Patient with Grade 4 acne Before therapy



After 12 weeks of therapy

Salicylic acid with Microdermabrasion



Fig no 11 Patient with Grade 4 acne before therapy



After 12 weeks of therapy
Salicylic acid with microdermabrasion



Fig no 12 Patient with Grade 3 acne Before therapy



After 12 weeks of therapy

COMPLICATIONS:

Four patients had initial irritation and peeling of skin occurred in all patients during the first three peels. Photo sensitivity was observed in 1 patient and hyper pigmentation was observed in 4 patients which subsided with continuation of Microdermabrasion

On the whole patients treated with only chemical peel at the end of 12 weeks Group 1 (Glycolic acid peel) had 51.1% reduction of acne lesions and Group 3(Salicylic acid peel) had 63.8% reduction of acne lesions

Table 5: Comparison of Patients treated with Glycolic acid and Salicylic acid peel alone (average number of lesions)

Weeks	Glycolic acid peel	Salicylic acid peel
0	8.4	10.5
2	7.4	9.1
4	6.4	7.8
6	5.5	6.6
8	4.9	5.5
10	4.0	4.5
12	4.1	3.5
% of Improvement	51.2%	63.8%

Table 7: Comparison of patients treated with Glycolic acid alone and Glycolic acid with microdermabrasion: (average number of lesions)

Weeks	Glycolic acid	Glycolic acid and microdermabrasion
0	8.4	14.1
2	7.4	12
4	6.4	10.7
6	5.5	9.0
8	4.9	7.2
10	4.0	5.5
12	4.1	3.9
% of Improvement	51.2%	72.3%

There was a vast improvement in patients treated with both Glycolic acid peel and Microdermabrasion than Glycolic acid peel alone. The side effects like Hyper pigmentation improved with the Microdermabrasion.

Comparison of improvement of Acne Lesions

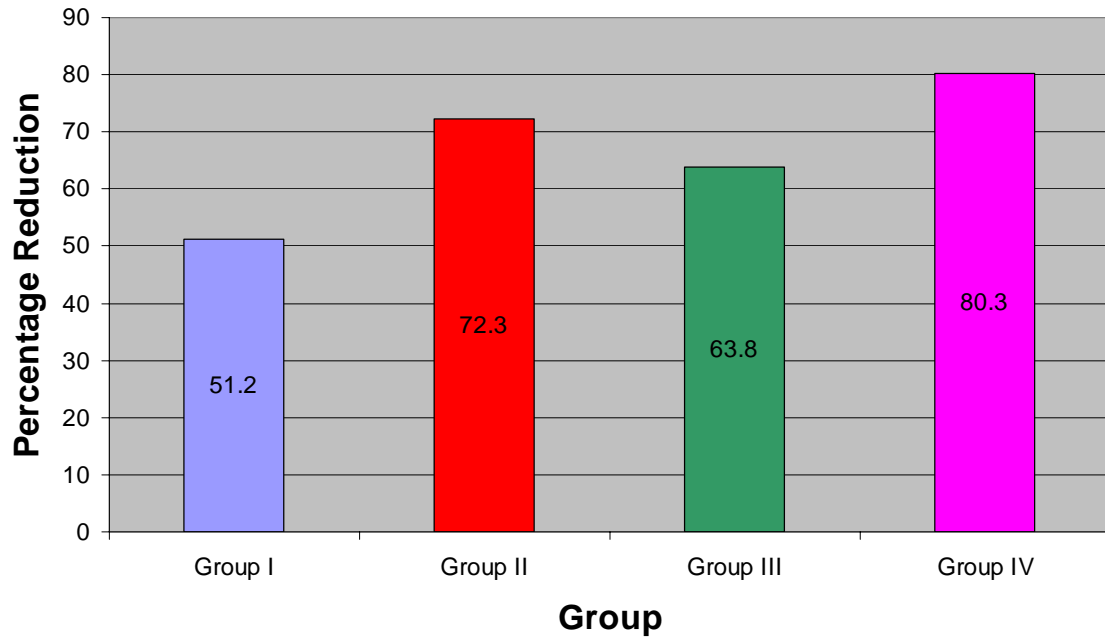


Table 8: Comparison of patient treated with Salicylic acid and salicylic acid with Microdermabrasion: (average number of lesions)

Weeks	Salicylic acid	Salicylic acid and microdermabrasion
0	10.5	15.3
2	9.1	13.0
4	7.8	11.5
6	6.6	8.8
8	5.5	5.1
10	4.5	4.2
12	3.4	3.0
% of Improvement	63.8%	80.3%

The comparison chart clearly shows a 16.5% improvement when microdermabrasion is combined with Salicylic acid.

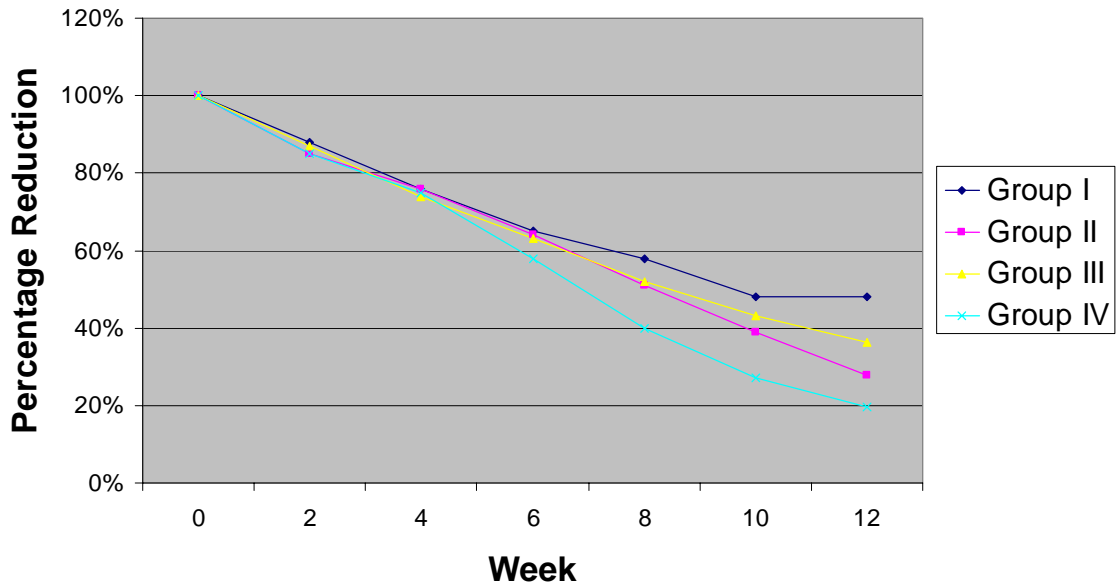
Table 9: Comparison of percentage of reduction of acne lesions after every 2 weeks in all 4 groups

Weeks	Group I	GroupII	GroupIII	GroupIV
2	12%	15%	13%	15%
4	24%	24%	26%	25%
6	35%	36%	37%	42%
8	42%	49%	48%	60%
10	52%	61%	57%	73%
12	52%	72.3%	63.8%	80.3%

Table 10: Improvement Grade at the end of 12 weeks of therapy in patients from Group I to Group IV:

Therapy	Group I	Group II	Group III	Group IV
Group I	0	3	7	0
Group II	5	5	0	0
Group III	2	3	5	0
Group IV	7	3	0	0

Comparison of rate of decrease of Acne Lesions



Excellent response was observed in seven patients and good response in three patients in Group IV (Salicylic acid and Microdermabrasion). In Group II patients, excellent response was noted in five patients and good response for the rest of the patients in the group. Moderate response was observed in seven patients in Glycolic acid peel (7/10) and in five patients in Salicylic acid peel (5/10).

DISCUSSION

Acne vulgaris, the “Stigma of Adolescence” exceeds all other causes of suffering in adolescence age group. Many patients do not seek physician’s advice. Only a few cosmetically conscious adolescents who are to be married shortly came for treatment, however mild the condition may be.

Family history of acne vulgaris was present in 25% of patients. Hereditary factor in the causation of acne has been documented.

Three female patients reported pre-menstrual flare up .this is said to be due to pre-menstrual change in the hydration of pilosebaceous epithelium. Exacerbation of acne lesion during the time of physical and mental stress and summer exacerbation have all been observed.

Acne therapy aims at reduction of sebum production, correcting the abnormal ductal keratinisation, reducing the colony of propionibacterium acnes and preventing the release of inflammatory mediators that are basically responsible for the pathogenesis of acne.

GROUP I: GLYCOLIC ACID PEEL ALONE:

Patients in this group showed a moderate response in both inflammatory and non-inflammatory lesions after the second sitting. There was 24% reduction of acne lesions at the end of 4 weeks and 42% reduction at the end of 8 weeks. At the end of 12 weeks of treatment, a good amount of lesions subsided with an overall reduction of 51.2%. This is less compared to the study by Dr. Vinay Saraf.⁶⁷ Good response was noted in three patients and moderate response in 7 patients.

The side effects such as irritation, erythema, erosion of skin, peeling of skin and dryness which have been seen in 8 patients in the present study has also been noted in the literature.

GROUP:II GLYCOLIC ACID PEEL & MICRODERMABRASION:

Patients in this group showed a sustained and satisfactory reduction in the inflammatory and non-inflammatory lesions. Reduction in the total lesion counts from the 4th week onwards. At the end of 4 weeks the reduction rate of acne lesions was 24% which was similar to group I as only glycolic acid peel was used up to this period. On subsequent use of microdermabrasion, the rate of reduction was 49% at the end of 8 weeks and 61% at 10 weeks. At the end of 12 weeks total lesion reduced to about 72.3%. At the end of 10 weeks of treatment, 2 patients had skin irritations

and 2 patients had hyper-pigmentation which subsided on continuation of microdermabrasion.

A study by Lloyd RI, ⁶³ using microdermabrasion alone in acne showed a 38 % of excellent and 34% of good response as compared to our study, which showed a 50% of excellent and 50% good results. This improvement is probably due to the combination of chemical peel with microdermabrasion.

By the end of therapy, five patients had excellent response and five patients had good response.

GROUP: III SALICYLIC ACID PEEL ALONE:

Reduction of both inflammatory and non-inflammatory lesions was noted from the second sitting onwards in this group of patients. At the end of 4 weeks, there was 26% of reduction of acne lesions and 48% reduction at the end of 8 weeks. At the end of 12 weeks of treatment the total lesion counts reduced by 63.8%.

In a study conducted by Grimes²⁴, salicylic acid peel on acne showed an excellent to moderate response in 88% and mild clearance in 12%. In this study excellent to good response in 50% of patients and moderate response in 50% of patients. Salicylic acid peel showed better response in this study when compared to the literature.

Side effects were noted in 20% of patient in Grimes study,²⁴ which showed crusting, hypo pigmentation and transient hyper pigmentation. In the present study 30% had side effects like mild skin irritation, erythema, photosensitivity and hyper pigmentation, and all the patients had peeling of skin .After applying mild steroids and emollients along with peeling results in reduction of the side effects.

GROUP: IV SALICYLIC ACID PEEL & MICRODERMABRASION:

Rapid and sustained reduction of both inflammatory and non-inflammatory acne lesions was noted after 4 weeks of starting the therapy. At the end of 4 weeks, there was 25% reduction of acne lesions, which was similar to group III. Then on subsequent use of microdermabrasion along with the peel after 6 weeks, there was a reduction of 42% at the end of 6 weeks and a reduction of 60% at the end of 8 weeks. At the end of 12 weeks most of the lesions subsided with an overall reduction of 80.3% of lesions.

In Grimes study,²⁴ salicylic acid peel showed 88% moderate to excellent response and microdermabrasion alone showed 38% of excellent and 34% of good response. In this study seven patients showed excellent response and three patients showed good response.

Side effects such as irritation, photosensitivity and hyperpigmentation were noted in 5 patients which subsided with continuation of microdermabrasion. These side effects were consistent with the literature.

Salicylic acid peel was shown to be superior to glycolic acid peel for acne (without microdermabrasion) in reducing acne lesion after 12 weeks of treatment.

Likewise, salicylic acid peel with microdermabrasion was shown to be superior to glycolic acid peel with microdermabrasion in reducing total acne lesion count after 12 weeks of treatment.

Overall, there is a good improvement in the acne lesions if microdermabrasion was used along with the chemical peel for the treatment of acne. Side effects like salicylism and infections, which are found in the literature, are not noted in this study.

CONCLUSION

1. Patients in whom chemical peeling was followed with microdermabrasion showed better results compared to patients in whom chemical peeling alone was done.
2. If chemical peeling is done alone or combined with microdermabrasion, salicylic acid peel is superior to glycolic acid peel in the treatment of acne.
3. Though salicylic acid peel with microdermabrasion showed better results, the improvement rate was more with glycolic acid with microdermabrasion than salicylic acid peel with microdermabrasion.
4. Hyper-pigmentation is more common side effect with salicylic acid peel, while erosion and burning sensation were more with glycolic acid peel.
5. Though the side effects observed with these peels reduced by itself, application of emollients, mild steroids and sun screeners hastened the recovery.

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PROFORMA

Department of Dermatology

Madras Medical College and Research Institute

Chennai

Date:

SIno.:

OP.no:

Name:

Age

:

Sex :

Address:

Occupation :

Marital status:

Present complaints:

Precipitating factors:

Diet :

Sunlight:

Stress :

Premenstrual flare up:

H/O Herpes infection:

Others:

Previous treatment history:

Topical:

Oral:

Personal history:

Past history :

Family history :

Drug history :

Steroids:

ATT :

Anti epileptics:

Anti depressants:

Allergy history:

General examinations:

Systemic examination:

Dermatological examination:

Acne Duration

Distribution

Grade

Other dermatosis

Treatment

Side effects	2nd week	4th week	6th week	8th week	10th week	12th week
Erythema						
Burning						
Peeling						
Erosion						
Irritation						
Blister formation						
Dryness						

Post therapeutic follow up

Lesions	0	2	4	6	8	10	12
Comedones							
Papules							
Pustules							
Total lesions							

CASE RECORD BOOK

Patient No: _____

Patient Names with Initials: _____

Age: _____

Sex: _____

INFORMED CONSENT FORM

I, _____, exercising my free power of choice hereby give my consent to perform chemical peel/Microdermabrasion on my face for the treatment of acne vulgaris. I was informed fully about the effects and side effects of the above treatment. I understand that I may be treated with the above methods for the disease I am suffering from. The attending physician informed the purpose of the clinical study and the nature of treatment to me and it was up to my satisfaction.

Signature of the
Attending Physician

Signature / Thumb
Impression of Patient

Date:

Date:

MASTER CHART

SN O	AGE	SEX	OCC	DUR	PPF	PT	RH	TR	GR-0	GR-4	GR-8	GR-12	OD	%R	S/E
1	19	M	O	6 M	PS	-	-	S+D	4	3	2	1		83%	HP
2	18	M	s	1Y	-	+	-	G	3	3	2	2		54%	MI
3	23	M	s	8M	-	-	+	S	4	3	2	2	TV	62%	
4	17	M	O	6M	-	-	+	G	3	2	2	0		50%	
5	21	M	s	1Y	-	+	-	S+D	4	4	2	2	PC	100%	MI
6	18	M	s	6M	-	+	-	G+D	4	4	2	1	PC	82%	
7	22	M	O	8M	-	-	-	G	3	2	2	1		42%	E
8	19	M	s	2Y	D	-	-	G+D	3	2	1	1		86%	
9	18	M	s	9M	-	-	+	G+D	2	2	1	2		89%	MI
10	20	M	O	10M	-	-	+	G+D	4	3	3	2	PC	62%	
11	21	M	s	11M	-	-	+	S	4	3	3	1	TV	62%	MI
12	18	M	s	6M	-	-	-	S	2	1	1	1		71%	HP
13	25	F	O	6M	PM	-	-	G	2	1	1	1		67%	MI
14	21	M	s	3Y	-	-	-	G	2	1	1	1		67%	
15	18	M	s	6M	-	-	-	G	1	1	1	1		50%	MI
16	22	M	O	1Y	St	-	+	S	2	1	1	1		67%	
17	21	F	s	6M	-	-	-	G	2	1	1	1		50%	HP
18	20	F	O	1Y/2Y	PM	-	-	S+D	2	1	1	1	H/P C	86%	MI
19	23	M	O	9M	-	-	-	G+D	2	1	1	1		89%	
20	16	M	s	8M	-	+	+	G	2	1	1	1		50%	MI

21	33	F	O	5Y	-	+	+	S	3	2	1	1	H	80%	
22	21	M	O	2Y	-	+	-	S	3	2	2	1		75%	MI
23	19	M	s	5Y	-	+	-	S+D	3	2	2	1		77%	HP
24	19	M	s	2Y	-	+	-	S+D	4	3	2	1		76%	MI
25	29	M	s	1Y	-	-	+	S+D	3	2	1	1		77%	
26	16	F	s	6M	PM	+	-	S	2	2	2	1		56%	MI
27	29	M	O	2Y	St	+	-	S	3	2	1	1	PC	67%	
28	19	F	s	1/2Y	-	+	-	G+D	4	4	3	2		68%	MI
29	19	F	s	1Y	St	-	-	S+D	4	3	2	1		80%	HP
30	21	M	s	2Y	Su	+	-	S	3	2	2	2	V	50%	HP
31	18	M	s	2Y	-	-	-	G	3	2	2	2		33%	MI
32	17	F	s	1Y	-	-	-	G+D	4	3	2	2		67%	HP
33	20	M	s	2Y	D	-	-	G+D	3	2	2	1		75%	
34	26	M	O	3Y	-	+	-	S+D	3	3	2	2	PC	64%	HP
35	25	M	O	3Y	Su	-	-	S+D	3	2	2	1		60%	
36	18	M	s	8M	-	+	-	G+D	3	2	2	1		67%	
37	20	F	s	1-1/2Y	-	+	-	G	3	2	2	1	H	50%	E
38	19	M	s	1Y	-	+	+	S+D	3	2	1	1		60%	MI
39	22	M	s	1Y	+	+	-	S	2	2	2	2		45%	
40	20	M	s	8M	+	+	-	G+D	3	2	2	1		60%	HP

KEY TO THE MASTER CHART

D	-	Diet
DUR	-	Duration
E	-	Erythema
FH	-	Family History
G	-	Glycolic Acid Peeling
GR	-	Grade
G+D	-	Glycolic Acid with Microdermabrasion
H	-	Hirsutism
HP	-	Hyper Pigmentation
M	-	Month
MI	-	Mild Irritation
O	-	Other Occupation
OD	-	Other Dermatological Problem
PC	-	Pityriasis Capitis
PPF	-	Precipitating Factors
PT	-	Previous Treatment
PM	-	Pre-Menstrual Flare
PS	-	Photosensitivity
S	-	Salicylic Acid
s	-	Student
S+D	-	Salicylic Acid with Microdermabrasion

S/E	-	Side Effects
St	-	Stress
Su	-	Summer Exacerbation
TR	-	Treatment
TV	-	Tinea Versicolor
V	-	Vitiligo
Y	-	Year
%R	-	Percentage Reduction