Acne, acne vulgaris, skin disease, hair-follicle disease, pilosebaceous gland disorder, sebaceous gland, comedones, papules, pustules, blackheads etc.

1. Introduction
Acne vulgaris (acne) is the formation of comedones, papules, pustules, nodules, and/or cysts as a result of obstruction and inflammation of pilosebaceous units (hair follicles and their accompanying sebaceous gland). Acne develops on the face and upper trunk. It most often affects adolescents. Acne is the most common disease treated by dermatologists based upon data from the 1996 U.S.Census. People of all races and ages get acne. But it is most common in teenagers and young adults. An estimated 70 percent of all people between the ages of 12 and 24 have acne outbreaks at some point. Some people in their forties and fifties still get acne [1, 2]. The lifetime prevalence of acne approaches 90%. Acne is a chronic disease with the highest prevalence in adolescents. The onset of acne occurs during the onset of puberty, which is at a younger chronological age in females than in males. Both females and males have acne that extends into adulthood, with females often having a higher prevalence of the disease as well as more severe forms during adulthood. When untreated, acne usually lasts for several years until it spontaneously remits. Most cases continue into the mid-twenties, and there is evidence that the duration of acne may last into middle age for most women [3, 4].

1.1 Risk factors [1, 2, 4]
- **Age:** 12 to 24 years; Research has shown that 85% of young people between the ages of 12 and 24 have acne [5].
- **Genetic predisposition:** The concordance rate for the prevalence and severity of acne among identical twins is high. One study concluded that 81% of variance in acne was attributable to genetics, and only 19% to environmental factors [6-8].
- **Greasy skin/increased sebum production:** Sebaceous follicle size and the number of lobules per gland are increased in patients with acne. Androgens stimulate sebaceous glands to enlarge and produce more sebum, which is most prevalent during puberty. Sebum production is fairly high during the first 6 months of life, declines and remains stable throughout childhood, and dramatically increases during puberty [9].
- **Medications:** Acne form eruptions can be caused or exacerbated by some medications, including androgens, corticosteroids (topical, oral, or injected), antiepileptic medications (e.g., dilantin), isoniazid, lithium, and ACTH [9].
- **Endocrine disorders:** Patients with endocrine disorders such as polycystic ovary syndrome, hyperandrogenism, and precocious puberty are more likely to have severe acne.
- **Dietary factors:** Few studies have examined the role of diet in acne. Several studies focusing on chocolate consumption found no effect on acne. While Western diet has been associated with increased incidence of acne, these observations are limited by their ability to separate genetic factors from environmental and dietary influences [2].
- **Female gender/estrogens:** The role of estrogen in acne is unclear, but estrogen is known to decrease sebum production. Many women note worsening of their acne prior to menstruation, and oral contraceptives often help mitigate this cyclical worsening. Suppression of sebum production
requires higher doses of estrogen than does suppression of ovulation [5, 6].

- **Obesity/insulin resistance:** Insulin and insulin-like growth factor (IGF) can stimulate keratinocytes and sebaceous glands. Elevated IGF-1 levels are found in women with postadolescent acne and obesity has been found to be associated with an increased prevalence of acne in people aged 20 to 40 years. However, no association between obesity and acne was found in patients aged 15 to 19 years [7, 10].

- **Hyperandrogenism:** The rise in circulating androgens at the onset of puberty is associated with increased production of sebum and the development of comedonal acne, but most patients with acne have normal androgen levels. Rare cases may be associated with pathologically elevated androgen levels due to an underlying disorder, such as polycystic ovary disease [11, 12].

### 1.2 Etiology [12–16]

Acne is polygenic and multifactorial. Four main pathogenetic factors contribute to the disease:

- **Sebaceous gland hyperplasia and excess sebum production.**
- **Abnormal follicular differentiation.**
- **Propionibacterium acnes colonization.**
- **Inflammation and immune response.**

The initial step in the development of acne is the formation of the microcomedo. Earliest changes in the hair follicle occur when the follicular canal becomes blocked with abnormally keratinized desquamating cells. This plug starts above the opening of the sebaceous gland into the follicular canal and causes gradual expansion of cells and sebum within the canal. The plug becomes visible at the skin surface as a white papule (“white-head,” or closed comedo). If the opening of the follicular canal dilates, this plug protrudes from the canal and turns a dark color (“blackhead,” or open comedo) [2]. Follicular keratinocytes that exhibit increased cohesiveness do not shed normally, leading to retention and accumulation. Androgens stimulate enlargement of sebaceous glands and increased sebum production, and the abnormal keratinaceous material and sebum collect in the microcomedo. Although sebum production increases during adolescence, particularly in boys, increased sebum alone does not cause acne. This leads to a buildup of pressure, and whorled lamellar concretions develop. At this stage, a non-inflammatory comedo may be seen clinically [2, 4].

This microenvironment allows the proliferation of P. acnes. These enzymes hydrolyze sebum into free fatty acids, which stimulate the inflammatory process. Chemotactic factors are released by this reaction, attracting neutrophils. As the follicular wall becomes inflamed, an erythematous papule appears at the skin surface. With increased sebum production, obstruction and bacterial colonization, the follicular unit ruptures, spilling its contents into the dermis. The inflow of neutrophils causes the formation of pustules. Continuation of severe inflammation leads to formation of nodules and subsequent cysts [2].

With increased pressure and recruitment of inflammatory mediators, the microcomedo may rupture and release immunogenic keratin and sebum, thus stimulating an even greater inflammatory response. Depending on the specific inflammatory cells present, suppurative pustules or inflamed papules, nodules, or cysts may develop. If a sufficient amount of inflammation and tissue damage results, postinflammatory hyperpigmentation and scarring may result.

![Figure 1: Stages of Acne](image-url)
There are many types of acne or pimples. The most common types are:

- **Whiteheads**: These are pimples that stay under the surface of the skin.
- **Blackheads**: These pimples rise to the skin's surface and look black. The black color is not from dirt.
- **Papules**: These are small pink bumps that can be tender.
- **Pustules**: These pimples are red at the bottom and have pus on top.
- **Nodules**: These are large, painful, solid pimples that are deep in the skin.
- **Cysts**: These deep, painful, pus-filled pimples can cause scars.

**Most common types are as follows:**

1.3.1 **Inflamatory Acne**

Inflamatory acne is a more painful, serious form of acne. Allergy Escape reports that inflamatory acne can develop into the more severe phases of acne, such as cysts or nodules. Since acne can have lasting consequences, it is important to understand why it occurs and how it can be treated. Inflamatory acne effects more than one’s skin, it can also leave scars on the self-esteem and social life [11, 12].

This type of acne includes pustule, papule, cysts or nodule. The cysts can be large and quite painful. The nodules will be inflamed.

1.3.2 **Acne Conglobata**

This type of acne is classified as being the most severe form of Acne vulgaris. It is more commonly found in males than females. It has been characterized by many large lesions, which are interconnected. There will also be a widespread of blackheads. It can cause severe damage to the skin. It is commonly found on the chest, face, back, upper arms, thighs and buttocks. Acne conglobata is a highly inflamatory disease presenting with comedones, nodules, abscesses, and draining sinus tracts. Symptoms of acne conglobata include swelling and pain in the lesions with severe inflammation and sensitivity are the common symptoms of this acne. The cysts may erupt and the lesions ooze out also [12].

1.3.3 **Acne Vulgaris**

Acne vulgaris (cystic acne or simply acne) is a common human skin disease. Acne vulgaris starts when oil and dead skin cells clog up your pores. Some people call it blackheads, blemishes, whiteheads, pimples, or zits. It characterized by areas of skin with seborrhea (scale red skin), comedones (blackheads and whiteheads), papules (pinheads), pustules (pimples), nodules (large papules) and possibly scarring. Acne affects mostly skin with the densest population of sebaceous follicles; these areas include the face, the upper part of the chest, and the back. Severe acne
is inflammatory, but acne can also manifest in non inflammatory forms \cite{4, 11, 16}.

1.3.4 Acne Rosacea

Rosacea is a chronic skin condition that makes your face turn red and may cause swelling and skin sores that look like acne. Symptoms include redness of the face, blushing or flushing easily, a lot of spider-like blood vessels (telangiectasia) of the face, red nose (called a bulbous nose), acne-like skin sores that may ooze or crust \cite{12}.

1.3.5 Acne Fulminans

Acne fulminans is a catastrophic, scarring disease characterized by a sudden appearance of massive, inflammatory tender nodulo-ulcerative lesions over the chest and the back, associated with fever and arthralgia, occurring exclusively in teenage boys. The pathogenesis of this disease is unknown. Hypersensitivity, delayed hypersensitivity response to Propionibacterium acnes and immune complex mediated mechanisms because of decreased complement levels have been reported as some of its possible mechanisms \cite{4, 12}.

2. Diagnosis \cite{11, 12, 14}

2.1. Physical exam

Non-inflammatory acne manifests as whiteheads (closed comedones) and blackheads (open comedones). Inflammatory lesions begin as microcomedones but may develop into papules, pustules, nodules, or cysts. Both types of lesions are found in areas of dense sebaceous glands. Acne may affect only the face, but the chest, back, and upper arms are often involved. Individuals may form more hypertrophic scars at areas of prior involvement. Severe nodulocystic acne presenting with fever, arthralgia, myalgia, hepatosplenomegaly, and osteolytic bone lesions suggests acne fulminans.

2.2. Laboratory evaluation

Routine endocrinologic testing is not indicated for the majority of patients with acne. In patients with acne and evidence of hyperandrogenism, hormonal evaluation for free testosterone, dehydroepiandrosterone sulphate (DHEA-S), luteinising hormone (LH), follicle stimulating hormone (FSH) is done.

Routine microbiologic testing is unnecessary in the evaluation and management of patients with acne. If lesions centered in the perioral and nasal areas are unresponsive to conventional acne treatments, bacterial culture and sensitivities to evaluate for gram-negative folliculitis are considered.

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hormonal Evaluation</strong></td>
<td>Elevated total testosterone, luteinising hormone (LH), follicle stimulating hormone (FSH)</td>
</tr>
<tr>
<td>Rarely required.</td>
<td>Free testosterone is the most sensitive test to establish the presence of hyperandrogenism; ordered only when there is concern for hyperandrogenism.</td>
</tr>
<tr>
<td>If laboratory tests are abnormal, referral to an endocrinologist is indicated.</td>
<td></td>
</tr>
<tr>
<td><strong>Bacterial Culture</strong></td>
<td>Elevated numbers of P.acne</td>
</tr>
<tr>
<td>Rarely required.</td>
<td>Ordered only when standard treatments are not efficacious and there is clinical suspicion of gram-negative folliculitis. Sample should be collected from multiple inflammatory lesions and also from a pustule if present. Treatment based on isolation and sensitivities of bacteria.</td>
</tr>
</tbody>
</table>

Figure 6: Acne Vulgaris

Figure 7: Acne Rosacea

Figure 8: Acne Fulminans
**TABLE 2: Tests to consider**
**Table 3: Differential Diagnosis**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Differentiating signs/symptoms</th>
<th>Differentiating tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acne keloidalis nuchae</td>
<td>Most often seen in black patients; lesions are typically localized to the posterior neck. They begin as papules and pustules and may progress to confluent keloids.</td>
<td>Clinical differentiation usually suffices</td>
</tr>
<tr>
<td>Acneiform eruptions</td>
<td>Possible etiologies to consider include oral medications, topical corticosteroids, contrast dye, testosterone, and cosmetic products. Clinical clues include the abrupt onset of lesions within days of exposure, widespread involvement, atypical locations, atypical age, and improvement with cessation of medication or exposure.</td>
<td>Clinical diagnosis usually suffices.</td>
</tr>
<tr>
<td>Chloracne</td>
<td>Comedones, pustules, and cysts are most commonly found behind the ears and in the axillae and groin. Consider exposure to halogenated aromatic hydrocarbons (e.g., chlorinated dioxins and dibenzofurans). Patient may have systemic complications such as ophthalmic, neuropathic, hepatic, and lipoprotein abnormalities.</td>
<td>Clinical differentiation usually suffices. Consider laboratory tests such as liver enzymes and lipid panel.</td>
</tr>
<tr>
<td>Favre-Racouchot syndrome</td>
<td>Multiple open and closed comedones on the periorbital and malar areas, usually on older people with significant chronic sun exposure. Typically noninflammatory.</td>
<td>Clinical differentiation usually suffices. Skin biopsy shows increased elastic tissue with thickened, tortuous fibers in the upper and mid-dermis.</td>
</tr>
<tr>
<td>Folliculitis (nongram-negative)</td>
<td>Common condition that manifests as erythematous papules and pustules, which are follicularly based. As opposed to acne, folliculitis often affects the trunk and extremities.</td>
<td>Clinical differentiation usually suffices. Pustular lesions that do not respond to typical acne antibiotics may be cultured.</td>
</tr>
<tr>
<td>Gram-negative folliculitis</td>
<td>Occurs in patients with acne treated with long-term antibiotics that subsequently develop pustules or nodules on the anterior nares, which then spreads. Can also occur in people after hot tub immersion, as well as in HIV patients.</td>
<td>Lesions may be cultured to isolate the gram-negative bacteria if acneiform lesions do not respond to typical antibiotic regimen.</td>
</tr>
<tr>
<td>Lupus miliaris disseminatus faciei</td>
<td>Firm yellowish-brown or red smooth papules periorbitally and characteristically on the eyelid skin.</td>
<td>Diascopy reveals yellowish-brown lesions. Skin biopsy reveals caseating epithelioid cell granulomas.</td>
</tr>
<tr>
<td>Milia</td>
<td>White keratinaceous cysts that are found on the face, particularly on the eyelids. Lesions are fixed and persistent.</td>
<td>Skin biopsy shows small cysts derived from the infundibulum of the vellus hair.</td>
</tr>
<tr>
<td>Perioral dermatitis</td>
<td>Common perioral eruption of papules and pustules on an erythematous and/or scaling base, often the result of topical corticosteroid use. Localized symmetrically around the mouth, with a clear zone around the vermilion border.</td>
<td>Clinical differentiation usually suffices.</td>
</tr>
</tbody>
</table>

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Pyoderma faciale

- Rapid onset of reddish or cyanotic erythema with abscesses, cysts, and occasionally sinus tracts. No comedones and no involvement of back or chest.
- Skin biopsy shows a grenz zone and mixed inflammatory infiltrate in the upper and mid-dermis, with extravasation of RBCs and hemosiderin deposition.

Rosacea

- Typically affects older people than acne vulgaris, most often women aged 30 to 50 years.
- Various forms, but classically presents with background erythema and telangiectasias, and inflammatory papules and pustules occasionally superimposed.
- Environmental factors often act as triggers.
- Clinical differentiation usually suffices.

Syringoma

- Noninflammatory small papules that occur primarily on the eyelids and upper cheeks, usually multiple.
- Disproportionately more prevalent in Japanese women.
- Skin biopsy shows a dense fibrous stroma with dilated cystic spaces that have small comma-like tails resembling tadpoles.

Adenoma sebaceum (angiofibromas)

- Small, translucent, waxy papules distributed symmetrically over the central cheek, nose, and forehead.
- Multiple lesions associated with tuberous sclerosis.
- Skin biopsy shows dermal fibrosis and vascular proliferation and dilatation.

3. Current Treatment

Acne treatments work by reducing oil production, speeding up skin cell turnover, fighting bacterial infection, reducing the inflammation or doing all four.

3.1 Synthetic Drug Treatment

Active ingredients may have one or more of the following properties:
- Antibiotics and antiseptics to reduce counts of *Propionibacterium acnes* bacteria: (e.g., doxycycline, tetracycline, clindamycin, topical clindamycin, daptomycin, topical erythromycin, minocycline, trimethoprim/sulfamethoxazole.)
- Anti-inflammatory effects to calm red, inflamed skin by inhibiting lipase production by *P. acnes*
- Anti-oxidants to protect cells from damage by free radicals
- Retinoid-like agents (e.g., topical tretinoin, adapalene, tazarotene, isotretinoin)
- Comedolytics to unplug blocked follicles (comedones)
- Keratolytics to peel off surface scale (exfoliants)
- Agents that affect keratinisation, i.e., that normalise skin cell maturation

i. Antibiotics Treatment

Usually acne antibiotics work pretty fast; they can help to soothe the skin and prevent acne scars. Most of the antibiotics are taken orally; it is a proved and safe method. Oral antibiotics kill the bacteria that cause acne and decrease the level of hormones that cause inflammation.
- Erythromycin
- Clindamycin
- Doxycycline
- Minocycline

ii. Hormonal Treatment

Hormonal acne treatment is one of the many options that people have explored, and a lot has been discovered about how hormones can be used to treat acne. The reason that hormonal acne treatment is effective is due to the fact that acne can be caused by hormones. This means that only those that have hormonal acne will be able to benefit from the hormonal acne treatment, as using the hormones to help stabilize the internal balance will reduce the production of acne.

Hormonal therapy is initiated in following cases:
- When acne has not responded to standard treatment (topical and oral antibiotics or its combination)
- When there are contraindications to intake of isotretinoin
- When rapid relapse has occurred after repeated courses of isotretinoin
- When menstrual control and/or contraception are required alongside acne therapy

iii. Antiseptic Treatment

Antiseptics are chemical agents that slow or stop the growth of micro-organisms (germs) on external surfaces of the body and help prevent infections. Antiseptics should be distinguished from antibiotics that destroy micro-organisms inside the body, and from disinfectants, which destroy micro-organisms found on inanimate surfaces.
Some of the antiseptics used are:
- Azelaic acid
- Benzoyl peroxide
- Resorcinol

iv. Retinoid Treatment [23-24]

a. Oral Retinoid Treatment
A daily oral intake of vitamin A derivative isotretinoin over a period of 4–6 months can cause long-term resolution or reduction of acne. Isotretinoin works primarily by reducing the secretion of oils from the glands, it also affects other acne-related factors as well. Isotretinoin has been shown to be very effective in treating severe acne and can either improve or clear well over 80% of patients.

b. Topical Retinoid Treatment
Topical retinoids are medications that normalize the follicle cell life cycle. This class includes tretinoin (Retin-A), adapalene (Differin), and tazarotene (Tazorac). They are administered topically and they generally have much milder side effects.

v. Other Drugs Treatment [16, 28-32]
- Adapalene topical
- Tazarotene topical
- Salicylic acid topical
- Dapsone topical
- Trimethoprim
- Nicotinamide (Vitamin B₃)
- Spironolactone

3.2 Natural Medicinal Treatment/Home Remedies
[11,13,16, 33-35]

Some of the home treatments for the removal of acne are:
- Ginger has anti-inflammatory characteristics. If you want faster relief from acne, mix a tea spoon of ground ginger with a cup of hot water and take it twice daily.
- Intake of cold sea water fish works fast to combat against the menace of acne.
- A mild scrubbing pad soaked in 3 oz of hot water along with 1 table spoon of apple cider and half a tea spoon of hydrogen peroxide is an effective acne home remedy that works fast, if applied thrice daily.
- Dab a little lemon juice on it. The astringent properties in lemon juice will suck out some of the oil and shrink the blemish.
- Make a paste with cinnamon and honey. Use approximately 1/2 tsp of cinnamon and add to the honey. Mix to make a paste. Apply to the blemish and leave on for 20 minutes (or overnight).
- Rub a slice of potato on them. Leave it on for at least 20 minutes, or all day.
- Apples and honey to the rescue. Grate a small bit of apple into a fine mush and add honey to make a sticky, fruity paste. Apply to the pimples and leave on for 20 minutes.
- Make ice cubes out of green tea. Just rub one of those things over the blemish area two or three times a day. Swelling will go down, skin will be soothed, and the green tea acts as an astringent.
- Pound an orange peel. Take a small amount of orange peel, sprinkle some water on the pith-side and pound it with a (clean) hammer or meat tenderizer for 10 seconds. Rub the peel over the pimply areas (you could even tape it there for a few minutes for extra action).
- Dab it with strong basil tea. Boil 1 cup of water and add 1 Tbsp. dried basil leaves. Steep for 10 minutes. Strain. Refrigerate until cool. Apply to the pimples (or all over your face, if it’s oily).
- Spice it up with some nutmeg and cloves. Put 2 tsp. whole cloves and 1 tsp ground nutmeg in 1/4 cup milk. Heat on low for 10 minutes. Allow the milk to cool. With a small washcloth or other fabric, apply the spicy milk to the pimply areas – or all over your face!
- Splurge on some neem (Azadiracta indica). Neem powder and neem oil are two magical pimple treatments. With the oil, just daub the oil directly onto the blemish. Or, with the powder, mix with a tiny amount of water to form a paste that you then apply to the pimply areas. Leave on all night for extra zit-busting effectiveness.
- Rosewater and lemon juice – a fragrant remedy. Take 1 tsp rosewater and 1/2 tsp lemon juice and apply to the blemishes. The rosewater will act as a calming and helps repair the skin, while the lemon juice is astringent and drying.
- Take your honey straight. At night, slather on a little honey over an extra-large pimple. Cover this with a bandaid, and sleep with the bandaid on. In the morning, remove the bandage.
- Apple Cider Vinegar. The good old standby, the liquid that does more things than Madonna (career-wise, I mean) – just dab a bit onto the affected area and let it dry. Leave it on for 20 minutes … or forever!
- A sprig of mint’ll do you. Take one leaf from a sprig of fresh mint, rub it well between your fingers until it’s bruised. Rub the “juice” from the mint leaf over the pimple. Leave on.
- Localized egg white mask. Just separate the yolk from the white, whip the white a time or two, and
dab on to spotty area. Leave on overnight and wash well in the morning.

- Aloe vera gel or leaf.
- Consumption of unhealthy “junk” foods (eg, potato chips, soft drinks) has had a negative impact on general and skin health, resulting acne flares, chocolate in moderate quantity may helps in acne vulgaris [38].
- Away from stressful lifestyle [36]

Figure 9: Herbal and home treatment of acne [33, 34]

3.3 Skin Surgery
Isolaz is the only laser treatment for acne which has been approved by the FDA (Food & Drug Administration) for the treatment of the four different types of acne, which are otherwise difficult conditions to treat. A treatment course is recommended for optimum results, this is usually 4 - 6 treatments. Acne scar surgery is one of the most commonly prescribed treatments for severe acne scarring. This is because it has a high success rate, can be done in at little as ten minutes and is suitable to treat all types of acne scars. While acne surgery does not completely remove your scars it does minimize them to a point at which they can heal themselves, disappear with further treatment of laser skin resurfacing or go unnoticed. There are four main kinds of this surgery: punch excision, punch elevation, punch grafting and subcutaneous incision [37].

Figure 10: Isolaz therapy

3.4 Phototherapy
3.4.1 Light Emitting Diode Therapy
Light exposure has long been used as a short-term treatment for acne. Recently, visible light has been successfully employed to treat mild to moderate acne (phototherapy or deep penetrating light therapy) Used twice weekly, this has been shown to reduce the number of acne lesions by about 64% and is even more effective when applied daily. LED Light Therapy is the use of specific types of light which give off energy that stimulates your cells, thereby increasing the production of collagen and elastin. In turn, this makes your skin firmer, less wrinkled, and younger looking. This is known as LED Light Therapy. LED stands for Light Emitting Diode. Diodes give off energy in the form of light. These little diodes are compact, durable, powerful,
bright, efficient, and when used in combination with Light Stim’s patent pending engineering, produce rejuvenating effects on the skin. LED blue light therapy helps to kill the acne causing bacteria, reduce inflammation and provide a general rejuvenation effect on the skin \[^{38}\].

3.4.2 Photodynamic Therapy
Photodynamic therapy (PDT) refers to the use of aminolevulinic acid (ALA), methyl-aminolevulinic acid (MAL), or other photosensitizing agents to enhance the effect of subsequent light or laser therapy. Topical ALA is taken up by epithelial cells and converted into protoporphyrin IX, accumulating both in epithelium and pilosebaceous units. Illumination after ALA treatment leads to photoactivation of protoporphyrin IX and subsequent cell damage. ALA also induces porphyrin production by \(P.\ Acnes\). \(P.\ Acnes\) cultures grown in the presence of ALA led to a 5-fold decrease in culture viability after 3 illuminations of high intensity blue light \[^{38}\].

3.4.3 Laser Treatment
The term "LASER" stands for Light Amplification by Stimulated Emission of Radiation. Lasers work by producing an intense beam of bright light that travels in one direction. This laser beam can cut, seal or vaporize skin tissue and blood vessels. The laser has the unique ability to produce one specific color (wavelength) of light which can be varied in its intensity and pulse duration. Ordinary light from non-laser sources is composed of many different colors and appears white. The wavelength and power output of a particular laser determines its medical application \[^{37}\].

The FDA has approved the use of a cosmetic laser for the treatment of acne. However, efficacy studies have used very small sample sizes for periods of six months or less, and have shown contradictory results. Also, laser treatment being relatively new, protocols remain subject to experimentation and revision, and treatment can be quite expensive. Also, some Smooth beam laser devices had to be recalled due to coolant failure, which resulted in painful burn injuries to patients.

3.5 Cosmetic Procedure \[^{32, 39}\]
3.5.3 Comedo Extraction
4. References


34. www.g2microsystems.com/acne-home-remedies-that-work-fast.htm