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# CLINICAL ASSESSMENT OF HYPERPIGMENTATION IN THE THIRD TRIMESTER OF PREGNANCY IN RELATION TO FETAL GENDER, SKIN TYPE AND MATERNAL AGE

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**Abstract:**

**Background** Hyperpigmentation, caused by increased serum levels of MSH, oestrogen, or progesterone, is the most prevalent pregnancy symptom. Progesterone, which is a byproduct of melanin deposition into dermal and epidermal macrophages, enhances the impact of oestrogen, which in turn increases the output of melanin by the melanocytes. While hyperpigmentation can happen everywhere, it usually shows up darker in naturally darker areas, including the inner thighs, periumbilical area, areolas (sometimes called "secondary areolas"), nipples, genitalia, and axillae.

Furthermore, the skin's creases and intertrigonous spaces could become darker.

1. Hyperpigmentation's existence.
2.Level of hyperpigmentation distribution.
3.Should hyperpigmentation be associated with foetal gender?

**Methodology** :This study used a cross-sectional design.Data collection technique: convenience sample ,Attending healthcare facilities is the target population for all pregnant women in their third trimester. All items on the checklist were checked off when we conducted in-depth interviews with pregnant women.

**Results**: Pregnant women carrying male foetuses are more likely to experience hyperpigmentation of the neck, breasts, flexures (neck, axilla, inner thigh, and genitalia), linea nigra, and overall hyperpigmentation compared to pregnant women carrying female foetuses. Compared to mothers under the age of twenty, there is a strong correlation between maternal age and the development of hyperpigmentations of different types. Pregnant women with skin colour type 4 are more likely to have hyperpigmentation than those with skin colour type 1.

**Conclusion:** The study found that there is a correlation between the gender of the foetus and the development of skin hyperpigmentation in pregnant women. Specifically, women who carry male foetuses are more likely to experience skin pigmentary alterations than those who carry female foetuses. Maternal age is

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associated with an increased risk of skin hyperpigmentations, or pigmentary alterations, in women over the age of twenty. Women with darker skin tones are more likely to experience hyperpigmentation because of the correlation between skin tone and the onset of this condition.

**Keywords** Hyperpigmentation, Maternal age , skin, gender, third trimester.

## Introduction

The colour of the skin Normal skin colour is determined by several chromophores, the most important of which is melanin: Major contributors to skin colour are carotenoids, haemoglobin (oxygenated and decreased) and melanin. Melanosomes are organelles carrying melanin that are related to racial and ethnic differences in skin tone by virtue of their number, size, shape, location, and degradation. They are produced by melanocytes and delivered to the keratinocytes of the epidermis. In man, two types of melanin pigmentations [1] are known. 1. Constitutive skin colour is the genetically set quantity of melanin pigmentation which remains constant regardless of external circumstances such as sun exposure. The second is a sun-induced tan, commonly known as "facultative (inducible) skin colour." 'Facultative (inducible) skin colour'. Endocrine, aparacrine and autocrine factors are responsible for other factors causing hyperpigmentation [1, 2]. "The melanocyte" skin pigmentation group. All the pigment cells in the body weigh about 1.5 grams. Most of these cells are epidermal melanocytes [1]. Melanin production in melanocytes occurs in a three-stage process that encompasses not only melanogenesis, the synthesis of melanosomes within the cell, but also the transfer of these pigment granules to nearby epidermal keratinocytes through long branching dendrites. In 1963, Fitzpatrick and Breathnach [2, 3] identified each epidermal melanocyte as part of an "epidermal melanin unit" comprising the epidermal cells it supplies. The intricate interface (5) between melanocytes and their keratinocytes is vital for skin colour. For the skin to have sufficient pigmentation, the production of this

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organelle itself is as vital as the appropriate transfer and transport of melanosomes to keratinocytes.

The melanocyte:Melanocytes are located in the basal layer of the epidermis. The amount of melanocytes in the skin is not much different between the sexes and racial groups. Studies indicate that melanocyte density in exposed skin is about double that of non-exposed skin. Each portion of the body contains a different number of active epidermal melanin units, but each melanocyte serves the same number of keratinocytes. One melanocyte can provide melanosomes to about 36 living keratinocytes. The amount of melanocytes varies in different areas of the body [4]. Melanocytes create the pigmented polymer melanin, which is delivered to keratinocytes by dendritic processes of the melanocyte from cell organelles in the cytoplasm called melanosomes [5]. Ultraviolet light (UV) irradiation is the dominant route for the release of signals from keratinocytes. The highest densities of melanocytes are seen in the genitalia and the face, with densities ranging from roughly 550 to over 1,200 per square millimetre [5]. All races have the same amount of melanocytes; hence, skin pigmentation is more dependent on the activity of melanogenesis, the percentage of fully developed melanosomes and their transport and dispersion in keratinocytes than the number of melanocytes [5]. Darker pigmented skin contains more melanosomes in stage IV, but lighter pigmented skin often contains more melanosomes in stages II and III. Other factors include the rate of disintegration of individual melanosomes once they have been delivered to the surrounding keratinocytes, which is to some extent size-dependent. The spinosum middle layer degrades the smaller melanosomes of light-pigmented skin, which occur in clusters of 2 to 10 within keratinocyte secondary lysosomes. In heavily pigmented skin the melanosomes are larger, more equally distributed in the lysosomes of the keratinocytes and degraded more slowly, leaving melanin granules in the stratum corneum [6].

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**Melanin biosynthesis:**

Biosynthesis of Melanin Melanosomes participate in the synthesis of two separate types of melanin, eumelanin and pheomelanin. Pheomelanin is light in colour and soluble; it contains sulphur and is reddish yellow in colour. Eumelanin is dark brown to black in colour and is insoluble. Melanins are indole derivatives of 3,4 di-hydroxy phenylalanine (DOPA) that are formed in melanosomes by a sequence of oxidative processes [7]. The melanosomal pH affects the polymerisation of melanin and the activity of melanogenic enzymes. The rate-limiting catalytic step in the synthesis of both types of melanin in the Raper-Mason route is the oxidation of the amino acid tyrosine to DOPA by the enzyme tyrosinase. This reaction is also known as 'monophenol', 'DOPA oxidase', 'tyrosine oxidase' or just 'DOPA'. The critical rate-limiting step in melanogenesis is thought to be the conversion of tyrosine to DOPA since melanin synthesis is prevented when this is blocked. In both processes, DOPA is a co-factor and a tyrosinase substrate. The exact mechanism of tyrosinase binding to its substrates is not known, but in vitro kinetic studies have demonstrated that tyrosinase binds to tyrosine and DOPA at different sites and that binding of DOPA induces a conformational change in tyrosinase that results in an increased affinity for both substrates. DOPA is oxidised from melanogenicon to form dopaquinone. Two more conversions of DOPAquinone are possible to dopachrome. The first is 5,6-dihydroxyindole (DHI). The second is 5,6-dihydroxyindole-2-carboxylic acid (DHICA). The second reaction is catalysed by the enzyme DOPAchrome tautomerase, also known as TRP-2. This DHI / DHICA ratio seems to correspond with the ratio of brown vs black eumelanin as well. A lower DHI / DHICA ratio produces brown eumelanin, while a higher DHI / DHICA ratio produces black eumelanin. Pheomelanin is a soluble molecule with low molecular weight. It is yellow or red in hue. DOPAquinone may also react with glutathione or cysteine to form cysteinylDOPA [5].

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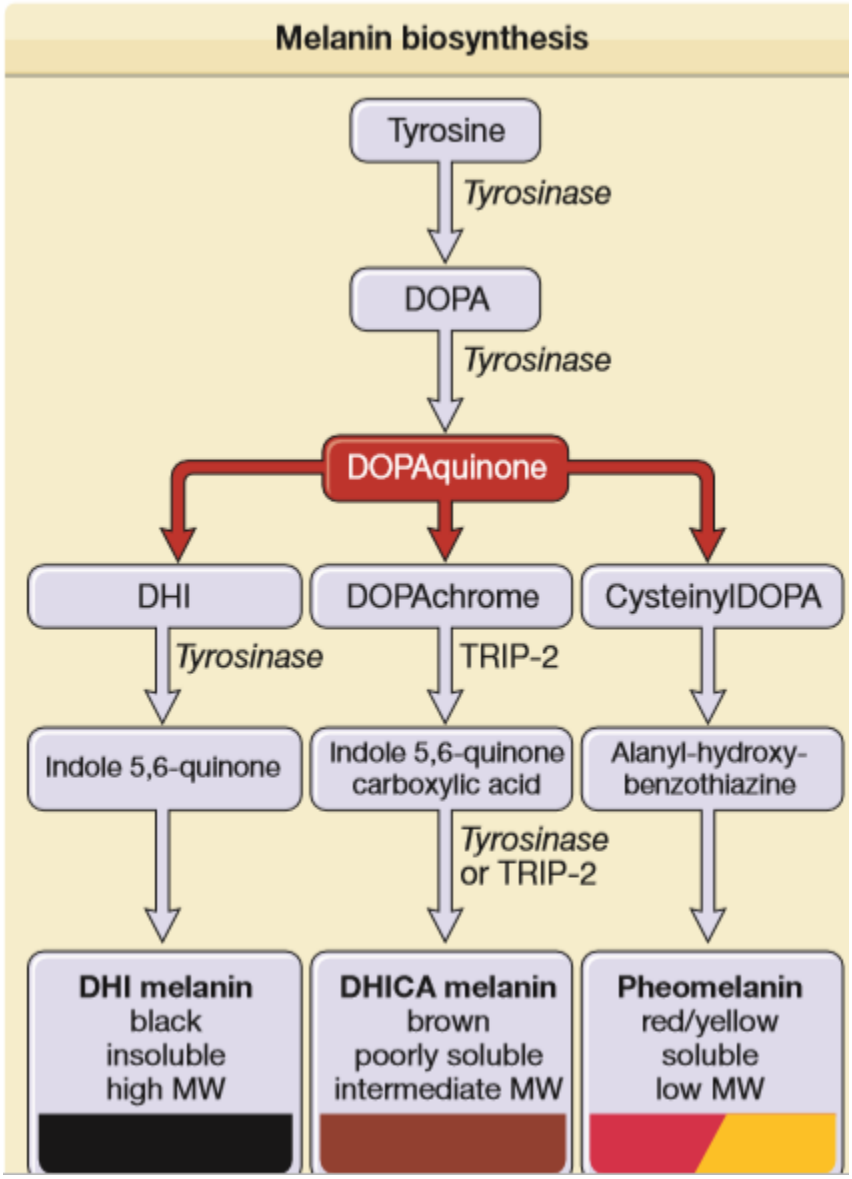


Figure 1.1 Melanin biosynthesis [5].

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**Regulation of melanocyte function:**

In skin, melanocyte behaviour is mostly governed by autocrine signalling, environmental cues such as UV irradiation and signals from neighbouring keratinocytes. It is known that ultraviolet radiation can directly stimulate melanocyte dendricity and melanin production [8], as well as most chemicals synthesised and secreted by keratinocytes. The process of melanogenesis takes place in the specialised organelles of melanocytes, the melanosomes, which can be stimulated by several paracrine cytokines, namely α-melanocyte-stimulating hormone (α-MSH), stem cell factor (SCF), endothelin-1 (ET-1), nitric oxide (NO), adrenocorticotropic hormone (ACTH), prostaglandins, thymidine dinucleotide and histamine. Tyrosinase (TYR), microphthalmia-associated transcription factor (MITF), tyrosine-related protein-1 (TRP-1) and tyrosine-related protein-2 (TRP-2) are pigment-related proteins that are activated and produced by numerous stimuli that cause melanogenesis through distinct signalling pathways [9]. Cytokines such as IL1α or TNFα are released by keratinocytes and can trigger fibroblasts to secrete chemicals that encourage melanocytes such as HGF or SCF [10]. Eicosanoids such as prostaglandin D2 (PGD2), prostaglandin E2 (PGE2), leukotriene C4 (LTC4) and leukotriene B4 (LTB4) modulate the synthesis of elanocyte pigment. It has been proven that LTB4 may directly stimulate single human melanocytes to produce pigment [11]. Transcription factors and signalling pathways involved in the initiation and regulation of melanin synthesis include MITF, the tyrosine kinase receptor KIT and its ligand SCF. Genetic, biochemical and pharmacological studies point to MC1R signalling as the major determinant controlling melanogenesis. The MC1R is one of the five class A G protein-coupled receptors comprising the MC1R subfamily. The MC1R agonists α-MSH and ACTH stimulate eumelanin production, while ASP mimics synthesis of pheomelanin. The MC1R is another mechanism by which α-MSH influences the ratios of pheomelanin and eumelanin [12]. α-MSH is one of several peptides derived from proteolytic cleavage of the

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large pro-opiomelanocortin (POMC) precursor protein. α-MSH is mostly produced in the pars intermedia of the pituitary gland. Under humans, the pars intermedia of the pituitary gland is poorly developed, and little α-MSH is secreted except under clinical conditions. The skin is one of the extra-pituitary sites of production of α-MSH and other melanocortin peptides. The epidermis contains keratinocytes that are a rich source of these peptides. There is some evidence that melanocortin peptides such as a-MSH mediate the pigmentary effects of UVR. Melanocytes and keratinocytes release melanocortin peptides in response to ultraviolet light. Therefore, α-MSH might serve as an autocrine and paracrine factor to regulate the melanocytes and the skin pigmentation. [13].

**Melanocyte regulation by endocrine factors:**

The hormonal variables favouring pigmentation are oestrogen, α-MSH, ACTH and endorphin. The last three are derived from the cleavage of proopiomelanocortin/POMC. It is oestrogen that causes pigmentation during pregnancy. The influence of oestrogens on skin pigmentation has been recognised for over 60 years. The increased pigmentation of the face, areola, lower central abdomen and genitalia in pregnancy, and the immune response of the mother altering considerably to allow for it, are thought to be due to the elevated levels of oestrogens. Melanocytes express oestrogen receptors, and increased amounts of oestradiol stimulate enzymes such as tyrosinase, tyrosinase-related protein 1 and tyrosinase-related protein 2 [15].

**Pigmentary changes:**

Abnormal migration of melanocytes from the neural crest to skin during development may cause pigmentary diseases. Furthermore, pigmentation disorders can be caused by the lack of transfer of melanosomes to the adjacent keratinocytes, alteration in the synthesis of melanin, or a defect in the breakdown or removal of melanin. Loss of melanocytes from the immune system or a harmful

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substance might cause pigmentation disorders. Pigmentation problems can be inherited or acquired and are classified as hypo or hyperpigmentation. They may be either confined or diffuse in appearance. [16]. An acquired pigmentary illness might appear as either lightening or darkening of the skin. Lesions in melanin synthesis, such as leukoderma and hypopigmentation, induce a decrease in the amount of melanin in the epidermis, making the skin look lighter. Another reason for skin lightening, such as in vitiligo, is the lack or death of melanocytes. Pigmentation of the skin may be due to an excess of melanocytes, as in epidermal melanocytosis or lentigines, or to an increased amount of melanin produced by a typically sized population of melanocytes, as in epidermal melanosis or freckles. Pigmentary incontinence and dermal melanosis, however, are disorders in which the melanin in the skin is irregularly distributed, resulting in a darker complexion. Acquired hypermelanoses such as lentigo senilis and UV B melanosis are basically involved in the up-regulation of the melanogenic paracrine cytokine network [17]. Diffuse hyperpigmentation can be due to systemic diseases such as haemochromatosis, Addison's disease or hyperthyroidism. It could be a side effect of the medicine you are taking [18].

**Pregnancy and physiological abnormalities**

Pregnant women undergo complicated and obvious changes to their skin, some of which may last over 90% of the time. These changes are mainly due to the complicated hormonal, immunologic, metabolic, and circulatory changes of pregnancy and may affect the skin in numerous ways.

Some of these physiological changes are caused by the endocrinological changes [19]. In the endocrinology of human pregnancy, the immune response of the mother alters considerably to allow for the metabolic and endocrine changes involved in response to physiological adjustments at the placental-foetal interface. Progesterone and oestrogen are two of many essential hormones [20]. During pregnancy, the immune response of the mother alters considerably to

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allow for the attachment of the foetus. The cytokine profile is altered towards the Th2 cytokines that serve to maintain the foetus (IL-4, IL-5, IL-10, and IL-13). Th1 cytokines (IL-2, TNF-α, and IFN-γ) are increased in the postpartum period. This process is facilitated by hormonal changes during pregnancy, in addition to the general trend of the Th2 cytokine profile. Progesterone induces Th2 cytokines such as IL-4, IL-5 and IL-10, while oestrogen inhibits IL-2 production. Glucocorticoid levels remain elevated during pregnancy, and progesterone suppresses TNF-α release. It induces the synthesis of IL-10, IL-4 and IL-13 and inhibits the production of IL-1, IL-2, TNF-α and IFN-γ [21].

**Changes in pigment:**

Hyperpigmentation, a common sign of pregnancy, is most commonly due to increased serum levels of MSH , oestrogen or progesterone. Melanin synthesis is raised by oestrogen in the melanocytes, and the oestrogenic effect is enhanced by progesterone, which is created by melanin deposition into dermal and epidermal macrophages [19]. It's not understood exactly how. But it is considered that skin hyperpigmentation is produced by a combination of hormonal variables, genetic predisposition and UV exposure. Melanocytes may be more reactive to larger quantities of oestrogen, progesterone, Z-endorphins, α- and Z-melanocyte-stimulating hormone and perhaps other chemicals. These hormones are assumed to promote melanin production and cause the hyperpigmentation observed clinically. One of the probable mechanisms by which human placental lipids promote melanin production is through the up-regulation of tyrosinase. Generalised hyperpigmentation occurs anywhere, but the most common sites are the areolas (also called 'secondary areolas'), nipples, genitalia, axillae, periumbilical area and inner thighs. Discolouration can also develop in intertriginous areas and skin folds. Patients may develop new acanthosis nigricans lesions, or existing lesions may worsen. The linea alba, a line down the midline of the lower abdomen and suprapubic area, darkens into the linea nigra.

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Darkening is most typically detected in the area of the umbilicus and the pubic symphysis [22].

**Melasma:**

Melasma (chloasma or the 'mask of pregnancy') is a kind of facial hyperpigmentation that affects 45-75% of pregnant women and one-third of women on oral contraceptive pills. It is symmetrical, blotchy, strongly margined and tan to dark brown. It might be malar, mandibular or centrofacial in distribution. Although the malar pattern is the most common, it can also affect the entire central face. The majority of individuals include the entire central face, even though the malar pattern is the most common. in the majority of individuals. It covers the chin, nose, top lip, cheeks and forehead. Melasma is histologically determined by the location of the melanin, which might be epidermal or dermal. The cause is likely a combination of sun exposure, genetics, cosmetics and a rise in melanocyte-stimulating hormone, oestrogen and progesterone. In as many as 90% of individuals, melasma from pregnancy disappears within one year after partum [23]. Histologically, melasma is characterised by increased pigmentation in the epidermis. There has been no increase in the number of melanocytes. The metabolic activity of the melanocytes is raised, as demonstrated by their hypertrophy and an increase in the number of dendrites and of cytoplasmic organelles. There is more melanin in the epidermis and more mature melanosomes. There is moderate mononuclear infiltration, mast cells, increased vascularity and elastosis in the dermis. There is no difference in skin pigmentation between melasma-affected skin and adjacent normal skin [24].

**CHANGES TO THE NAILS**

Longitudinal melanonychia striata (pigmented bands running the length of the nail plate) are more common in Black people.

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Table 3. List of physiologic and pathologic causes (many more illnesses are included). Hyperthyroidism, Psoriasis, Amyloidosis Trauma, pregnancy, and Cushing's syndrome [25].

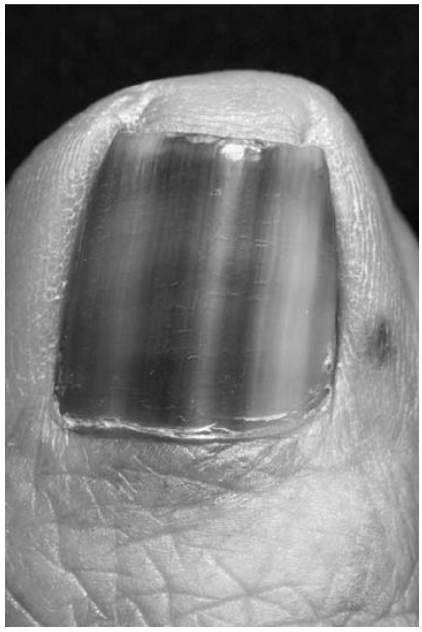

Figure 1.4 longtudina melanonychia [23].

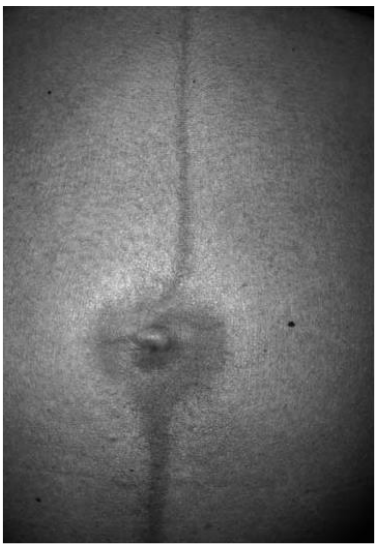

Figure1.2 linea nigra [23].

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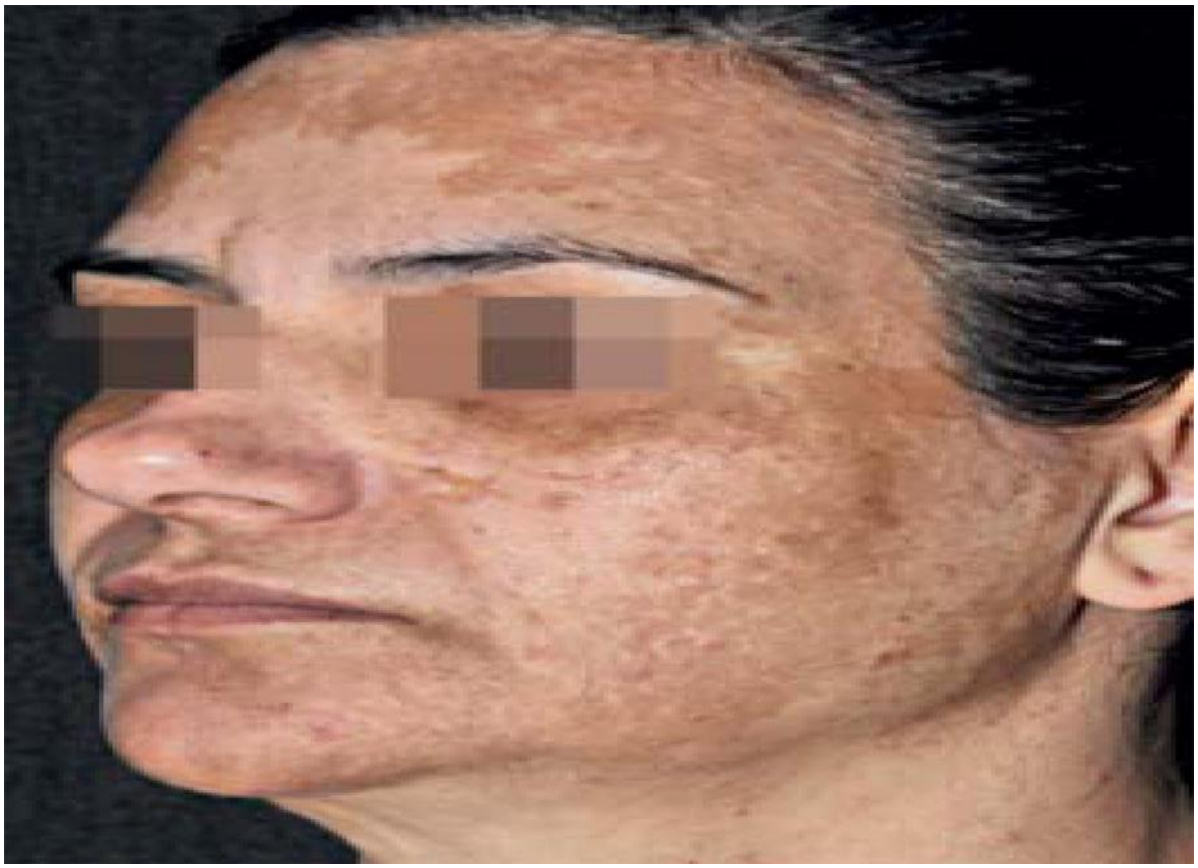

Figure 1.3 melasma [24] female patient with frontal, temporal, parotid, mandibular, zygomatic, mentonian lesions and lesion in the upper lip

## Patient and Methods

**Methodology**:

This cross-sectional study included 309 pregnant ladies in their third trimester attending Al_alweya hospital for gynaecology and Obstetrics, Fatimat_Al_zahraa Hospital for gynaecology and Obstetrics, Al_mustanserya primary health care centre (PHCC) or Bab al_muatham primary health care centre (PHCC), seeking antenatal care services and treatment during the period from October 2018 to June 2019, during which direct interviews were made with pregnant ladies and

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apretested check list was Determine the sex of the foetus by sonographic examination. Thorough evaluation of physiological skin pigmentation detection. Inclusion criteria: Women of any age who are first-time mothers, have a single foetus, and are classified as Fitzpatrick skin types 3 or 4 [26]. Exclusion criteria: Pregnant women who have a previous history of long-term health conditions or chronic skin disorders.

Long-term health conditions

A chronic skin disorder.

| *Skin type* | *Skin/hair/eye color* | *Characteristics* |
|---|---|---|
| I | White; very fair; red or blond hair; blue eyes; freckles | Always burns, never tans |
| II | White; fair; red or blond hair; blue, hazel, or green eyes | Usually burns, tans with difficulty |
| III | Cream white; fair with any eye or hair color; very common | Sometimes mild burn, gradually tans |
| IV | Brown; typical Mediterranean/Caucasian skin | Rarely burns, tans easily |
| V | Dark brown; Middle-Eastern skin | Very rarely burns, tans very easily |
| VI | Black | Never burns, tans very easily |

Figure 2.1Fitzpatrick skin type classification[26].

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**Ethical Consideration:**

1. The Study idea will be approved by the scientific and ethical committee in Al Kindy Medical College.
2. The institutions involved in gynaecology and obstetrics (Al-Alweya Hospital, Fatimat Al-Zahraa Hospital, Al-Mustanserya Primary Health Care Centre and Bab al-Muatham Primary Health Care Centre) would be consulted prior to the start of the study. Verbal consent of pregnant women will be taken before starting the interviews, outlining to them the objectives of the study and assuring them of the confidentiality of their data.

**Statistical analysis:**

The data will be collected and statistically analysed using the SPSS program version 24 and Excel 2016. In descriptive research, the data were presented in tables and graphs. The significance of the relationship between the variables was tested using the Q-squared test. The discrimination cut-off will be a P value <0.5.

Check list of pregnant lady in thirdtrimester:

ID:

Age:

skin color:

Presence of pigmentation(yes or no):

melasma:

Linea nigra:

Hyperpigmentation of breasts and flexers:

Generalised pigmentation:

Nail pigmentation:

Fetus gender:

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**Results:**

The study showed that about 47.9% of pregnant ladies aged 20 years or younger while 52.1% of those aged older than 20 years, 66% with skin type 3 while 34% with skin type 4.

Ultra sound examination proved that 57.6% of pregnant ladies with male fetuses while 42.4 of pregnant ladies with female fetuses as shown in table 3.1 and figure 3.1.

Table 3.1 distribution of studied patients according to essential characteristics

| | | N | % |
|---|---|---|---|
| Age | =<20years | 148 | 47.9% |
| | >20years | 161 | 52.1% |
| Skin colour | 3 | 204 | 66.0 |
| | 4 | 105 | 34.0 |
| Fetus gender | Male | 178 | 57.6 |
| | Female | 131 | 42.4 |

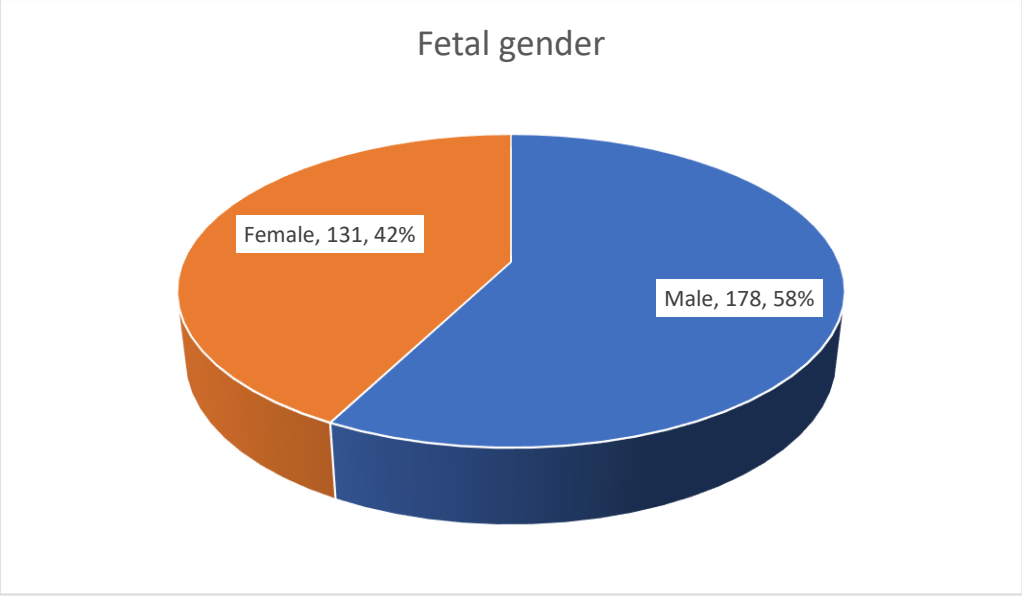


Figure 3.1

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Table 3.2 showed that melasma was present in about 25.3% of pregnant ladies with male fetus in comparison to 21.4% pregnant ladies with female fetus, no significant association between fetus gender and presence of melasma ,P value 0.426.

Table 3.2 association between fetus gender and Melasma

Table 3.3 showed that about 96.6% of pregnant ladies with male fetus got linea nigra in comparison to 90.6% of pregnant ladies with female fetus, significant association between fetus gender and presence of linea nigra ,P value 0.018.

Table 3.3 association between fetus gender and Linea Nigra

| | | Present | | Absent | | P value |
|---|---|---|---|---|---|---|
| | | N | % | N | % | |
| Fetus Gender | Male | 172 | 96.6% | 6 | 3.4% | 0.018 |
| | Female | 118 | 90.1% | 13 | 9.9% | |

Table 3. 4 showed about 85.4%of pregnant ladies with male fetus got hyperpigmentation of breasts and flexures in comparison to 34.4% of pregnant ladies with female fetus , significant association between fetus gender and presence of hyperpigmentation of breasts and flexures, P value 0.001

| | | Present | | Absent | | P value |
|---|---|---|---|---|---|---|
| | | N | % | N | % | |
| Fetus Gender | Male | 45 | 25.3% | 133 | 74.7% | 0.426 |
| | Female | 28 | 21.4% | 103 | 78.6% | |

Table 3.4 association between fetus gender and hyperpigmentation of breasts and flexures

| | | Present | | Absent | | P value |
|---|---|---|---|---|---|---|
| | | N | % | N | % | |
| Fetus Gender | Male | 152 | 85.4% | 26 | 14.6% | 0.001 |
| | Female | 45 | 34.4% | 86 | 65.6% | |

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Table 3.5 showed that about 42.1% of pregnant ladies with male fetus developed generalized pigmentation in comparison to 11.5% of pregnant ladies with female fetus, significant association between fetus gender and presence of generalized pigmentations, P value 0.001.

Table 3.5 association between fetus gender and Generalised Pigmentations

| | | Present | | Absent | | P value |
|---|---|---|---|---|---|---|
| | | N | % | N | % | |
| Fetus Gender | Male | 75 | 42.1% | 103 | 57.9% | 0.001 |
| | Female | 15 | 11.5% | 116 | 88.5% | |

Table 3.6 show that 7.9% of pregnant ladies with male fetus developed nail pigmentations in comparison to 3.1% of ladies pregnant with female fetus, no significant association between fetus and presence of nail pigmentations, P value 0.074.

Table 3.6 association between fetus gender and Nail Pigmentations

| | | Present | | Absent | | P value |
|---|---|---|---|---|---|---|
| | | N | % | N | % | |
| Fetus Gender | Male | 14 | 7.9% | 164 | 92.1% | 0.074 |
| | Female | 4 | 3.1% | 127 | 96.9% | |

Table 3.7 show about 13.5% of pregnant ladies aged 20 years old and younger got melasma in comparison to 32.9% of pregnant ladies aged older than 20 years old, significant association between age of ladies and presence of melasma, P value 0.001.

And about 16.7% of pregnant ladies with skin colour type3 developed melasma in comparison to 37.1% of those ladies with skin colour type 4, significant

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association between developement of melasma in pregnancy and skin colour type, P value 0.001.

Table3.7 association between factors other than fetus gender and Melasma

| | | Present | | Absent | | P value |
|---|---|---|---|---|---|---|
| | | N | % | N | % | |
| Age | =<20years | 20 | 13.5% | 128 | 86.5% | 0.001 |
| | >20years | 53 | 32.9% | 108 | 67.1% | |
| Skin colour | 3 | 34 | 16.7% | 170 | 83.3% | 0.001 |
| | 4 | 39 | 37.1% | 66 | 62.9% | |

Table (3.8) show about 89.9% of pregnant ladies aged 20 years and younger got linea nigra in comparison to 97.5% of those ladies aged older than 20 years, significant association between age of pregnant ladies and presence of linea nigra, Pvalue 0.005.

Also show about 92.2% of pregnant ladies with skin colour type3 got linea nigra in comparison to 97.1% of those ladies with skin colour type 4, no significant association between skin colour type and presence of linea nigra, P value 0.084.

Table(3.8) association between factors other than fetus gender and Linea Nigra

| | | Present | | Absent | | P value |
|---|---|---|---|---|---|---|
| | | N | % | N | % | |
| Age | =<20years | 133 | 89.9% | 15 | 10.1% | 0.005 |
| | >20years | 157 | 97.5% | 4 | 2.5% | |
| | 3 | 188 | 92.2% | 16 | 7.8% | 0.084 |
| | | | | | | |
| | | | | | | |
| Skin colour | 4 | 102 | 97.1% | 3 | 2.9% | |

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Table (3.9) show about 56.1% of pregnant ladies aged 20 years and younger got hyperpigmentations of breasts and flexures in comparison to 70.8% of those ladies aged older than 20 years, significant association was found between age of mother and presence of hyperpigmentation of breasts and flexures , P value 0.007. And about 54.4% of pregnant ladies with skin colour type 3 developed hyperpigmentation of breasts and flexures in comparison to 81.9% of those ladies with skin colour type 4, significant association between skin colour type and presence of hyperpigmentation of breasts and flexures in pregnancy, P value 0.001.

Table (3.10) show that about 20.3% of pregnant ladies aged 20 years and younger

Table 3. 9 association between factors other than fetus gender and hyperpigmentation of breast and flexures

| | | Present | | Absent | | P value |
|---|---|---|---|---|---|---|
| | | N | % | N | % | |
| Age | =<20years | 83 | 56.1% | 65 | 43.9% | 0.007 |
| | >20years | 114 | 70.8% | 47 | 29.2% | |
| Skin colour | 3 | 111 | 54.4% | 93 | 45.6% | 0.001 |
| | 4 | 86 | 81.9% | 19 | 18.1% | |

develope generalized pigmentations in comparison to 37.3% of those aged older than 20 years, significant association between age of mother and presence of generalized pigmentations , P value 0.001.

And about 19.1% of pregnant ladies with skin colour type 3 got with generalized pigmentation in comparison to 48.6% of ladies with skin colour type 4, significant association was noticed between skin colour type and development of generalized hyperpigmentation pigmentation, P value 0.001.

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Table 3.10 association between factors other than fetus gender and Generalised Pigmentation

| | | Present | | Absent | | P value |
|---|---|---|---|---|---|---|
| | | N | % | N | % | |
| Age | =<20years | 30 | 20.3% | 118 | 79.7% | 0.001 |
| | >20years | 60 | 37.3% | 101 | 62.7% | |
| Skin colour | 3 | 39 | 19.1% | 165 | 80.9% | 0.001 |
| | 4 | 51 | 48.6% | 54 | 51.4% | |

Table (3.11) show that about 2.7% of pregnant ladies aged 20 years old and younger got with nail pigmentation in comparison to 8.7% of those ladies aged older than 20 years, significant association between age of mother and development of nail pigmentation during pregnancy, P value 0.025.

And show about 2.0% of pregnant ladies with skin colour type 3 got nail pigmentation in comparison to about 13.3% of those ladies with skin colour type 4, significant association between skin colour and development of nail pigmentation, P value 0.001.

Table 3.11 association between factors other than fetus gender and Nail Pigmentations

| | | Present | | Absent | | P value |
|---|---|---|---|---|---|---|
| | | N | % | N | % | |
| Age | =<20years | 4 | 2.7% | 144 | 97.3% | 0.025 |
| | >20years | 14 | 8.7% | 147 | 91.3% | |
| Skin colour | 3 | 4 | 2.0% | 200 | 98.0% | 0.001 |
| | 4 | 14 | 13.3% | 91 | 86.7% | |

**Discussion:**

Changes in the skin may be due to several physiological processes, including endocrinological, immunological, metabolic and vascular changes. Hyperpigmentation is the most visible transformation of the skin. Features that

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are prevalent include melasma (often referred to as the 'mask of pregnancy'), darkening of the nipples and the skin around the nipples (areolas), flexures (neck, axilla, inner thigh, and genitalia), linea alba, and generalised hyperpigmentation. Figure 3.1 reveals that 58% of the pregnant women in this study were carrying a male foetus and 42% were carrying a female foetus. This difference may reflect societal norms that expect moms to seek medical care more often for sons than for daughters.

This study shows that skin pigmentation of various types {melasma, hyperpigmentation of breasts and flexures and generalised hyperpigmentation} is more common in mothers older than twenty. These findings can be explained by the fact that IL-1α secretion increases as cells grow older, and the increased secretion of IL-1α by aged keratinocytes may stimulate HGF production in dermal fibroblasts paracrinely and ET-1 production in keratinocytes autocrinely, which stimulates melanocyte proliferation and induces an increase in TYR tyrosinase activity in melanocytes. IL-1α is a major mediator responding to inflammation and injury, which may be constantly increased in ageing skin because of the genes involved in skin inflammation. Melasma is more likely to develop in women, especially during the reproductive years, with the highest incidence between 20 and 30 years [27]. The increased secretion of interleukin 1 beta by aged keratinocytes in the skin may explain the increased pigmentation of the skin and other signs of skin ageing [27]. This may suggest that our skin pigmentation is associated with maternal age, as it is infrequently documented before puberty [28].

Just as there is a trend for women with darker skin and hair to have more visible hyperpigmentation during pregnancy, the current study indicated hyperpigmentation of different types was more common in skin types 4 and 5 compared with types 3.

Pregnancy is known to modify the maternal immune environment; however, whether there are differing changes in maternal immunology depending on foetal

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sex is not understood. When a woman is pregnant with a male foetus, her hormones and immune system alter to include more proinflammatory and proangiogenic growth factors. When a woman is pregnant with a female foetus, her immune system and hormones change to include a greater production of regulatory cytokines. We hypothesised that pregnant women carrying male foetuses would have an inflammatory response due to the differences in growth factors and cytokines in their bodies, which may explain the variable pregnancy outcomes depending on foetal sex. Indeed, women carrying male foetuses exhibited greater levels of inflammatory cytokines at numerous periods throughout gestation.

Our results indicate that moms of male foetuses are more likely to develop skin pigmentary alterations. This may reflect the association of these proteins with elevated concentrations of IL-12p70, IL-21, IL-33 and G-CSF in maternal plasma during pregnancy, which start to rise above female levels as early as 6 weeks post-conception [30].

## Conclusion

The present study relates foetal gender to the development of skin hyperpigmentation in pregnant women. Pregnant women carrying male foetuses are, in particular, more likely to develop skin pigmentary changes than those carrying female foetuses.

Pigmentary changes are more common in women beyond twenty years than in younger women. There is an association between maternal age and the development of hyperpigmented skin.

The type of skin that a person has is related to the possibility of acquiring hyperpigmentations, with women with darker skin tones being more likely to have modifications of this type.

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**Possible Limitations:**

There has been no research on this subject before.
time required
A word of warning:
Further general studies.

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