Cutaneous Manifestations Of Polycystic Ovarian Syndrome

Supervised by:

Ass. Lec. Yasir Abdullah

Submitted by;

Aya Hashim mohammed

Abstract :

Five to ten percent of women who are of reproductive age suffer from polycystic ovarian syndrome (PCOS), one of the most prevalent endocrine illnesses in women. Among the primary signs of PCOS are the dermatological indications of hyperandrogenism, primarily hirsutism, acne vulgaris, androgenic alopecia, and acanthosis nigricans [1].

Objective:

To study the cutaneous manifestations in patients with PCOS and to correlate these skin manifestations with hormonal changes.

Keywords: Cutaneous manifestations ,acne ,androgenic alopecia, hirsutism Polycystic ovary syndrome,acanthosis nigricans, skin tags,stria

Introduction:

i. With a prevalence of 6–10% among women in their reproductive years, polycystic ovarian syndrome (PCOS) is the most frequent endocrinopathy affecting women and the most common form of androgen excess condition. It is a multisystem metabolic illness that affects both fertility and quality of life significantly.[3.4]
The Rotterdam 2003 PCOS diagnostic criteria state that two of the three following conditions must be met in order to diagnose PCOS: [6]

ii. Anovulatory ovulation and/or anovulation (few than eight menses in a 12-month period).
iii. Laboratory or clinical hyperandrogenism.
IV. Polycystic ovaries on ultrasonography (>10 ml in ovarian volume and/or ≥12 follicles in each ovary measuring 2–9 mm in diameter).[7] One of the first signs of PCOS is cutaneous alterations, which include skin pigmentations, hirsutism, acne, virilization, and skin tags. PCOS is linked to changes in the PSU unit's growth and function, which can lead to hirsutism, acne, and female hair loss.[8]

PCOS affects women of reproductive age without regard to ethnicity; however, depending on the diagnostic criteria utilised, the signs and symptoms may vary throughout ethnic groups. The Rotterdam criteria indicate a five-fold increase in prevalence above the National Institutes of Health's (NIH) criterion. Obesity, insulin resistance (IR), and the chance of

developing Type 2 diabetes mellitus (T2DM) have all recently been linked to PCOS.Women who have disorders related to metabolism and reproduction are more likely to experience infertility and endometrial cancer, thus early detection and adequate treatment are crucial. [9.10]

Skin conditions associated with polycystic ovarian syndrome:

Acne and seborrhea:

The prevalence of acne is high, affecting 10–34% of those with the disorder. Androgens are key players in the emergence of acne. They lead to an excess of sebum—an oily substance—being produced by the skin's glands.[12] Sebum and dead skin cells accumulate inside hair follicles, trapping bacteria beneath the skin and causing acne. Inflammation and pimple production result from this.[13]
Acne can appear in a number of places on a PCOS patient, including the:

- **face**
- **neck**
- **chest**
- **upper back**

Acne lesions in women with PCOS may be bigger, deeper, and take longer to heal. In PCOS, acne typically gets worse around menstruation time.[13]
Acne caused by PCOS typically appears in regions of the body that are more hormonally sensitive. These comprise the lower

face, which includes the jawline and lower cheekbones.
It used to be widely believed that acne associated with hormone disorders, such as PCOS, was typically worse than acne that wasn't hormonal. However, more research indicates that this isn't really the case.

There are several types of acne lesions. These can be pustules, open comedones (blackheads), or closed comedones (whiteheads). Nodules, cysts, and inflammatory nodules,are amonge the severe vairites and are the most likely to cause scar

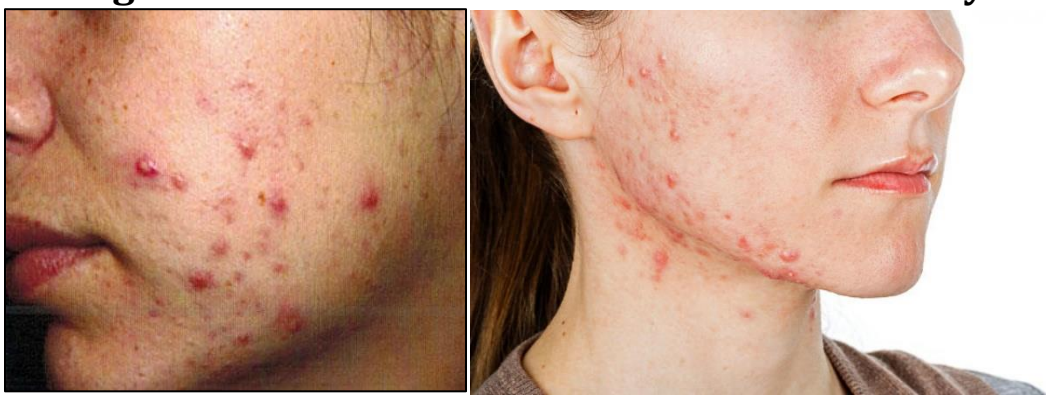


Fig.1.Acne on the jaw and

Seborrhea : Seborrhoeic dermatitis (also known as pityriasis simplex capiliti) or erythematous patches with yellow-gray scales that typically affect the upper chest, back, and face. Environmental and genetic factors combine to cause seborrhoeic dermatitis.An inflammatory response in the skin is first triggered by an external factor, such as stress or disease. The body's oil-making glands go into overdrive as a result, creating an excessive amount of Malassezia yeast.This creature resides on the surface of the skin.

A sequence of skin changes are brought on by the immune system's reaction to yeast that grows too quickly. This causes the skin patches typical of seborrhoeic dermatitis to

Other triggers include:

1-hormonal changes

2-illness

3-harsh detergents or chemicals

4-cold, dry weather

5-certain medical conditions, like Parkinson's disease, psoriasis, HIV, and acne

6-drugs,[15]

The pathophysiology of PCOS and eczema is related to elevated male hormone levels and inflammation, which could partially explain the connection. The skin's barrier is increased by oestrogen but inhibited by progesterone and androgens.[16] Given that patients with PCOS have lower oestrogen and higher testosterone levels, it makes reasonable that these individuals may also have issues with their skin barrier. A compromised skin barrier is also the cause of seborrhoeic dermatitis. Eczema and PCOS don't seem to be associated, even if there is no proof that one causes the other.

Androgenic alopcia A prevalent cause of hair loss in both men and women is androgenetic alopecia. It is sometimes referred to as female pattern hair loss in women and male pattern baldness in men.[18]

Men lose hair in a definite pattern that starts above both temples. The hairline gradually recedes to take on the distinctive "M" shape. Additionally, hair thins at the crown or vertex of the head, frequently leading to partial or total baldness. Women's hair loss patterns vary from men's patterns in this regard. When hair loss occurs in a female pattern Midway through the scalp, the thinning will begin and progressively spread in a circular manner.[19]

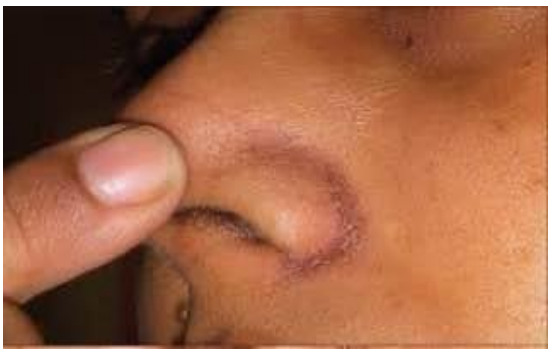


Fig.2.Seborric dermatitis on the scalpas on sides of the nose its

Starting at the hairline, the thinning will progressively go backward down the scalp's centre in a triangle pattern (often called a "Christmas tree pattern" since it is narrower at the rear and wider at the front).
Female pattern baldness usually results in shorter and thinner hair than total baldness. Nevertheless, the thinning may still be noticeable enough to reveal a sizable portion of the scalp.
In order for males and females to operate normally, their bodies need to have different amounts of androgens and oestrogens. Numerous hormonal issues might arise when the equilibrium is upset.

This may set off a disease called hyperandrogegism, in which the body overproduces androgens. The most important one is the androgen Male sex traits including facial hair, larger stature and muscular growth, and a deeper voice are all caused by testosterone in males. DHT is also the cause of hair loss in female PCOS patients. It also contributes to hair loss since testosterone is converted into the androgen known as DHA. Roughly 10% of testosterone in both males and females gets changed into DHT. DHT can attach to receptors on scalp hair follicles when it is released into the bloodstream, which will cause the hair follicles to shrink. The hair may noticeably thin even if it does not fall out.3.
Because of the high levels of DHT and testosterone that PCOS patients have in their systems, they are susceptible to androgenic alopecia[21].

Fig.3.Androgenic alopecia in fimales in temporal and crown area of the sculp

J

Hirisutism:

The most prevalent cause of hirsutism in women is polycystic ovarian syndrome (PCOS), which is described as the presence of terminal hair with a masculine distribution in women[22]. Compared to women in the general community, who have a prevalence of 4% to 11%, PCOS patients have a prevalence of 70–80% hirsutism. In PCOS, hirsutism is linked to an excess of androgen generated from the ovaries as well as individual susceptibility of the pilosebaceous unit to Certain vellus hair follicles respond to higher androgen levels throughout puberty by developing into terminal hair, which is darker, bigger, and more noticeable, turning them into sexual hair follicles. Although axillary and pubic hair are most susceptible to minute amounts of androgens, Higher androgen concentrations might be required in other regions for follicle terminalization. The primary active component of plasma testosterone, free testosterone, is what gives androgens their activity.In order to visually evaluate abundant terminal hair, the modified Ferriman-Gallwey (mFG) scoring method has been widely adopted in clinical practice. This has helped to standardise the evaluation of hirsutism and facilitate data comparison. While assessing [23]hirsutism in various populations, skin type, ethnic variances, and other characteristics should be taken into account, even though a common mFG score threshold would be helpful for comparisons. Conversely, it has been demonstrated that there is little correlation between the degree of hirsutism and testosterone levels as determined by traditional methods. In fact, the majority of women with PCOS and hirsutism also

have serum androgen levels that are greater than reference values, while others may not

.ANDROGEN-SENSITIVE SITES OF HAIR GROWTH:

COMMONER lower back, inner thigh, lower lip, beard, and breast area. LESS COMMON upper back, upper abdomen, and sternum

A clinical estimate is used to classify hirsutism as mild, moderate, or severe. Introduced in 1961, the Ferriman–Gallwey (F–G) scoring system was designed to measure hair growth and accurately quantify the degree of hirsutism.1. Nine body locations are now included in the original scheme, which was based on the presence of hair in 11 different areas.9. The range on the scale is 0 (no terminal hairs) to4 (extensive terminal hair growth) and the numbers are added[24]

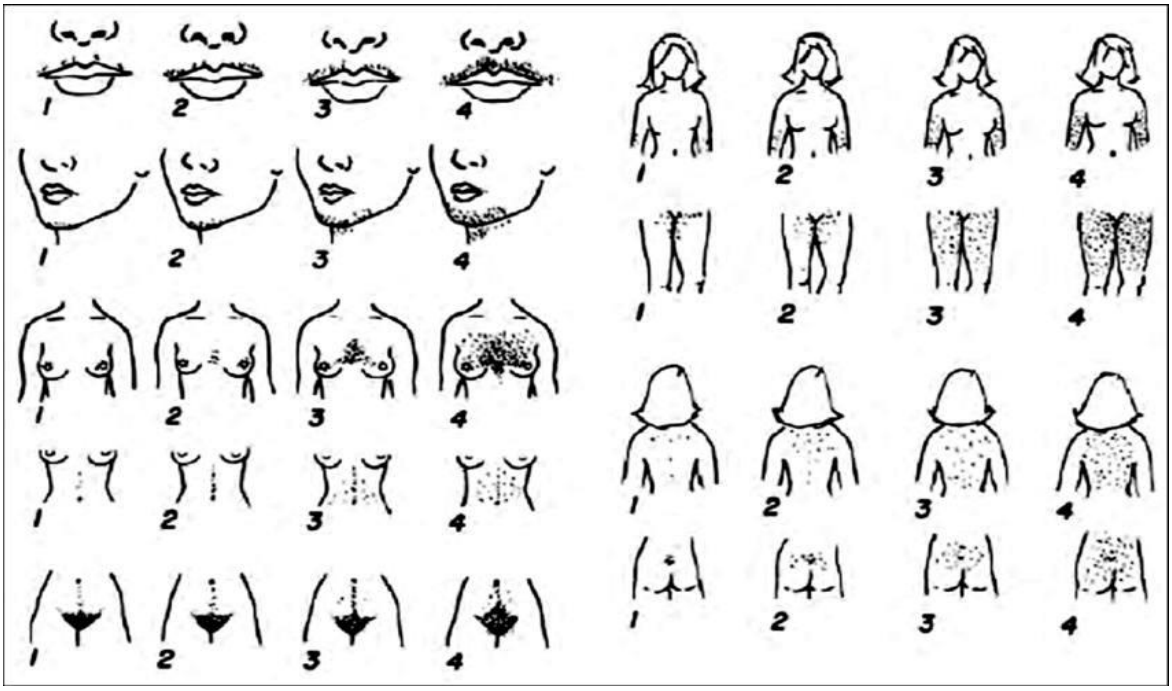


Fig.4.Ferryman gallwey scoring system

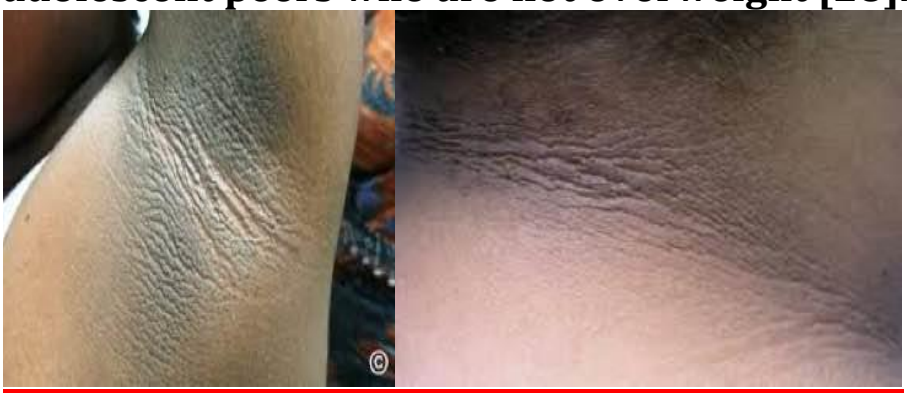
Acanthosis nigricans:

The velvety darkening of the skin known as acanthosis nigricans commonly affects intertriginous areas. The borders of this poorly defined hyperpigmentation are frequently found in skin fold locations, like the groyne, axilla, and back of the neck. In obese populations, the hyperpigmentation may also include skin thickness.Clinically speaking, Acanthosis nigricans (AN) is characterised by thickening, darkly pigmented skin that produces both dermal and epidermal in the absence of true acanthosis and melanocytosis on histologic inspection, hyperplasia with orthokeratotic hyperkeratosis, papillomatosis of the stratum spinosum, and basal layer hyperpigmentation. This is a reactive skin alteration that is closely linked to endocrinopathy, cancer (especially gastrointestinal adenocarcinoma), obesity, insulin resistance, and hyperinsulinemia. The frequency fluctuates, The basic mechanism of AN is the interaction between insulin-like growth factor 1 (IGF-1) and IGF binding protein 1 (IGFBP-1), which is linked to elevated insulin levels, albeit the pathophysiology of AN is not entirely understood. Hyperinsulinemia may cause the liver to produce more IGF-1 and impede the synthesis of IGFBP-1. The proliferation of dermal fibroblasts and epidermal keratinocytes may be triggered by an increase in IGF-1 circulating concentrations and IGF-1 receptor activation.[26]

Adolescents experience hyperinsulinemic physiological states during puberty. Even greater

adipokines, which in turn may impair glucose transport activity, resulting in IR. IGF-1 may play an important role in this relationship[27]. IGF-I binds to insulin receptors, stimulating glucose transport in the adipose and muscle tissues, inhibiting hepatic glucose output, and simultaneously suppressing insulin secretion, which may lead to a decrease in blood glucose []. However, in the presence of IR and consequent hyperinsulinemia, lower levels of IGFBP-1 may lead to disturbances in glucose homeostasis and possibly to prolonged s may explain the higher prevalence of A olescents, compared to adults and adolescent peers who are not overweight [28].

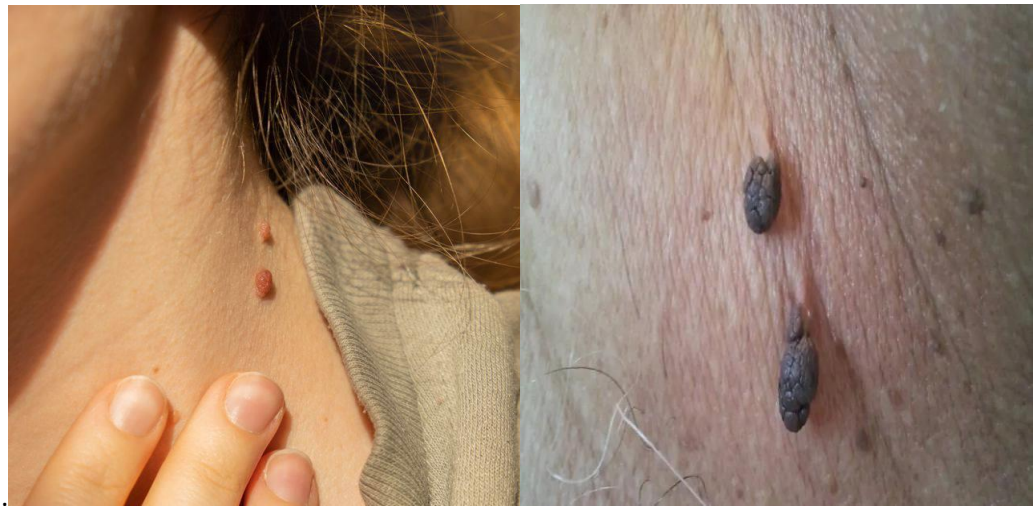
Fig.5.Acanthosis nigricans in fluctural surface



Skin tags:

Skin tags, sometimes referred to as "acrochordons," are benign skin growths that are frequently observed as soft excrescences of piled-up skin. Although it is not as often as other symptoms of polycystic ovarian syndrome, it can nevertheless happen if

insulin resistance sets in. Skin tags are usually little, pliable lumps or skin flaps. Skin tags are more common in those who are overweight, pregnant, or have loose skin; they also arise in the same location where dark spots are prevalent.[29] Additionally, having diabetes, metabolic syndrome (high blood pressure, abnormal blood sugar levels, excess fat around the waist, or abnormal cholesterol levels), or having a blood family with skin tags incrseases your risk of developing skin tags.It's crucial.

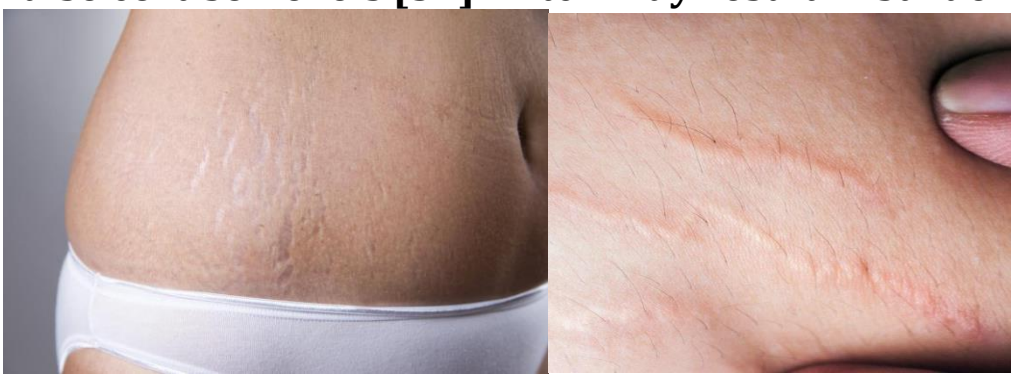


Skin tags in fluctural surface.fig.6

Striae:

The cause of striae distensae, also referred to as stretch marks, is indentation stripes that can develop on the buttocks, hips, abdomen, or other parts of the body. Skin stretching is the root cause of stretch marks. Numerous factors, including as your genetics and the level of skin stress, influence how severe they

are. There's also a chance that your cortisol levels are involved. The hormone cortisol is generated by the adrenal glands. It causes the skin's elastic fibres to deteriorate, and it has a connection to PCOS because both conditions are stressful and raise cortisol levels [31].Witch may result in striae.



Striae in different body surfices.fig.7

Conclusion:

PCOS is fairly common disorder in women of reproductive age group and is associated with various complications if left unrecognized and untreated. Cutaneous features of PCOS are first to manifest clinically and their screening in suspected cases help in early diagnosis.