Acne

Acne is a chronic inflammatory skin condition. While acne is most common in adolescents, acne can affect people of all ages and all races. It usually becomes less of a problem after the age of 25 years, although about 15% of women and 5% of men continue to have acne as adults. It may also start in adult life.

How does acne present?

It presents as inflammatory and non-inflammatory lesions. These include;

- Open and closed comedones (blackheads and whiteheads)
- Papules (small red lumps)
- Pustules (yellow spots)

- Nodules (large painful red lumps)
- Pseudocysts (soft swellings)

Secondary skin changes can occur such as;

1. Picked or scratched spots
2. Red marks from recently healed spots, often seen in fair skin,
3. Pigmented or dark marks from old spots, mostly seen in people those with dark skin.

Acne can result in various types of scarring, some of which are very difficult to treat. Unfortunately, some scarring can become more apparent as patients age, as the collagen in their skin reduces.

What causes acne?

There are four pillars involved in the development of acne.

These are; plugging of the oil ducts, increased sebum production with increased androgens, inflammation, and an increase in the activity of certain bacteria.

1. Follicular plugging and hyperkeratinization. Topical retinoids, such as retrieve and adapalene, are highly effective for this issue. Systemic isotretinoin is also a very effective option and is taken orally.

2. Excessive sebum production and androgens.

There are higher levels of sex hormones (androgens) after puberty.

- Sex hormones are converted in the skin to dihydrotestosterone (DHT), which stimulates the glands in the skin to enlarge and produce sebum. The androgens also cause changes in the composition of the sebum which may contribute to acne.

The oil glands are connected to hair follicles and these may become blocked with dead skin cells causing oxidisation of the sebum and activation of bacteria leading to more inflammation. The blocked follicles look like black heads if they don't have associated inflammation.

3. Colonisation of pilosebaceous units by a bacteria C. Acnes.

Bacteria contribute to inflammatory lesions in acne but acne is **not** a skin infection.

At puberty the number of bacteria on the skin surface increases. The most common bacteria is called *Cutibacterium acnes* (C.acnes)

However, the severity of a person's acne does not depend on the number of bacteria on the skin surface or in the sebaceous ducts (the ducts taking oil to the skin surface). However, it can be affected by how active the C. acnes bacteria are. Some *C. acnes* bacteria can produce active enzymes and inflammatory mediators which can cause inflammation in the surrounding skin.

Treatment options for this include benzoyl peroxide topically and low dose oral tetracyclines**.** They also have non-antibiotic anti-inflammatory

4. **4. Consequent inflammation.**

The pathogenesis of acne is characterized by complex interactions in the immune system, where C. Acnes plays a pivotal role. It triggers the activation of the Toll-like receptors on sebocytes (fat cells) and keratinocytes(skin cells), initiating the release of pro-inflammatory messengers called cytokines.

Early acne lesions are characterized by a significant presence of immune cells called CD4+ T-helper cells. The presence of CD4(+) lymphocytes around follicles in early lesions indicates that inflammation precedes hyperkeratinization.

C. Acnes also drives the production of matrix metalloproteinases (MMPs) which include lipase, hyaluronidase, and protease activities. These enzymes potentially contributes to the breakdown of the wall of the sebaceous gland, leading to extensive inflammation, and scarring.

Additionally, the growth hormone precursor called IGF-1 is increased by androgens. It also increases inflammation within the pilosebaceous unit. This may explain why acne often starts during puberty when androgen levels start to increase.

Treatment of mild acne

Most patients with mild acne can be treated with topical treatment regimes. Most of these can be obtained over-the-counter in New Zealand without prescription.

- Wash affected areas twice daily with a mild cleaner and water or a cleanser containing benzyl peroxide such as Cerave Acne Foaming cream cleanser (4% BPO)
- Apply a small amount of Adapalene Differin gel all over affected area. It will cause dryness initially so use sparingly and with a topical moisture following each application.
- It may take several weeks or even months to see convincing improvement.

Treatment of moderately severe acne

Treatment for moderately severe acne usually includes the topical treatment plus oral medication.

Suitable oral medications include:

- Antibiotics such as tetracycline, doxycycline or trimethoprim for around 3 months
- The combined oral contraceptive pill, in females
- Oral antiandrogens, such as low-dose cyproterone (in combination with oestrogen), and/or spironolactone, particularly if there are signs of hyperandrogenism
- The topical antiandrogen, clascoterone, for acne vulgaris

- Oral isotretinoin for resistant or persistent acne.

When oral antibiotics are discontinued, control should be maintained long-term by continuing topical therapy.

Treatment of severe acne

Treatment for severe acne requires oral treatment . This is often with isotretinoin. This can be safely prescribed in our dermatology centre in the dedicated acne clinic.

The following may also be prescribed:

- High dose oral antibiotics for six months or longer
- In females, especially those with polycystic ovary syndrome, oral antiandrogens such as oestrogen/cyproterone or spironolactone may be suitable long-term
- The topical antiandrogen, clascoterone, for acne vulgaris