"STUDY OF SKIN CHANGES IN PREGNANT WOMEN IN THE SECOND AND THIRD TRIMESTER,COMING TO A TERTIARY CARE HOSPITAL IN BELAGAVI-A ONE YEAR CROSS SECTIONAL STUDY"

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Dissertation

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In

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Under the guidance of
Dr. B. SIDDARAMAPPA MD,DVD
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LIST OF ABBREVATIONS USED

hCG : Human chorionic gonadotrophin

FSH : Follicle stimulating hormone

LH : Leutinising hormone

hPL : Human placental lactogen

hCS : Human chorionic somatomammotropin

GnRH :Gonadotropin releasing hormone

ACTH :Adrenocorticotropic hormone

MSH: Melanocytic stimulating hormone

PTH : Parathyroid hormone

SLE : Systemic lupus erythematosus

BMI : Body mass index

UDCA :Ursodeoxycholic acid

BMZ: Basement membrane

PG : Pemphigoid gestationis

PEP: Polymorphic eruption of pregnancy

ICP: Intrahepatic cholestasis of pregnancy

AEP: Atopic eruption of pregnancy

PUPPP: Pruritic urticarial papules and plaques of pregnancy

ABSTRACT

Background

Pregnancy is associated with multiple skin changes, most of which are physiological in nature. Pregnancy however is also associated with certain pruritic eruptions and specific dermatoses of pregnancy which not only cause distress to the pregnant female, but may influence the fetal outcome as well.

Methods

A total of 104 randomly presenting pregnant women in their second and third trimester coming with skin changes in the outpatient and inpatient care in at KLE's Dr Prabhakar Kore Hospital and MRC, Belagavi, during the period January 2016 to December 2016 were included. Ethical clearance was obtained from the Jawaharlal Nehru Medical College Institutional Ethics Committee of Human Subjects Research. Each pregnant woman underwent complete general, systemic and dermatological examinations and the findings were recorded in the proforma. Bedside laboratory investigations like Tzanck smear, KOH mount and Wood's lamp examination were done only if essential to confirm the diagnosis. The statistical analysis was performed by STAT 11.2 (college station TX USA) and IBM SPSS software.

Results

A total of 104 pregnant women were enrolled in the study. 88(85%) pregnant women were below the age of thirty and 16(15%) were thirty years and above. 55(53%) were primigravidae and 49(47%) were multigravidae. 62(60%) women were in their second trimester and 42(40%) women were in their third trimester. Itchy lesions were

the most common complaint 45(43%). 94(90%) women had physiological skin changes of pregnancy and 28(27%) women had specific dermatoses of pregnancy. Linea nigra 59(57%) was the most common physiological skin change. Pruritic urticarial papules and plaques of pregnancy 9(9%) and prurigo of pregnancy 9(9%) were the two most common specific dermatoses of pregnancy.

Conclusion

This study brings into focus various skin changes during the second and third trimester of pregnancy.

Key words

Physiologic changes, Pregnancy dermatoses, Specific dermatoses.

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INTRODUCTION

Pregnancy is characterized by various physiological skin changes as striae gravidarum, melasma, hair, nail and vascular changes which are due to hormonal effects.

The pre-existing skin conditions in pregnancy may either improve or exacerbate due to many immunological changes.

During gestation endocrinological,immunological, metabolic and vascular changes occur that makesthe pregnant women susceptible to changes of skin and appendages. Whether physiologic or pathologic, these changes affect virtually every organ of pregnant women including the skin. Some of these changes are due to de novo production of a variety of protein and steroid hormones by the fetoplacental unit as well as the increased activity of maternal pituitary, thyroid and adrenal glands.[1]

Cutaneous changes and eruptions during pregnancy are exceedingly common and in some cases a cause for substantial anxiety on the part of the prospective mother. Some of these are benign and reversible after delivery whereas others can have potential effects on the fetus in terms of morbidity and mortality.[1]

The concerns of the patient may range from cosmetic appearance,to the chance of recurrence of the particular problem during a subsequent pregnancy,to its potential effects on the fetus.[2]

However,pregnancy modifies the course of a number of pre-existing dermatological conditions.[3]

A new classification of these specific dermatoses of pregnancy has recently been proposedbased on the results of a retrospective study on more than 500 pregnant patients,[4] that includes pemphigoid gestationis, polymorphic eruption of pregnancy, intrahepatic cholestasis of pregnancy and atopic eruption of pregnancy.

The pruritus associated with polymorphic and atopic eruption of pregnancy is troublesome only for the mother.But pemphigoid gestationis may be associated with small-for-date babies, and intrahepatic cholestasis of pregnancy poses an increased risk for fetal distress, prematurity, and stillbirth.[5,6]

Spangler et al.[7]proposed that papular dermatitis of pregnancy was associated with a 30% fetal mortality.

Holmes and Black[8]classified the specific dermatoses of pregnancy in four groups pemphigoid(herpes) gestationis(PG),prurigo of pregnancy,pruritic folliculitis of pregnancy(PF) and polymorphic eruption of pregnancy(PEP).

The recent classification of pregnancy specific dermatoses has been proposed by Ambros –Rudolph et al.[4] in 2006,which includes pemphigoid gestationis, intrahepatic cholestasis of pregnancy, atopic eruption of pregnancy and polymorphic eruption of pregnancy.

The present study has been undertaken to know the frequency and pattern of skin changes in pregnant women.

AIMS AND OBJECTIVES

The study has been designed and conducted with the following aims and objectives.

PRIMARY OBJECTIVE: To study the spectrum,frequency and pattern of physiological skin changes in the second and third trimester of pregnancy.

SECONDARY OBJECTIVE: To observe and study the specific dermatoses of pregnancy and the skin diseases affected by pregnancy in the second and third trimester.

REVIEW OF LITERATURE

During pregnancy alteration in the appearance of the skin is common. There are many changes related metabolic and endocrine causes, although most are physiological they can also produce may pathological changes which range from mild cutaneous changes to pathological which are specific to pregnancy.

As cell mediated immunity is depressed during normal pregnancy, this is responsible for the increased severity and frequency of skin infections such as candidiasis. There are a few inflammatory skin dermatoses which are specific to pregnancy and are seen only in pregnancy. Most of these skin dermatoses in pregnancy resolve during postpartum period and are benign in nature, however a few can risk fetal life and require antenatal surveillance.

Therefore, the main concern and worry of the parents is the impact on the fetus.

Many pre-existing dermatological diseases and tumours show alteration during pregnancy.

ENDOCRINE CHANGES

At conception, a new endocrine gland is established and endocrine changes begin to manifest as soon as implantation occurs.

Maternal endocrine adaptations to pregnancy involve the hypothalamus, pituitary, parathyroid, thyroid, adrenal glands, and ovary, and are linked to the interactions of the fetal-placental-maternal unit.

Production of hormones by placenta is the peptide hormone, under which comes the Human chorionic gonadotrophin (hCG) which is produced by the syncytiotrophoblast cells,it is made up of of alpha and beta subunits, the alpha subunit is the same as the alpha subunit of leutinising hormone, FSH and thyroid stimulating hormone.It maintains corpus luteal oestrogen and progesterone production in first trimester and it maintains pregnancy until the placenta takes over.The hCG level peaks at ten to twelve weeks of pregnancy then declines to term.It can be detected in blood as early as six days after conception and in urine as early as fourteen days after conception.[9]

Human placental lactogen (hPL) also known as human chorionic somatomamotropin (hCS) it is produced by syncytiotrophoblast cell,it is similar to the human growth hormone,hPL, growth hormone and prolactin may come from a common progenitor hormone. HPL level rises throughout the pregnancy and peaks near term.[9]

STEROID HORMONE (OESTROGEN AND PROGESTERONE)

Placenta produces enough oestrogen and progesterone from maternal and foetal precursors to take over the function of corpus luteum after the sixth week of pregnancy.[9]

The other hormones produced by placenta are gonadotropin releasing hormone (GnRH) and inhibins, prolactin, endorphin and enkephalin.[10]

THE CHANGES IN PITUITARY HORMONES

There is an increase in prolactin,ACTH (adrenocorticotrophin) and MSH (melanocyte-stimulating hormone) and a decrease in growth hormone and gonadotrophin .

Other changes are increase in the adrenal hormones that include cortisol,aldosterone and renin and angiotensin.

In the thyroid hormones both T3 and T4 synthesis increase but however thyroid binding globulin also increases and the free plasma level of T3 and T4 remain unchanged.In the parathyroid hormone,PTH increase due to increased utilisation of free Ca2+.

Prostaglandin A increases 300% during first trimester due to systemic vasodilation and prostaglanding E only increases during third trimester.[10]

The corpus luteum fails to regress when fertilisation occurs and it enlarges due to hCG. The enlarged corpus luteum of pregnancy secretes oestrogen, progesterone, and relaxin. Corpus luteum starts to decline after eight weeks of pregnancy, but persists throughout pregnancy.[10]

Chorionic gonadotrophin, is the first hormone to be secreted which causes activation of the the corpus luteum to secrete estrogen and progesterone.The increase in levels of estrogen and progesterone leads to follicle stimulating hormone production suppression by the pituitary gland. In the fetal adrenal glands, further conversion of progesterone takes place leading to production of dehydroepiandrosterone. By eleventh week, the placenta produces more of estriol and progesterone.

Physiological changes observed during pregnancy including breast enlargement are believed to be due to the placental hormones.[11,12]

SKIN CHANGES ATTRIBUTABLE TO PREGNANCY

The various skin changes that are observed in pregnancy are directly or indirectly under the influence of the hormones secreted by the maternal-fetal-placental unit.

PIGMENTATION

Generalized and localized hyperpigmentation occurs in around 90% of pregnant women and is more common in darkly pigmented skin.[13]The pigmentary response which is regarded as endocrine in origin presents in already pigmented areas such as the linea alba, axilla, navel, vulva, and face.[14] Hyperpigmentation occurs in areas that are already pigmented, particularly the nipples, areolae (secondary areolae), and genital areas. The midline of the abdominal wall (linea alba) becomesdarker (linea nigra).

In some patients, an increased facial pigmentation that involves the cheeks, forehead, and bridge of the nose, presents a characteristic picture that has been called the “mask of pregnancy” or chloasma or melasma gravidarum.Melasma is the most common and visible pigmentary cutaneous change during pregnancy.

Three clinical patterns are centro-facial (64%), malar (27%) and mandibular (9%). From its appearance under Wood light (340–400nm), melasma is classified into epidermal (70%), dermal (10–15%) and mixed (20%). There is also a small group of melasma patients (2–3%) of dark skin types that cannot be classified using Wood’s light examination only.[15,16]

Genetic and hormonal influences in combination with ultraviolet (UV) radiation are the most important causes.

During pregnancy elevated levels of estrogen, progesterone and MSH especially in the third trimester, have often been found in association with melasma.[17,18] Estrogens and progestins may induce hyperpigmentary responses by stimulating of melanogenesis within melanocytes.[19]

Tyrosinase activity increased and cellular proliferation was reduced following treatment with b-estradiol of melanocytes in culture.[20]Sex steroids increase transcripts of genes for enzymes of melanogenesis in normal human melanocytes, especially those for Dopachrome tautomerase (DCT) and tyrosinase.[21]

These results agree with the significant increases in melanin synthesis and tyrosinase activity reported in normal human melanocytes under similar conditions in culture.[22]

Tyrosinase has been shown to use estradiol as a substrate and to hydroxylate it to a cathecol like compound.[23]which may then regulate its enzymatic activity .[22]

Since melanocytes contain both cytosolic and nuclear estrogen receptors [24]these pigmentary cells in patients with melasma may be inherently more sensitive to the stimulatory effects of estrogens and possibly to other sex steroid hormones. The presence of a second estrogen receptor (ERb) in human skin that appears to have different cell-specific roles from the classic estrogen receptor (ERa) indicates a potential for enhanced diversity in the mechanism of estrogen action.

After delivery, the pigmentation gradually fades. However, it is persistent in approximately 30% of patients and can be induced by estrogen-containing oral contraceptives (OCs). Chloasma also may to recur with subsequent pregnancies and appears earlier in gestational period with the subsequent pregnancies.

The patients who suffer from chloasma must be counseled regarding the recurrence in the setting of OCs and should be advised to use proper sunscreen because sun exposure increases hyperpigmentation.

Chloasma is more prevalent in patients with Fitzpatrick skin types IV-VI (moderate-brown Mediterranean skin tone to black skin tone).

In India Melasma (chloasma) is very common It is characterized by irregular, sharply marginated areas of hyperpigmentation distributed in a symmetrical pattern either on the central part of the face or on the forehead and temples, or both. After parturition it is believed to fade.Ingestion of contraceptive pills and exposure to sunlight are known to aggrevate the condition.[1,25,26]

HAIR AND NAIL CHANGES

Change in the thickness and hair growth and are quite common in pregnancy. Hirsutism is seen in many pregnant women and is more noticeable in women with dark and/or abundant body hair. It is due to increased ovarian production of androgens during pregnancy. Short lanugo hair present gives the skin a “furry” appearance, however, with the development of telogen (shedding) effluvium it disappears in the postpartum period.

Hyperandrogenic states in pregnancy are almost always the result of a condition that arises during pregnancy. The onset of virilization symptoms is often

very fast. The mother is protected against hyperandrogenism by a high level of sex hormone binding globulin (SHBG), by placental aromatase and a high level of progesterone. The fetus is protected from the mother's hyperandrogenism partly by the placental aromatase, that transforms the androgens into estrogens, and partly by SHGB.[27]

The clinically hyperandrogenism during pregnancy is not different from hyperandrogenism during nonpregnancy. This most frequently leads to an occurrence of hirsutism in androgen-dependent areas of predilection, such as the upper lip, chin, linea alba, groin, thigh and chest. There is often acne on the face, shoulders, back and chest. With some women there occurs hair loss with ensuing hair thinning in the temporoparietal area. During pregnancy, we relatively frequently see the rapid onset of masculine symptoms such as clitoromegaly and deepening voice caused by a larger larynx.[27]

During pregnancy, extensive changes take place in the mother's organism that enables the successful development of the fetus. The high level of estrogens causes an increase in protein synthesis in liver and with it an increase in specific binding proteins such as sex hormone-binding globulins (SHGB), whose level rises from the beginning of the pregnancy.

In pregnancy the synthesis of dehydroepiandrosterone (DHEA) in the adrenal glands increases significantly. DHEAS its sulphate is the main precursor of the synthesis of steroid hormones in the placenta, which is the reason the plasmatic level of both of these androgens drops by almost half during pregnancy.[27] Due to the increased level of SHBG from the start of pregnancy there occurs a change in the ratio of the free and bound testosterone. The level of the free testosterone is due to the

higher binding to SHBG up to the twentieth week of pregnancy lower than during non-pregnancy. However, it rises and peaks in the third trimester.[27,28,29]

Feedback set for levels of free testosterone leads to increased production of testosterone so that the total testosterone at the end of the first trimester reaches levels common with men.

The etiology of hyperandrogenic states can be divided into the four groups, the most frequent cause is a non-tumor ovarian condition like pregnancy luteoma, hyperreactio luteinalis and poly cystic ovarian syndrome. In contrast, adrenal tumor causes are extremely rare. A mother's hyperandrogenism caused by fetal etiology is created with a deficiency of placental aromatase. The cause of hyperandrogenism which isiatrogenic in nature, during pregnancy occurs with the use of preparations containing androgens,progestins and danazol.[30]

Treatment consists of patient reassurance and cosmetic removal.

Pregnancy associated hirsutism usually resolves gradually after delivery and may rarely disappear only to recur in subsequent pregnancies.[31]

During pregnancy thinning of the scalp hair is common.[32]

Telogen effluvium occurs one to five months after delivery and varies in its severity. Hair regrowth in a year is the usual course. Mild frontoparietal recession may be observed in pregnant women.[33]

Nail changes normally begin in the first trimester,but can also present in the second and third trimester with brittleness or softening and faster growth which is

common.The other nail changes reported during pregnancy are distal onycholysis,transverse and longitudinal grooving and softening.[34]

MUCOSAL CHANGES

Oral mucosa, is a specialized type of tissue that lines the mouth, it protects the body from infections and debris. The causes of oral mucosal changes, during pregnancy, are not well documented. The majority of changes observed are the gingivitis and pregnancy tumor .[35]

Pregnancy gingivitis is one of the most prevalent diseases of oral cavity during the pregnancy, which is observed in about 50%–98% of pregnant women. It starts around the second or third month and increases in the eight month and then declines with the reduction in synthesis of these hormones.[36]

Pregnancy tumours are one of the oral mucosal lesions, which are seen in 5% of pregnant women. Due to increased activity of estrogen and progesterone, these lesions are more common during pregnancy.[36]

High level of estrogen causes immune suppression by the inflammatory cell function inhibition. As a result, the body reaction to bacterial plaques changes leads to the increase in clinical presentation of the lesions.[37]

The most common mucosal change during pregnancy is a burning sensation. This could be due to systemic diseases like gestational diabetes, anemia or Candidiasis. The other cause of burning sensation can be attributed to iron deficiency anemia or folic acid deficiency during pregnancy.

High estrogen levels are responsible for the vascular changes noted on mucosal surfaces. Gingival hyperemia and edema, gingivitis and pyogenic granuloma are the most common alterations of the oral mucosa during gestation.[38]

Physiologic changes of the vulvovaginal area are mainly of vascular nature, and include among others varicose veins. [38]

The oral and vulvovaginal mucosal surfaces can be affected by diseases that can worsen or develop in pregnancy. Oral lesions are encountered in a large spectrum of diseases including aphthosis, pemphigus vulgaris, systemic lupus, and Behçet disease.[38]

During pregnancy the Infections of the vulvovaginal region which are caused by candida species, human papilloma virus, trichomononas vaginalis and herpes simplex virus have been associated with fetal risks.[38]

ECCRINE, APOCRINE, AND SEBACEOUS GLAND ACTIVITY

Alterations in glandular function during pregnancy are often reported. These alterations include changes in the function of the eccrine glands, apocrine glands, and sebaceous glands.

Eccrine sweating activity increases progressively near the end of pregnancy.[39,40] The alterations in autonomic nervous system function, producing symptoms and signs of increased vasomotor activity, may result in excess sweating [41,42]

Soft-tissue fibromas can occur on the face, neck, upper chest, and beneath the breasts during late pregnancy. These fibromas mostly disappear during postpartum.[43]

These physiologic adaptations to pregnancy often result in hyperhidrosis, increased frequency of miliaria, and an increased incidence of dyshidrotic eczema.[41]

Apocrine gland activity remarkably decreases during pregnancy, temporarily relieving the preexisting apocrine gland disorders such as Fox–Fordyce disease and hidradenitis suppurativa .[39,41]

Hidradenitis suppurativa is a chronic relapsing inflammatory skin disease, which characteristically begins after puberty, and is more prevalent in women than in men. This disease affects skin sites with apocrine glands, in particular the axillae and anogenital regions. It is characterized by recurrent abscess formation and draining sinus due to subcutaneous extension with induration, destruction of skin appendages, and ensuing scarring.[44]

Fox–Fordyce disease is a chronic disorder of the apocrine glands of the axillae, anogenital and periareolar regions in women.[44]

Hormonal influences probably play a causative role, but there is still debate as to the importance of estrogen, progesterone, and cortisol.[40,45]

The effect of pregnancy on sebaceous glands is controversial.[46] Some investigators suggest that sebaceous gland activity generally increases in the second half of pregnancy, with an increased rate of sebum excretion. The peak of symptoms is reached during the third trimester, when complaints of oily skin, especially on the face, are common.[40,41]Others believe there is no consistent change, with a variable effect on acne.[40,41]

VASCULAR CHANGES

Vascular changes are common during pregnancy, and occur to a variable extent in women. The changes include distension, instability, and proliferation of vessels.Spider angiomas is reddish elevations on the skin, particularly common on the face, neck, upper chest, and arm with radicles branching out from a vascular body. The condition is often designated as nevus, angioma, or telangiectasia. Palmar erythema is noticed in two-third of white women and one-third of black women.[47] The main clinical results of these abnormalities are spider angiomata (nevi aranei) and palmar erythema.[48] Other commonly seen abnormalities are flushing of skin and temporary edema of the face, hands, and feet.[49]Most rare are small hemangiomas of subcutaneous cavernous type which disappear shortly after delivery.[49]Some of the vascular changes are used as diagnostic features of pregnancy. Erythema of the vestibule and vagina, called the Jacquemier–Chadwick sign, results from distension of their vasculature, and occurs early in gestation.[47,1]The bluish discoloration of the cervix, known as the Goodell sign, is also a result of increased vascularity, this time of the cervix.[7]

Pregnancy is associated with vasodilation of the systemic vasculature and the maternal kidneys. The systemic vasodilation of pregnancy occurs as early as at five weeks and therefore precedes full placentation and the complete development of the uteroplacental circulation.[50]The remarkable vascular change is simply general vascular increase throughout the dermis.

Venous congestion and varicosities occur in almost half of all the pregnancies and can be particularly troublesome in the perineal and perianal area. They also occur

with leg and ankle edema and are attenuated with leg elevation, elastic stockings and modified positioning

On the head and neck in about 5% of pregnant women small hemangiomas may occur.

Rarely, pulmonary embolism can occur due to thrombosis of the deep veind.[51]

Early morning non-pitting oedema of the face, hands and feet may occur which gradually subsidesas the day passes. [51]

STRIAE GRAVIDARUM (STRETCH MARKS)

Striae gravidarum or also commonly known as stretch marks of pregnancy occur in most pregnancies during the second or third trimester and have a familial and racial predisposition.[52]

Fifty percent to 80% of the pregnant women experience at least a few striae and they are severe in about 10% of cases, especially in teenagers. Risk factors responsible for more severe striae include maternal family history, young maternal age, non-white race, higher baseline and delivery body mass index (BMI), and increased abdominal and hip girth. [53]

No evidence exists for the efficacy of anecdotal treatments, but elective laser cosmetic surgery is an option post-partum. Data in the literature are conflicting regarding the effects of tretinoin cream on the appearance of striae, but use of a topical retinoid during pregnancy is contraindicated.[54,55,56]

SKIN DISEASES MODIFIED BY PREGNANCY

DISEASES POTENTIALLY IMPROVED DURING PREGNANCY

Dermatological conditions like hidradenitis suppurative, fox–fordyce disease andallergic contact dermatitis,[57]improve during gestation due to the decreases in the function of the apocrine gland.

During pregnancy,[1]psoriasis is more likely to improve than worsen, 40% to 63% of pregnant women with psoriasis improve during pregnancy, whereas only 14% worsen.[58] This may be due to the high levels of interleukin-10 in pregnancy.[59] Psoriatic arthritis has been reported to develop or worsen during pregnancy and 30% to 45% of women had onset of psoriatic arthritis either postpartum or during the perimenopausal period.[60]

DISEASES POTENTIALLY WORSENED DURING PREGNANCY

Infections like Candida vaginitis is more frequent during pregnancy, up to 50% of neonates born to infected mothers are positive for candida. Infants may present with oral thrush and diaper rash. [61]

Condyloma acuminate can attain large size during pregnancy. Human papilloma virus may produce laryngeal papilloma in infant.[61]

Herpes Simplex infection can be transmitted during pregnancy and delivery.[61]

Leprosy will experience an exacerbation of the disease during pregnancy or within the first 6 months of lactation. The type-1 lepra reaction increases in frequency during the first trimester of pregnancy and then declines until delivery, whereupon it

again increases sharply. The type-2 reaction increases in frequency with peaks in the first and third trimesters and the first 9 months of lactation.[61]

Trichomoniasis is detected in 60% of pregnancy.[1]

Varicella zoster virus can also infect pregnant women. Varicella pneumonia in about 14% of affected individual, 3% of them have a fatal outcome. Congenital varicella syndrome can occasionally occur if primary maternal infection occurs during the first trimester.[61]

Untreated syphilis in pregnancy leads to adverse outcomes among more than half of the women with active disease, including early fetal loss, stillbirth, prematurity, low birth weight, neonatal and infant death, and congenital disease among newborn babies.[62] Clinical manifestations of acquired syphilis are not apparently altered by pregnancy.

Immune-mediated diseases like systemic lupus erythematosus (SLE) in pregnancy is well tolerated by mothers in remission for at least three months before conception, except those with nephropathy or cardiomyopathy. If conception the occurs during the active stage of SLE, approximately 50% of patients will worsen during pregnancy and a few will die or experience permanent renal damage.[63] Patients whose SLE first appears during pregnancy have a high frequency of severe manifestations.[1] Neonatal lupus may occur in the baby of a mother with lupus.

Dermatomyositis and polymyositis in some patients may experience deterioration.[60]

Pemphigus may be exacerbated during or soon after pregnancy, but generally to a mild degree. Although the rate of stillbirth was not as high as previously reported,

the rate of abortion was considerable. Pregnancy may have an uneventful course, especially in patients in clinical remission; However, careful monitoring of the high-risk mother and fetus is essential.[64]

Impetigo herpetiformis occurs during last trimester of pregnancy without any prior history of psoriasis. This shows that irregular erythematous patches with superficial pustules arranged in groups begin in intertriginous areas slowly involve the whole body with sparing of face, hand, and feet.[1]

Metabolic diseases like acrodermatitis enteropathica flares during pregnancy [58] as the serum zinc levels decline early in gestation. In some patients, the disease is recognized in pregnancy, but it may be misdiagnosed as impetigo herpetiformis or herpes gestationis, unless serum zinc is measured.

Women with porphyria cutanea tarda may experience chemical and biochemical deterioration.[59]

Connective tissue disorders like Ehlers-Danlos syndrome Type 1 and 4 may experience excessive bleeding, wound gaping and uterine laceration. Pseudoxanthoma elasticum can lead major gastrointestinal bleeding during pregnancy.[61]

Many dermatoses are aggrevated during pregnancy. The cell mediated immunity is suppressed during normal pregnancy [57]and this makes a gravid woman more susceptible to to infections and some become more severe.

Candidal vaginitis, trichomoniasis,genital warts, pityrosporum folliculitis, herpes simplex virus infection, varicella–zoster virus infections are the commonly encountered in pregnant women.[61]

Systemic lupus erythematosus is associated with a better outcome in pregnancy.

Systemic sclerosis in most women is not affected by pregnancy, however a few women may experience improvement in their condition.The course of dermatomyositis and polymyositis, are not affected by pregnancy but some may experience deterioration.[61]

In Ehlers–Danlos syndrome excessive bleeding, wound dehiscence, and uterine laceration are major problem in pregnancy. [61]

During pregnancy neurofibromas may appear for the first time or become larger. Rupture of major blood vessels and hypertension has been reported in pregnant women with neurofibromatosis.[61]

The course of mycosis fungoides and eosinophilic granuloma are adversely affected by pregnancy.

Pregnancy can trigger erythema multiforme and severe cases of Steven-Johnsons syndrome may cause vaginal stenosis.

Erythrokeratoderma variabilis worsens during pregnancy.[61]

In pregnancy the appearance of symptoms in HIV-infected women is hastened. Pneumocystis pneumonia in pregnant women with AIDS is fatal.Kaposi sarcoma is known to occur in AIDS-affected pregnant women.[65,66,67]

PRURITUS DURING PREGNANCY

It is a very common symptom during pregnancy, found in 3%–14% of pregnant women.[68,69]

Many dermatological conditions can be the cause of pruritus during pregnancy, like pediculosis, atopic dermatitis, urticaria, drug eruptions, neurodermatitis and scabies. Other causes of pruritus during pregnancy are diabetes mellitus, iron deficiency,chronic renal failure and parasitic infestations.[69]

The dermatoses known to be associated with the gravid state, are herpes gestationis, pruritic urticarial papules and plaques of pregnancy, papular dermatitis of pregnancy, prurigo gestationis, and impetigo herpetiformis[70,71,72]

PRURITUS GRAVIDARUM

First described in 1907 this condition is described as a syndrome of jaundice in late pregnancy associated with intractable pruritus that persisted until delivery and recurred in the following pregnancies.[73]

In Indian studies, the prevalence has reported to be 0.8%–1.1%.[74]

Clinically,it begins mostly in the last trimester though it may occur between eight and forty weeks of gestation, pruritus is the first symptom and persists throughout the course of the disorder.[35] It is episodic at onset and often localized to areas such as the abdomen, trunk, and extremities.The skin becomes excoriated and secondary infection ensue when the pruritus lasts for more than two weeks.[72]

Jaundice usually appears for one to two weeks (up to thirty weeks) after theonset of pruritus, the average time being ten days.[75]Such women complain of right quadrant fullness or tenderness as well as dark urine and light-colored stools.

The liver function tests must be carried out in all pregnant women with generalized itching with no clinically obvious cause.In a study done in Indian population serum alkaline phosphatase and serum bilirubin levels were found to be the best indicators of the disease.[76]

TABLE: 1A CHANGES IN SERUM LIVER FUNCTION TESTS DURING NORMAL PREGNANCY [77]

TESTS	FIRST TRIMESTER	SECOND TRIMESTER	THIRD TRIMESTER
Albumin	<	<	<
Alanine aminotrnaferase(ALT)	=	=	=
Aspartate aminotransferase(AST)	=	=	=
Total bilirubin	<	<	<
Alkaline phosphatase	=	= or >	>

=: no or slight changes, > : increase, < : decrease compared to non-pregnant women.

Normal albumin level is 3.5-5.2gm/dl,normal ALT level is 1-41 U/L,normal AST level is 1-40 U/L, normal total bilirubin level is 0-0.90mg/dl and normal alkaline phosphatase level is 40-129U/L.[77]

For the treatment, the drugs used are ursodeoxycholic acid (UDCA), epomediol and silymarin. The recommended dose of UDCA is between from 150 to 300 mg twice a day after meals. However currently, this drug is recommended for use in the third trimester only.[78] The mechanism of UDCA is unknown,one putative action of UDCA is that the drug inserts a key translocator or transporter protein, improving bile salt export from the liver and theoretically reducing the risk to the fetus.[79] The itching normally subsides six to twenty one days after delivery.

THE SPECIFIC DERMATOSES OF PREGNANCY

These include atopic eruption of pregnancy, polymorphic eruption of pregnancy, pemphigoid gestationis and intrahepatic cholestasis of pregnancy. Most of the specific dermatoses of pregnancy resolve postpartum and require only symptomatic treatment.

However,antepartum surveillance is recommended for patients with pemphigoid gestationis and intrahepatic cholestasis of pregnancy as they carry high fetal risk.

CLASSIFICATION

The very first classification of dermatoses of pregnancy was proposed by Holmes and Black[80]in 1983 and included four skin conditions namely pemphigoid gestationis (PG, syn. herpes gestationis), polymorphic eruption of pregnancy (PEP) (syn., pruritic urticarial papules and plaques of pregnancy [PUPPP]), prurigo of pregnancy (PP) and pruritic folliculitis of pregnancy (PF).

The second was later proposed by Shornick [81] in 1998, and included intrahepatic cholestasis of pregnancy (ICP) in addition to PEP, PP and PG.

The most recent and rationalized classification has been proposed by Ambros-Rudolph et al[4] in 2006 after their retrospective two-center study on 505 pregnant patients.

They introduced a new entity known as "Atopic eruption of pregnancy (AEP)". They also included three conditions pruritic folliculitis of pregnancy, eczema in pregnancy and prurigo of pregnancy under AEP.

Therefore, they presented four main conditions namely atopic eruption of Pregnancy, polymorphic eruption of pregnancy, pemphigoid gestationis and intrahepatic cholestasis of pregnancy under dermatoses of pregnancy.While AEP starts significantly earlier, PEP, ICP and PG present later in pregnancy.

TABLE 1 B: CLASSIFICATION OF THE DERMATOSES OF PREGNANCY[4]

CLASSIFICATION	SYNONYMS
Pemphigoid gestationis	Herpes gestationis Gestational pemphigoid
Polymorphic eruption of pregnancy(PEP)	Pruritic urticarial papules and plaques of pregnancy(PUPPP) Toxic erythema of pregnancy Late-onset prurigo of pregnancu Toxemic rash of pregnancy
Intrahepatic cholestasis of pregnancy(ICP)	Cholestasis of pregnancy Obstretic cholestasis Jaundice of pregnancy Pruritus/prurigo gravidarum
Atopic eruption of pregnancy(AEP)	Prurigo of pregnancy Prurigo gestationis (Besnier) Early -onset prurigo of pregnancy(Nurse) Papular dermatitis of pregnancy(Spangler) Pruritic folliculitis of pregnancy Linear IgM disease of pregnancy Eczema in pregnancy

PEMPHIGOID GESTATIONIS

Pemphigoid gestationis is also known as herpes gestationis it is a rare autoimmune bullous disorder that presents either soon after the postpartum period or during late pregnancy. Its incidence is 1 in 50,000 to 60,000 pregnancies and the disease is known to show a correlation with the haplotypes HLA-DR3 and DR4.[82]

The pathogenesis is mainly due to the circulating complement-fixing IgG antibodies of the subclass IgG1 (formerly known as “herpes gestationis factor”) that bind to a 180-kDa protein, BP-180 or bullous pemphigoid antigen 2, in the hemidesmosomes of the basal membrane zone (BMZ), which causes tissue damage and blister formation .[83]

Clinically PG presents with intense pruritus that may be occasionally preceded by the appearance of skin lesions.

Initially, erythematous urticarial papules and plaques develop typically on the abdomen, but may later spread to the entire skin surface.

The umbilical region is the region that is always involved. In the “pre-bullous” stage, differentiation between PG and polymorphic eruption of pregnancy is very difficult.

The diagnosis becomes clear when the lesions progress to form tense blisters that resemble those in bullous pemphigoid.The facial and mucous membranes are normally spared.[5]

Diagnosis can be done by a skin biopsy during thepre-bullous stage and it is characterized by edema of the upper dermis and the middle dermis with perivascular

inflammatory infiltrate, of lymphocytes, histiocytes, and a few eosinophils, during the bullous stage the skin biopsy reveals mainly subepidermal blistering that is ultrastructurally located to the lamina lucida of the BMZ.[84]

The gold standard in the diagnosis of PG is direct immunofluorescence of perilesional skin, which shows linear deposition of C3 along the BMZ along with IgG deposition .[84,85]

In indirect immunofluorescence in 30 to 100% of cases, circulating IgG antibodies in the patient's serum may be detected by bindingto the roof of the artificial cleft on salt-split skin. Using modern ELISA antibody levels can also be monitored.[84,85]

The natural course of PG is characterized by exacerbations and remissions during pregnancy, with frequent improvement in late pregnancy followed by a flare-up at the time of delivery in 75% of patients.[86]

The fetal prognosis is normally good; there is an increase in small-for-date babies but not in prematurity or stillbirths.

Due to the passive transfer of antibodies from the mother to the fetus around 10% of newborns develop mild skin lesions that resolve spontaneously within days to weeks .[85,86]

Treatment mainly depends on the stage and severity of the disease and the main aim is to control pruritus and to prevent any blister formation.

In cases of mild pre-blistering, topical corticosteroids with or without oral antihistamines are sufficient. Other cases require systemic corticosteroids

(prednisolone, usually started at a dose of 0.5–1 mg/kg/day) that are considered safe in pregnancy.[84] Cases that are unresponsive to systemic corticosteroid treatment may benefit from immunopheresis.[85]

POLYMORPHIC ERUPTION OF PREGNANCY

Polymorphic eruption of pregnancy (PEP), also known as pruritic urticarial papules and plaques of pregnancy (PUPPP), toxic erythema of pregnancy, toxemic rash of pregnancy, late-onset prurigo of pregnancy) it is self-limited pruritic inflammatory disorder that is benign in nature and usually affects primigravidae during the last few weeks of pregnancy or sometimes immediately postpartum (15%).

Its incidence is 1:160 pregnancies and the condition is mainly associated with excessive maternal weight gain and multiple pregnancies .[86,87]

The exact pathogenesis till date is unclear,the various theories proposed are due to abdominal distension hormonal, and immunological factors.[86]

PEP starts within the striae distensae at the time of greatestabdominal distension that favours connective tissue damagedue to overstretching playing a central role. It issuggested that previously inert structures develop anantigenic character, thus triggering the inflammatory process.Hormonal and immunological changes and association with increasedbirth weight or male sex of the newborn have not been confirmed.[86]

Clinically the lesions starton the abdomen, usually within the striae distensae, associated with intense pruritic urticarial papules that later coalesce into plaques, which spread to the proximal thighs and the buttocks.The eruption remains located to these sites but can quickly generalize in severe cases. A characteristic finding is the

sparing of the umbilical region.The clinical picturelater becomespolymorphous, as vesicles (but never bullae), widespread non-urticated erythema, and targetoid and eczematous lesions appear in 50% of patients.The rash normally resolves within four to six weeks.[87]

The histopathology is non-specific. The diagnosis is mainly done clinically. In a skin biopsy superficial to mid-dermal perivascular lymphohistiocytic infiltrate with eosinophils, in the early biopsies a prominent dermal edema is seen and in the later biopsies show epidermal changes that include spongiosis and hyper- and parakeratosis.In PEP direct and indirect immunofluorescence are essentially negative.[88]

The maternal and fetal prognosis is excellent and there is no cutaneous involvement of the newborn.

Lesions are self-limited and PEP tends not to recur; the exception to this is seen in multiple pregnancies, where both earlier presentation and manifestation in a subsequent pregnancy may occur.[88]

Treatment is mainly symptomatic with topical corticosteroids with or without antihistamines.[87,88]If systemic antihistamines are needed during pregnancy, older sedating substances such as dimethindene, clemastine, and pheniramine are preferable due to the greater experience with their use. If a non-sedating antihistamine is required, loratadine and cetirizine can be administered safely in the second and third trimesters. If topical corticosteroids are Ineffective a short course of systemic corticosteroids (prednisolone, 40–60 mg/day, for a few days)can be given and is usually effective .[89]

INTRAHEPATIC CHOLESTASIS OF PREGNANCY (ICP)

Intrahepatic cholestasis of pregnancy is also known as jaundice of pregnancy, obstetric cholestasis, pruritus/prurigo gravidarumand cholestasis of pregnancy, it is a hormonally triggered form of cholestasis that is reversible and is typically presents in late pregnancy in genetically predisposed individuals.

In contrast to the other dermatoses of pregnancy, ICP presents with pruritus and exclusively secondary skin lesions due to scratching.

The incidence of ICP shows a very striking geographical pattern; whereas its prevalence in Central Europe is0.2 to 2.4%, it is particularly frequent in Scandinavia and South America, with the highest rates in Chile (15–28%), ICP runs in families and tends to recur in subsequent pregnancies (45–70%).[6]

Pathogenetically, there is elevated bile acids in the serum due to a defect in the excretion of bile salts. This leads to severe pruritus in the mother, and, because toxic bile acids can pass into fetal circulation, it may have deleterious effects on the fetus due to acute placental anoxia and cardiac depression.

Genetic, hormonal, and exogenous factors are some of the multifactorial reason for this defect.[6]

Mutations ofcertain geneshave been identified in some ICP patients encoding for transport proteins necessary for bile excretion (ABCB4 [MDR 3] gene).[90]

It has been demonstrated that estrogen and progesterone metabolites are cholestatic themselves.[91]Some authors also describe additional environmental and

dietary factors influencing the manifestation of ICP, like decreased serum selenium levels.[92]

Clinically ICP typically presents with severe pruritus that may start on the palms and soles but quickly becomes generalized which is sudden in onset. It persists throughout pregnancy.

ICP is not associated with any primary skin lesions. Clinical features correlate with disease duration .[93]

The skin may be unaffected; at the beginning of pruritus later on, secondary skin lesions develop from subtle excoriations to severe prurigo nodules due to scratching as pruritus persists.

Skin lesions usually involve sites such as the shins and lower arms, but may also be present on other sites such as on the buttocks and abdomen.

Jaundice, due to concomitant extrahepatic cholestasis, occurs in only 10% of patients, usually after two to four weeks, complicating the most severe and prolonged episodes.[94]

Patients are at risk of developing steatorrhea with malabsorption of fat-soluble vitamins, including vitamin K, and potential bleeding complications, as well ascholelithiasis.

In diagnosis histopathology is non-specific; direct and indirect immunofluorescences are negative.

For the diagnosis of ICP an increase in the serum bile acid level is the most sensitive indicator and the routine liver function tests may not show any abnormality in up to 30% of the patients.[95]

In healthy pregnancies, total serum bile acid levels are slightly higher than in non-pregnant women, and levels up to 11.0 μmol/l are accepted as normal in late gestation.[96,97]

The prognosis for the mother is generally good. During the postpartum period thepruritus goes within days to few weeks but may it may reoccurr with subsequent pregnancies and with the use of oral contraceptive pills.There is an increased risk for intra- and postpartum hemorrhage in both mother and child in cases of jaundice and vitamin K deficiency.

ICP is associated with an increased risk of prematurity in 19–60%, intrapartum fetal distress in 22–33%, and stillbirths in 1–2%, which correlate with higher bile acid levels, in particular if in excess of 40 μmol/l .[98]

In treatment ursodeoxycholic acid (UDCA) is the only drug that has been shown not only to reduce maternal pruritus but to also improve fetal prognosis.[99,100,101]

UDCA is a naturally occurring, hydrophilic, non-toxic bile acid that has been successfully employed in Chinese medicine for over 5,000 years in treating various liver diseases and plays a key role in treating hepatobiliary disorders and used nowadays.

In ICP, a dose of 15 mg/kg/day or, independent of body weight, 1 g/day is administered either as single dose or divided into two to three doses until delivery,

when it usually can be stopped.No adverse effects occur with the exception of occasional mild diarrhea .

However, UDCA is not licensed for use in pregnancy; thus, it requires special patient information.[101]

Other drugs, including antihistamines,Sadenosyl- L-methionine, dexamethasone, and cholestyramine, do not improve fetal prognosis. Of note, cholestyramine and other bile acid exchange resins may contribute to malabsorption of vitamin K and therefore should be avoided due to possible consecutive bleeding complications.[101]

Interdisciplinary management of ICP by dermatologists, hepatologists, gynecologists, and pediatricians is absolutely mandatory.

ATOPIC ERUPTION OF PREGNANCY(AEP)

Prurigo of pregnancy or atopic eruption of pregnancy is also known as pruritic folliculitis of pregnancy, prurigo gestationis, early-onset, prurigo of pregnancy and eczema in pregnancy it is a pruritic disorder of pregnancy in predisposed females with a personal/family history of atopy and/or elevated IgE levels and which includes eczematous and/or papular lesions after exclusion of the other dermatoses of pregnancy and it is benign in nature.[4]

It accountsfor 50% of patients, it starts early in 75% before the third trimester and due to its atopic background, tends to reoccur in the subsequent pregnancies.[4]

The pathogenesis of AEP is thought to be triggered by pregnancy-specific immunological changes; reduced cellular immunity and reduced production of Th1

cytokines (IL-2, interferon gamma, IL-12) stands in contrast to the dominant humoral immunity and increased secretion of Th2 cytokines (IL-4, IL-10).[102]

Predominant Th2 immune response that is typical for pregnancy explains the exacerbation of preexisting atopic dermatitis as well as the first manifestation of atopic skin changes.[102]

Clinically 20% of patients suffer from an exacerbation of pre-existing atopic dermatitis with a typical clinical picture.

The remaining 80% experience atopic skin changes for the first time ever or after a long remission of these, two-thirds present with widespread eczematous changes which often affect typical atopic sites such as face, neck, upper chest, and the flexural surfaces of the extremities, whereas one-third have papular lesions. These lesions include small erythematous papules disseminated on trunk and limbs, as well as typical prurigo nodules, mostly located on the shins and arms.

According to Hanifin and Rajka akey finding is the often extreme dryness of the skin and frequent atopic “minor features” [103]

Hanifin and Rajka Diagnostic Criteria for Atopic Dermatitis (AD)[103]

Major criteria: Must have three or more of: Pruritus, typical morphology and distribution flexural lichenification or linearity in adults and facial and extensor involvement in infants and children. Personal or family history of atopy and chronic relapsing dermatitis.

Minor criteria: Should have three or more of early age of onset,raised serum IgE, immediate (type 1) skin-test reactivity, ichthyosis, palmar hyperlinearity, or

keratosis pilaris, white dermographism or delayed blanch, tendency toward cutaneous infections or impaired cell-mediated immunity, tendency toward non-specific hand or foot dermatitis, orbital darkening,cheilitis, nipple eczema, recurrent conjunctivitis, pityriasis alba, dennie-Morgan infraorbital fold, keratoconus, anterior subcapsular cataracts, facial pallor or facial erythema, anterior neck folds, intolerance to wool and lipid solvents, itch when sweating, perifollicular accentuation, food intolerance and course influenced by environmental or emotional factors.

ECZEMA IN PREGNANCY

Eczema in pregnancy is a very common condition.

The reason for increased incidence of atopic eczema in pregnancy is to be due to immunological changes in pregnancy.

Pregnancy is characterized by a lack of strong maternal cell-mediated immune function and T-helper 1 (Th1) cytokine production (eg, IL-12, interferon gamma) and a dominant humoral immune response and T-helper 2 (Th2) cytokine production (e.g., IL-4, IL-10) to prevent fetal rejection.[104]

The Th2 shift associated with pregnancy favours the exacerbation of atopic dermatitis.

Ambros-Rudolph et al,[4]confirmed these findings in a retrospective two-centerstudy on 505 pregnant patients. The eruption areseen more commonly in primigravida with single gestation pregnancy and skin lesions start mostly during early pregnancy in first and second trimester. The skin eruption can affect all parts of the body, including face, palms, and soles.

PRURIGO OF PREGNANCY

It is also known as Nurse's early onset PP. It is considered as an unusual variant of PUPPP.[105]Prurigo of pregnancy is reported to occur in approximately one in 300 pregnancies.It is characterized by mainly pruritic, often excoriated papules and nodules on the extensor surfaces of the legs and upper arms.

The abdomen may also be involved. The time of onset is very variable and it has been reported to occur in all trimesters.

The etiology and pathogenesis is not known, although there is sometimes a history of atopy.[106]This condition has no recognized adverse effects for the mother or fetus.

PRURITIC FOLLICULITIS

This is a rare dermatosis and occurs in the second and third trimester of pregnancy and affects an estimated one in 3,000 pregnancies.[107]Contrary to its name, pruritus is not the major feature and it is commonly mistaken for acne or microbial folliculitis.[108]

The clinical presentation is characterized by an acneiform eruption consisting of multiple, pruritic, two to four mm, follicular papules or pustules typically on the shoulders, upper back, arms, chest, and abdomen.

The diagnosis is made clinically after excluding other, more common rashes. The skin lesions resolve spontaneously after one to two months following delivery.

Histopathology, shows an acute sterile folliculitis is evident and direct immunofluorescence stains are negative. The disorder is not associated with any

maternal or fetal morbidity, although one small series of patients showed a reduction in fetal birth weight.[108]

Laboratory tests may reveal elevated serum IgE levels in 20 to 70%.[4]

Even in severe cases maternal prognosis is good because skin lesions usually respond quickly to therapy.Therecurrence in subsequent pregnancies is common. Fetal prognosis is unaffected, but there the infant may face a higher risk of developing atopic skin changes later on.

Basic treatment is essential and consists of regular application of emollients, often with urea (3–10%) or antipruritic additives such as menthol or polidocanol. Along with topical corticosteroids for several days, this will usually lead to quick improvement of skin lesions. Severe cases may require a short course of systemic corticosteroids and antihistamines. Phototherapy (UVB) is a helpful additional measure and is considered safe in pregnancy.[111]

TREATMENT

Atopic eruption of pregnancy and polymorphic eruption of pregnancy are benign skin conditions and they do not carry any fetal risk.[4,109] They usually resolve during the early post-partum period. During pregnancy, they respond well to topical emollients and moderately potent steroids in combination with oral antihistamines. Systemic steroids may be also be used for severe cases of PUPPP.[109,110]

Pruritic folliculitis responds to topical benzoyl peroxideand also responds with narrowband (TL-01) ultraviolet B phototherapy.[111]

However, patients with pemphigoid gestationis and intrahepatic cholestasis of pregnancy carry high fetal risk and require specific treatment.

The mild pruritus can be treated with oral antihistamines but the patients with more severe cases of PG require systemic corticosteroids.[112]PG has also been treated with high-dose intravenous immunoglobulins.

ICH, ursodeoxycholic acid is considered to be the drug of choice, as it is the only therapy that has been shown to decrease both maternal pruritus and fetal mortality.[99,100]

PHARMACOKINETICS IN PREGNANCY

The unique physiologic changes of pregnancy, affects the pharmacokinetics of medications used by the pregnant women. During pregnancy, a woman's plasma volume increases by 30-50% and cardiac output and glomerular filtration rate also increase in similar proportion. These factors contribute to lower circulating concentration of some drugs (especially those excreted by kidney) in a pregnant woman and possibly to sub therapeutic drug levels. Also, there is increase in body fat during pregnancy,which in turn increases the volume of distribution of fat soluble drugs. A decrease in plasma albumin concentration during pregnancy increases the volume of distribution for highly protein bound drugs like anticonvulsants. But the unbound drugs are excreted out more rapidly by the kidney and liver, and thisoffsets the effect of increased volume of distribution. Due to the effect of progesterone and gastric emptying time is decreased particularly in the third trimester thus delaying the onset of effect of the drug.[113]

Concurrent use of other common medications during pregnancy such as antacids, iron and vitamins could also bind and inactivate some drugs. Intramuscular absorption of drug is generally more rapid due to increased blood flow; which enhances systemic drug absorption and the rate of onset of action.[113]

Lastly estrogen and progesterone alter hepatic enzyme activity, which increases the drug accumulation or decreases the elimination of some drugs.[114]

PLACENTAL TRANSFER OF DRUGS

The placenta is the product of conception and it is the functional unit between the fetal blood and maternal blood. In order for a drug to cause a teratogenic or pharmacological effect on the fetus, it must cross from maternal circulation to fetal circulation through the placenta by diffusion.[115] The rate of transfer depends on the chemical properties of the drug such as protein binding, pH difference, lipid solubility and molecular weight of the drug.[116]Only free unbound drug crosses the placenta.

During pregnancy, the maternal plasma albumin decreases while the fetal albumin increases. As a result, the concentration of free drug increases which crosses the placenta to reach the fetus. The maternal pH is less acidic compare to the fetal Ph, and weak bases are more likely to cross the placenta.[117]Moderately lipid soluble drugs can easily diffuse across the placental membrane. Lower molecular weight drugs (<500 g/mol) across the placenta and higher molecular weight drugs (between 500-1000 g/mol) cross the placenta less easily. Thereare a few drugs with molecular weightof (>1000 g/mol) which do not cross the placental membrane.[116]

Transplacental transfer of drugs increases in the third trimester due to increased maternal and placental blood flow, decreased thickness and increased surface area of the placenta.[113]

FDA CATEGORIES FOR DRUG USE IN PREGNANCY [118]

Food and Drug Administration developed a system stating the teratogenic risk of drugs from the quality of data from animal and human studies in 1979.

It provides therapeutic guidance the category A is considered the safest category but some drugs from categories B, C and D are also used during pregnancy. Category X is the only rating that denotes a drug is absolutely contraindicated for use during pregnancy.[118]

COMMONLY USED DRUGS IN PREGNANCY AND THEIR CATEGORY[119]

TABLE 1C

Drugs	**Category**
Analgesics and Antipyretics	B and C
Acetaminophen	B
Phenacetin	B
Aspirin	C
Antiemetics	B and C
Doxylamine	B
Meclizine	B
Cyclizine	B
Dimenhydrinate	B
Antibiotics	B, C and D
Penicillin, Ampicillin, Amoxycillin,	B
Cloxacillin Cephalosporins	B

Drugs	Category
Erythromycin	B
Gentamicin	C
Amikacin	C/D
Streptomycin	D
Sulphonamides	B/D
Tetracyclines	D
Amoebicides	B
Metronidazole	
Anthelmentics	B
Piperazine	
Mebendazole	
Antimalarials	C
Antifungals	C
Anti TB Drugs	B and C
Ethambutol	B
INH	C
Rifampicin	C
Pyrazinamide	C
PAS	C
Vitamins	
B,C,D,E,folic acid	A
Hormones	
Thyroxin	A
Androgens	X
Estrogens	X
Progestogens-	
Hydroxyprogestrone	D
Medroxyprogestrone	D
Norethindrone	X
Norgestrel	X
Bronchodilators	C

Among the drugs prescribed during pregnancy, antihistamines are one of the commonest, either as an antipruritic agent or an antiemetic agent.[120]

According to the FDA pregnancy category classification of drugs.[121]

Chlorpheniramine, cyproheptadine, dexchlorpheniramine, tripelennamine, cetrizine, loratadine and levocetrizine belong to pregnancy category B

Hydroxyzine,promethazine,fexofenadine and desloratidine belong topregnancy category C Category A means well-controlled studies have failed to demonstrate the risk to the fetus in pregnancy.[122]

Pregnancy category B means the drug has failed to demonstrate a risk to the fetus in animal reproduction studies and there is a lack of well-controlled studies and pregnant women or animal studies have shown an adverse effect, but well-controlled studies in pregnant women have failed to demonstrate a risk to the fetus.[122]

Pregnancy category C means that animal reproduction studies have shown an adverse effect on the fetus but there are no well-controlled studies in humans, although potential benefits may warrant the use of the drug in pregnant women.[122]

Category D means there is an evidence of human fetal risk based on the adverse reaction data from investigational, marketing experience or studies in humans, although the potential benefits may warrant use of the drug in pregnant women.[122]

Category X means studies in animals or humans have demonstrated fetal abnormalities and/or there is an evidence of human fetal risk based on data from investigational, marketing experience, and the risks involved in use of the drug in pregnant women clearly outweigh the potential benefits.[122]

MATERIALS AND METHODS

The present study 'Skin changes in pregnant women in the second and third trimester' a cross sectional study was conducted in a tertiary care hospital in Belagavi during the period January 2016 to December 2016.

A total of 104 pregnant women in the second and third trimester coming with skin changes in the outpatient and inpatient care in a tertiary care Hospital in Belagavi were selected for the study.

Procedure

1. Sample size:Considering the formula

   n= (z2 x p x q)d2

   where p is prevalence

   q is 100-p

   d is absolute error ie 10%

   z for 95%confidence is 1.96~2

   =4pq/d2

   Where p=42.3%

   q=100-p=100-42.3=57.7

   d=absolute error =10%

   n=4 x 42.3 x57.7/(10 x 10)

   97.6

   Therefore, the sample size =100

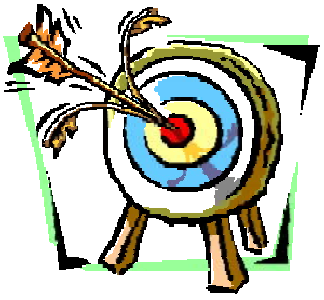
2. Ethics committee approval was obtained before the start of the study.
3. Sample selection criteria: All pregnant women in their second and third trimester presenting with dermatological complaints attending the opd and in patient services at a tertiary care Hospital in ,Belagavi, were recruited as per the inclusion and exclusion criteria.

INCLUSION CRITERIA : All pregnant women in their second and third trimester , presenting with chief complaints of cutaneous changes in the outpatient and inpatient services,willing to participate in the study.

EXCLUSION CRITERIA: Pregnant women seeking ICU care,and who do not provide informed consent were excluded in the study.

4. A written informed consent was taken from all patients in their own vernacular language.
5. Detailed history was taken and recorded in a proforma.
6. Each pregnant women underwent complete general,systemic and dermatological examinations and the findings were recorded in the proforma.
7. Photographs of the skin lesions were taken with prior consent of the patient.
8. Bedside laboratory investigations like Tzanck smear,KOH mount and Wood’slamp examination were done only if essential to confirm the diagnosis.

Introduction



Objectives

Review of Literature

Methodology

Results

Discussion

Conclusion

Summary

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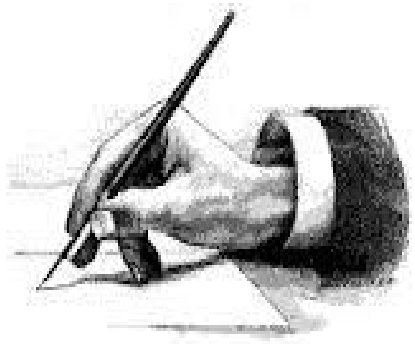


Annexure-I

Annexure-II

Annexure-III

Annexure-IV

Annexure-V

OBSERVATION AND RESULTS

The statistical analysis was performed by STAT 11.2 (college station TX USA) and IBM SPSS software .Chi-square test has been used to measure the association between the skin changes both physiological and the specific dermatoses of pregnancy with

P value <0.05 is considered as statistically significant.

Total of 104 pregnant women were enrolled in this study.

DISTRIBUTION OF PREGNANT WOMEN ACCORDING TO THEIR AGE

(TABLE .1 and FIGURE.1)

Age of the pregnant women (years)	Number of cases	Percentage
<30	88	85%
≥ 30	16	15%
Total	104	

TABLE. 1

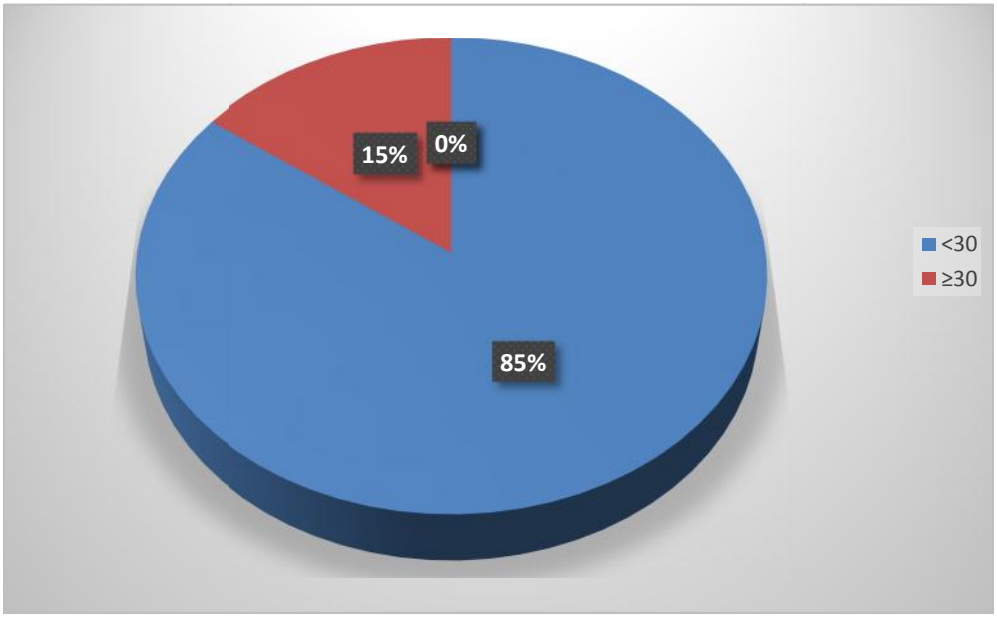


FIGURE.1

Table. 1 and Figure.1 shows 88 (85%) pregnant women were less than 30 years of age ,16 (15%) pregnant women were 30 years of age and above.

DISTRIBUTION OF PREGNANT WOMEN ACCORDING TO GRAVIDA

(TABLE. 2 and FIGURE. 2)

Gravida score	No of cases	Percentage
G1	55	53%
G2	20	19%
G3	19	18%
G4	10	10%
Total	104	

TABLE.2

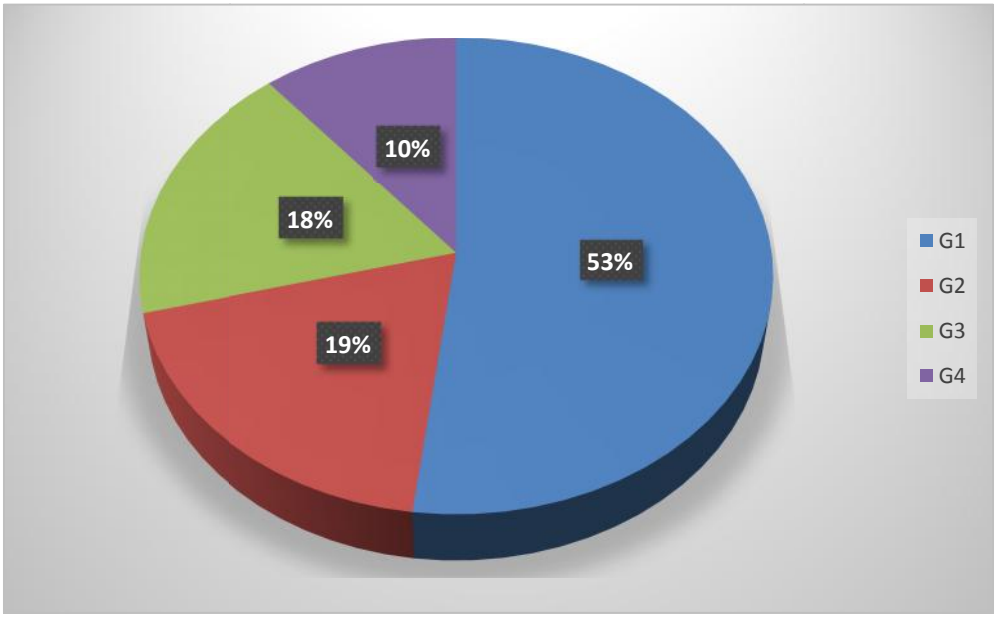


FIGURE .2

Table.2 and Figure.2 shows 55(53%) of the pregnant women had a gravida score of 1, 20 (19%) had a score of 2, 19(18%) had a score of 3 and 10(10%) of women had a score of 4 .

DISTRIBUTION ACCORDING TO PARITY

(TABLE.3 and FIGURE.3)

Parity score	Number of cases	Percentage
P0	17	16%
P1	40	39%
P2	28	27%
P3	14	13%
P4	5	5%
Total	104	

TABLE .3

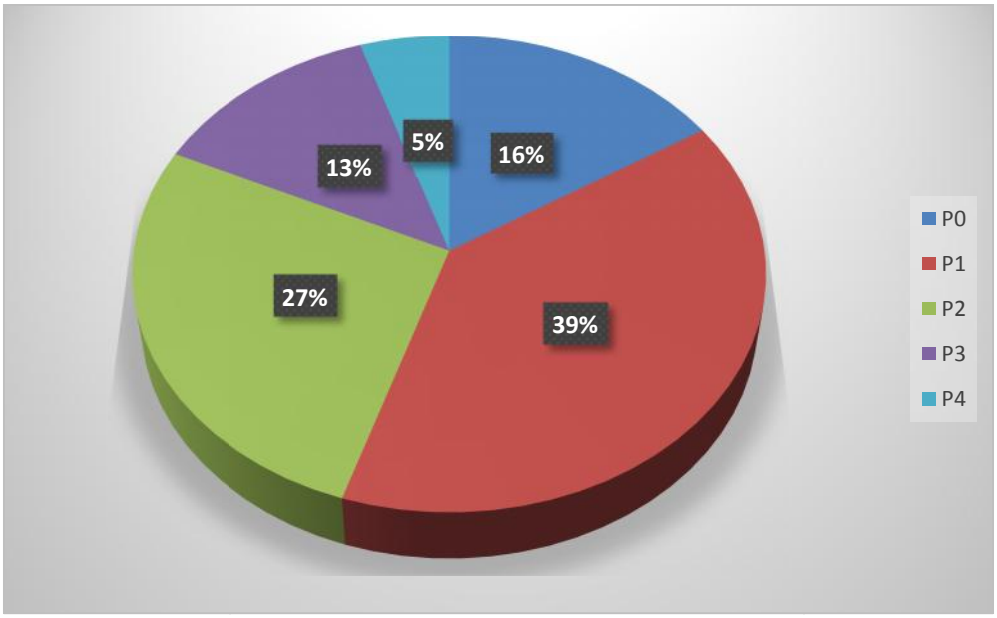


FIGURE 3

Table.3 and Figure.3 shows 17(16%) of the women had a parity score of 0 ,40 (39%) had a score of 1 ,28(27%) had a score of 2 ,14(13%) had a score of 3 and 5 (5%) had a score of 4

DISTRIBUTION BASED ON THE NUMBER OF LIVING CHILDREN

(TABLE. 4 and FIGURE.4)

Number of living children	Number of cases	Percentage
L0	54	52%
L1	28	28%
L2	18	18%
L3	2	2%
Total	104	

TABLE.4

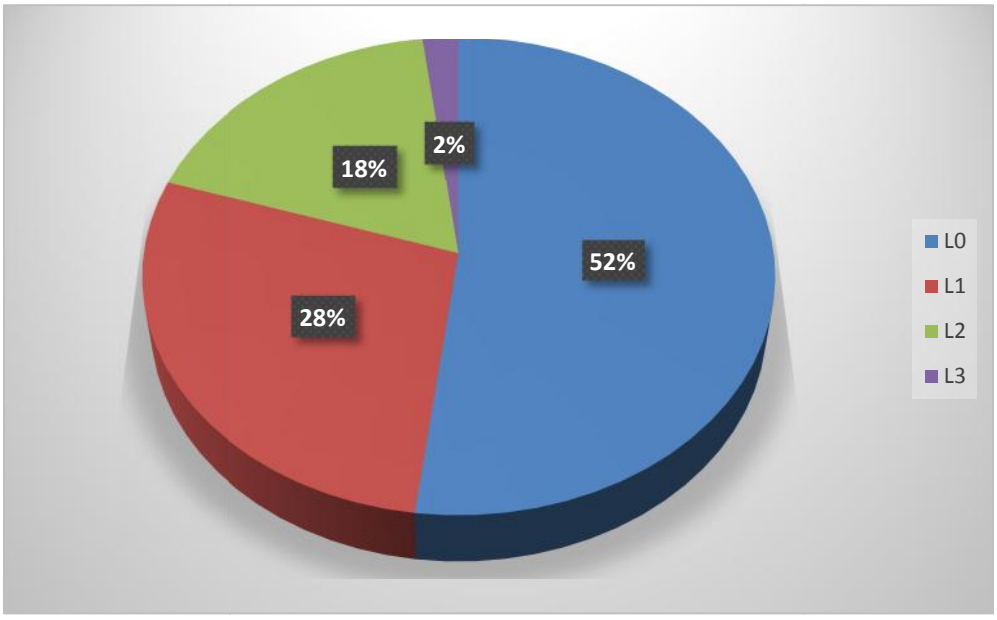


FIGURE.4

Table.4 and Figure.4 shows 54(52%) of women did not have any children,28 (28%) of women had 1 child ,18(18%) of the women had 2 children and 2 (2%) had 3 children

DISTRIBUTION BASED ON ABORTION

(TABLE.5 and FIGURE. 5)

Number of abortions	Number of cases	Percentage
A0	88	85%
A1	15	14%
A2	1	1%
Total	104	

TABLE. 5

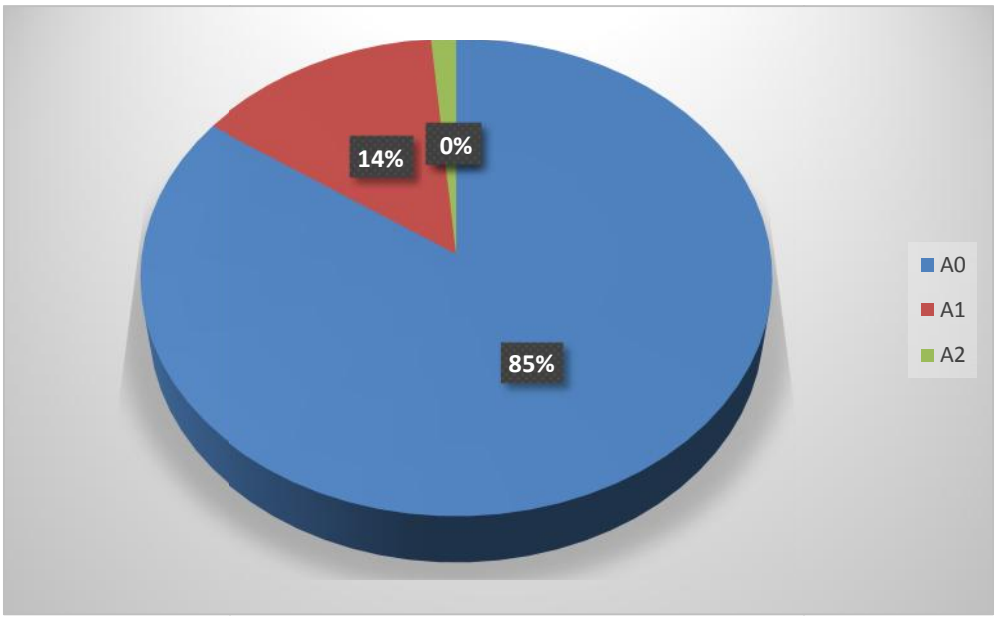


FIGURE. 5

Table.5 and Figure.5 shows 88(85%) of the women did not have any abortion, 15(14%) had 1 abortion and 1 (1%) woman had 2 abortions.

DISTRIBUTION BASED ON THE OCCUPATION

(TABLE.6 and FIGURE.6)

Occupation	Number of cases	Percentage
House wife	52	50%
Self employed	19	18%
Factory worker	7	7%
Officer	4	4%
Farmer	19	18%
Teacher	3	3%
Total	104	

TABLE .6

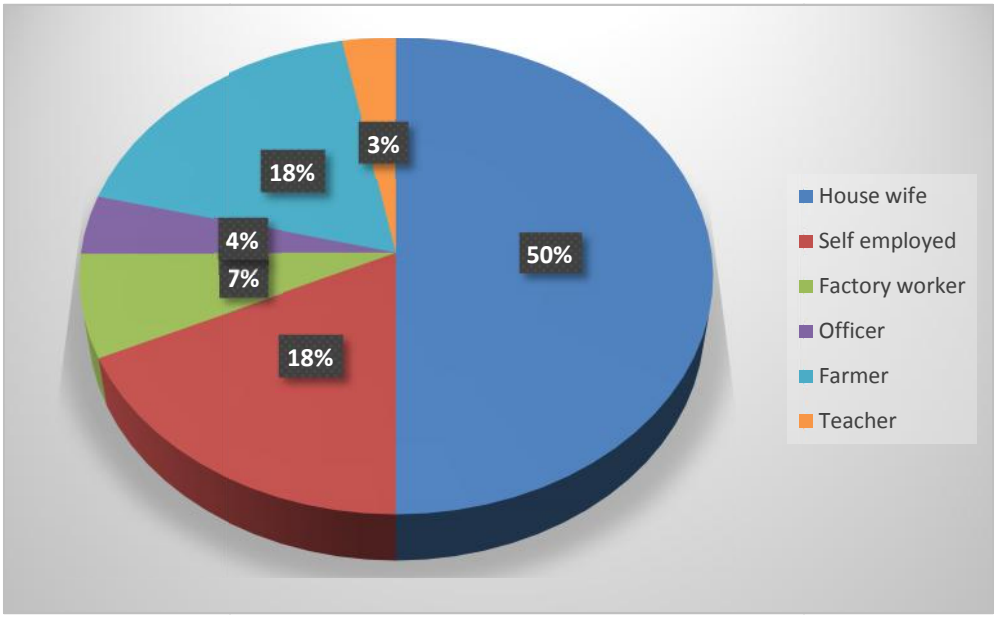


FIGURE..6

Table.6 and Figure.6 shows 52(50%) of the pregnant women were housewives,19 (18%) were self employed,7 (7%) were factory workers ,4(4%)worked in an office ,19(18%) were farmers, and 3 women were teachers.

DISTRIBUTION BASED ON THE TRIMESTER

(TABLE.7 and FIGURE.7)

Trimester	Number of cases	Percentage
2nd	62	60%
3rd	42	40%
Total	104	

TABLE .7

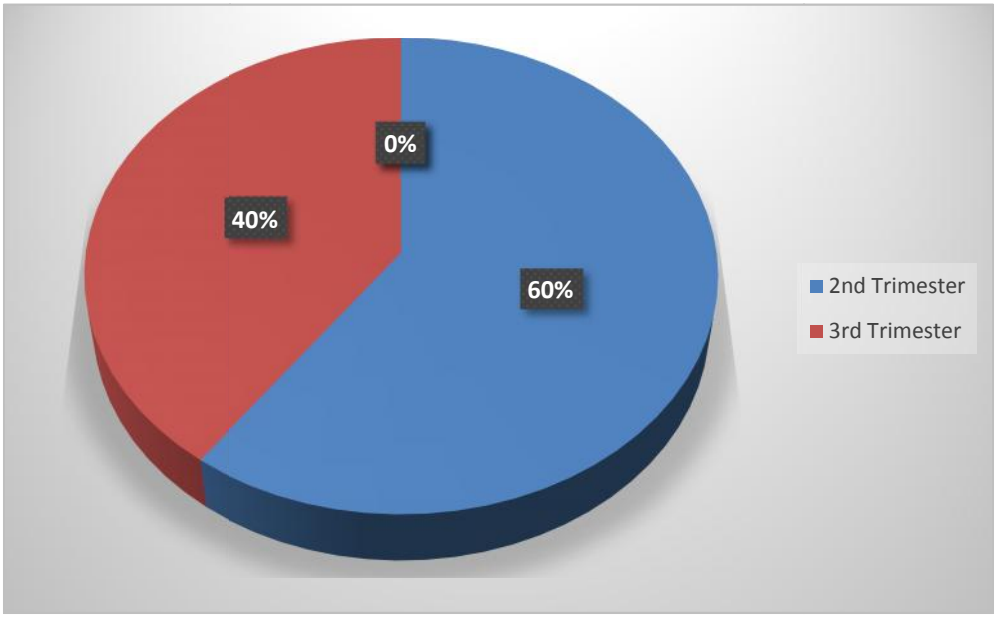


FIGURE .7

Table.7 and Figure.7 shows 62(62%)of the pregnant women were in their second trimester and 42 (40%)were in their third trimester.

DISTRIBUTION BASED ON THE CHIEF COMPLAINT

(TABLE.8 and FIGURE.8)

Chief complaints	Number of cases	Percentage
Itching	8	8%
Itchy lesions	45	43%
Asymptomatic lesions	11	10%
Pigmentation	20	19%
Strech marks	9	9%
Pimples	3	3%
Fluid filled lesions	2	2%
Swelling in groin	2	2%
Dryness	1	1%
Painful lesions	1	1%
Facial hair	1	1%
White patches	1	1%
Total	104	

TABLE.8

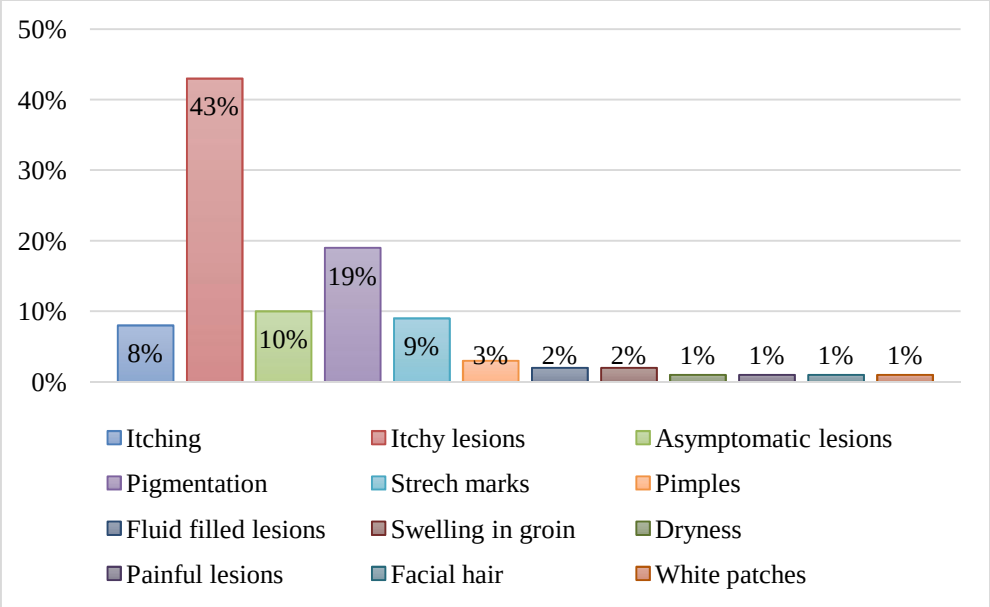


FIGURE.8

Table 9 and Figure 8 shows that the most common 45(43%) chief complaint was itchy lesions ,followed by pigmentation 20(19%)

11(10%) pregnant women had asymptomatic lesions

9 (9%) of the pregnant women had a chief complaint of stretch marks.

8(8%) complaint of itching

3(3%) complaint of pimples

2(2%)of the pregnant women had the complaint of fluid filled lesions

2 (2%)complaint of swelling in the groin.

1 (1%) woman complaint of dryness

1(1%)woman had the chief complaint of facial hair.

1(1%) woman complaint of white patch.

DISTRIBUTION OF PREGNANT WOMEN ACCORDING TO THE SKIN CHANGES

(TABLE.9 and FIGURE.9)

Skin changes	Number of cases	Percentage
Physiological	94	90%
Specific dermatoses	28	27%

TABLE.9

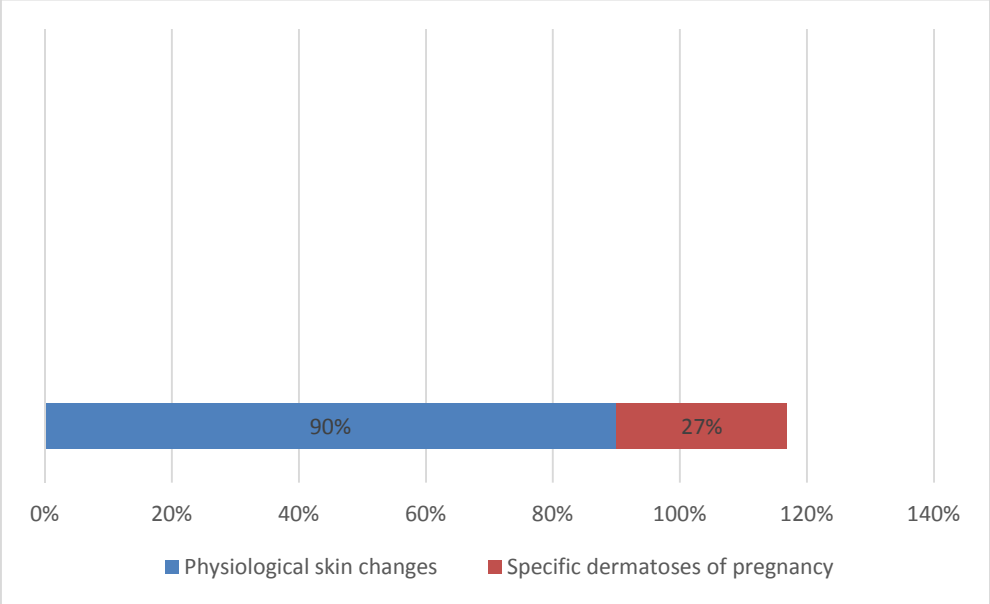


FIGURE.9

Table.9 and Figure.9 shows that out of the 104 pregnant women 94(90%)of the women had physiological skin changes and 28(27%) of the women had specific dermatoses of pregnancy.

PHYSIOLOGICAL SKIN CHANGES

(TABLE.10 and FIGURE.10)

Physiological skin changes	Number of cases	Percentage
Linea nigra	59	57%
Hyperpigmentation of areola	32	31%
Straie gravidarum	46	44%
Chloasma	17	16%
Oedema	11	10%
Varicosities	9	9%
Palmar erythema	4	4%
Pyogenic granuloma	3	3%
Gingival hyperplasia	1	1%
Hirsutism	3	3%
Subungual hyperkeratosis	6	6%
Distal onycholysis	2	2%
Nil	10	10%

TABLE.10

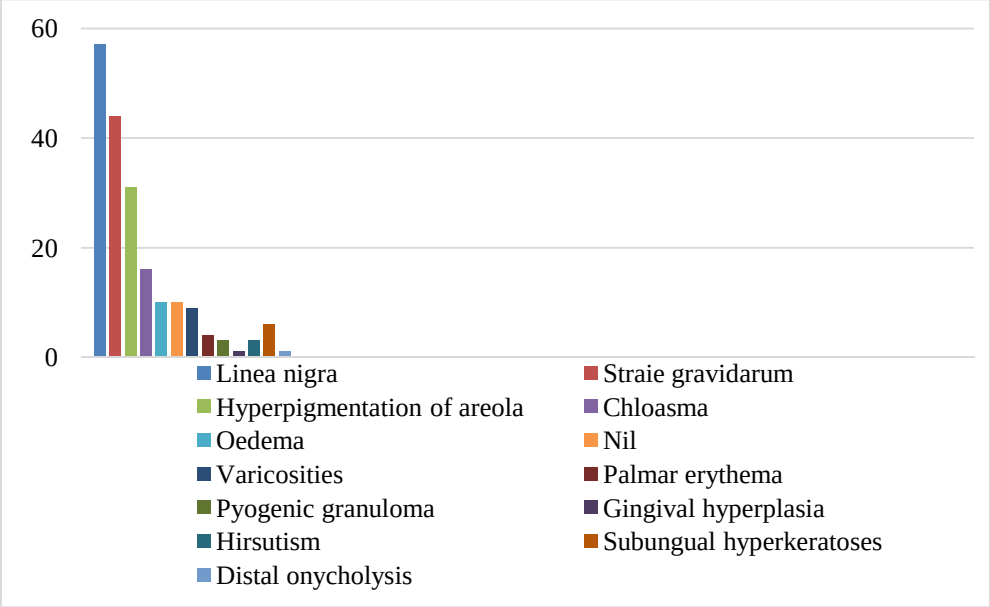


FIGURE.10

Table 11 and Figure 10 shows that out of the 104 pregnant women 94 (90%) had physiological skin changes.

The most common physiological skin change seen amongst 104 pregnant was linea nigra 59(57%).

The second most common physiological skin change noted was straie gravidarum 46(44%)

The third most common physiological skin change observed was hyperpigmentation of the areola.

17(16%)of the pregnant women had chloasma.

11(10%) had bilateral non pitting pedal oedma

9(9%)of the pregnant women had bilateral varicose veins of the lower limbs.

6(6%)of the women had subungual hyperkeratosis.

4(4%)of the pregnant women were observed with palmar erythema.

3(3%) were observed with hirsutism.

3(3%)had pyogenic granuloma.

2((2%)pregnant women had distal onycholysis.

1(1%)of the pregnant women was noted with gingival hyperplasia.

Out of the 104 pregnant women enrolled 10(10%)pregnant were not noted with any physiological skin changes.(Table.10 and Figure.10)

SPECIFIC DERMATOSES OF PREGNANCY

(TABLE.11 and FIGURE.11)

Specific dermatoses of pregnancy	Number of cases	Percentage
Pruritus of pregnancy	4	4%
Pruritic urticarial papules and plaques of pregnancy (PUPPP)	9	9%
Prurigo of pregnancy	9	9%
Pruritic folliculis of pregnancy	2	2%
Eczema in pregnancy	4	4%
Total	28	28%

TABLE.11

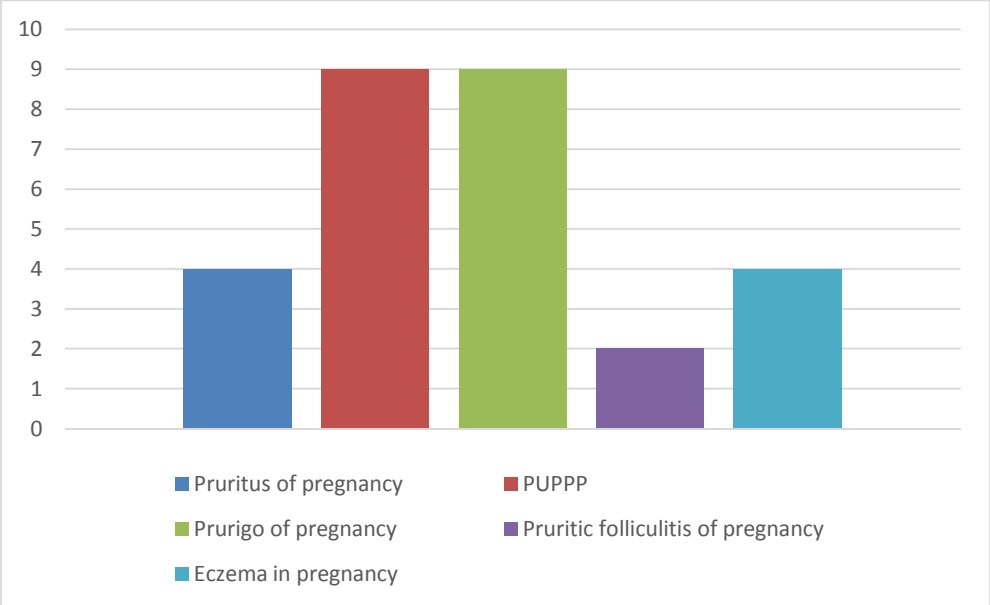


FIGURE .11

Table.11 and Figure.11 show that 28(27%)of the pregnant women had specific dermatoses of pregnancy.

Prurigo of pregnancy 9(9%) and pruritic urticarial papules and plaques of pregnancy(PUPPP) 9(9%)were the two most common specific dermatoses of pregnancy observed.

Followed by pruritus of pregnancy 4(4%)and eczema in pregnancy4(4%).

2(2%)of the pregnant women had pruritic folliculitis of pregnancy.

OTHER SKIN CONDITIONS NOT RELATED TO PREGNANCY

(TABLE.12 and FIGURE.12)

Other skin conditons	Number of cases	Percentage
Tinea corporis	14	13%
Tinea cruris	6	6%
Pityriasis versicolor(P.versicolor)	8	8%
Irritant contact dermatitis(ICD)	4	4%
Polymorphic light eruption(PMLE)	1	1%
Lymphadenoma	1	1%
Lymphangitis	1	1%
Intertrigo	1	1%
Acne vulgaris (AV)	3	3%
Varicella	3	3%
Vitiligo vulgaris	1	1%
Seborrheic dermatitis	1	1%
Acrochordons	1	1%
Herpes labialis	1	1%
Total	46	44%

TABLE.12

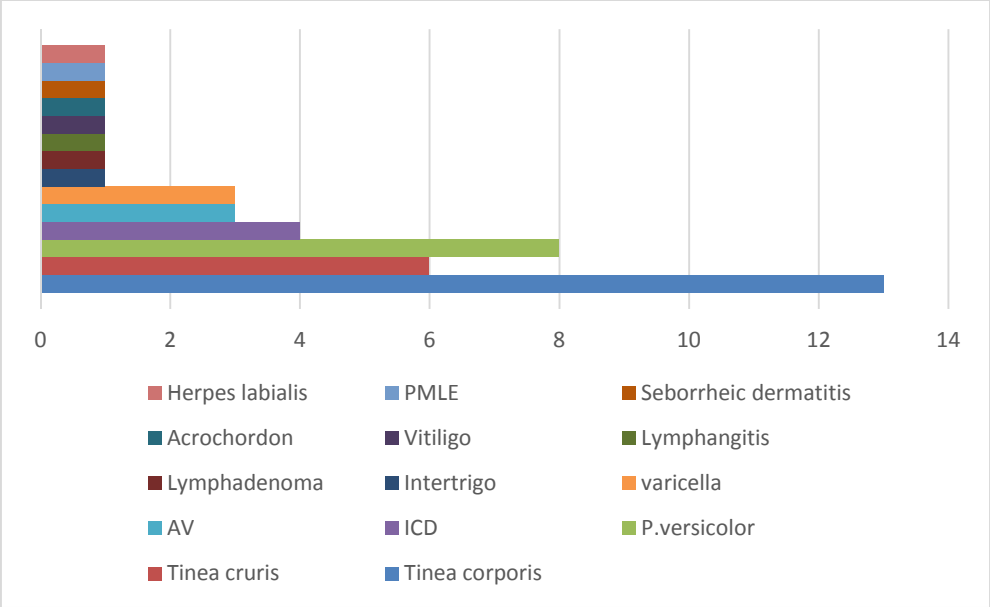


FIGURE.12

Table.12 and Figure.12 show that out of the 104 enrolled pregnant women 46(44%)had skin conditions which were not related to pregnancy.

The most common skin condition observed was tinea corporis 14(13%).

The second most skin condition noted was pityriasis versicolor 8(8%).

The third most common skin condition unrelated to pregnancy was tinea cruris 6(6%).

4(4%)of the pregnant women had irritant contact dermatitis.

3 (3%)of the pregnant women were noted with varicella.

3(3%)had acne vulgaris.

1(1%)women had polymorphic light eruption (PMLE).

1(1%) had lymphadenoma.

1(1%)pregnant woman had lymphangitis.

1(1%)had intertrigo.

1(1%)woman had vitiligo vulgaris.

1(1%)woman was noted with herpes labialis.

1(1%)had seborrheic dermatitis.

1(1%)woman had acrochordons

ASSOCIATION OF AGE WITH GRAVIDA

(TABLE.13 and FIGURE.13)

		Age		Total
		Age<30	Age≥ 30	
Gravida	G1	55(100%)	0	55
	G2	16(84%)	3(16%)	19
	G3	13(65%)	7(35%)	20
	G4	4(40%)	6(60%)	10
Total		88	16	104

TABLE.13

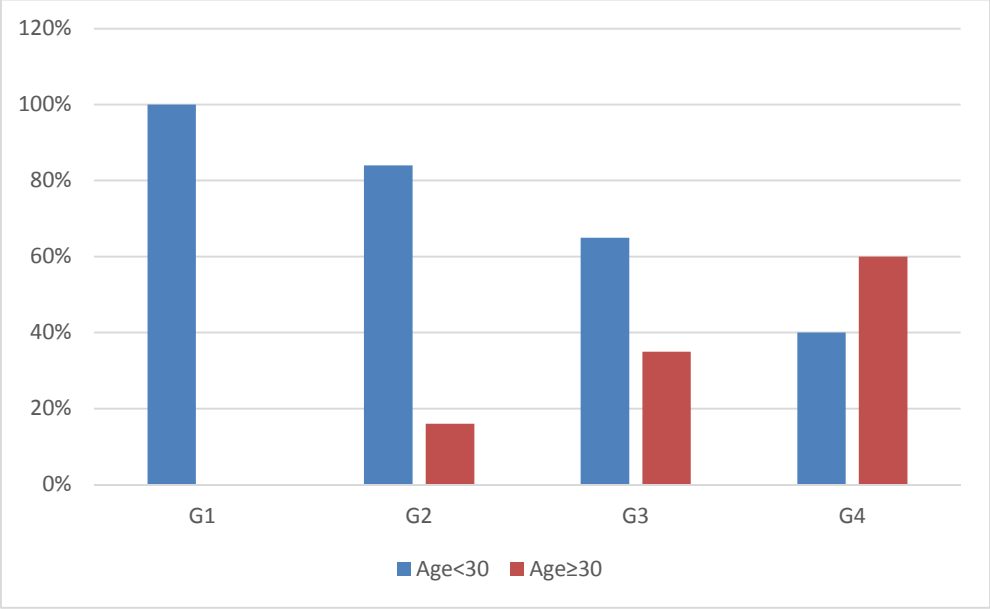


FIGURE.13

Table.13 and figure.13 shows the association of age with gravida ,in this study 55(100%) women were less than 30 years of age who had a gravida score of one(G1)

16 (84%)were below 30 years and had a gravida score of two(G2)

13 (65%)women were below 30 years and had a gravida score of three(G3)

4 (40%)women below 30 years had a gravida score of four(G4)

There were no women30 years and above with a gravida score of one (G1).

3(16%)women were 30 years and above and had a gravida score of two(G2)

7(35%)women were 30 years and above and had a gravida score of three(G3)

6(60%)women were 30 years and above and had a gravida score of four(G4)

ASSOCIATION OF AGE AND TRIMESTER

(TABLE.14 and FIGURE.14)

		Trimester		
		Second	Third	Total
Age ≥30		8(50%)	8(50%)	16
Age <30		55(63%)	33(25%)	88
Total		63	41	104

TABLE.14

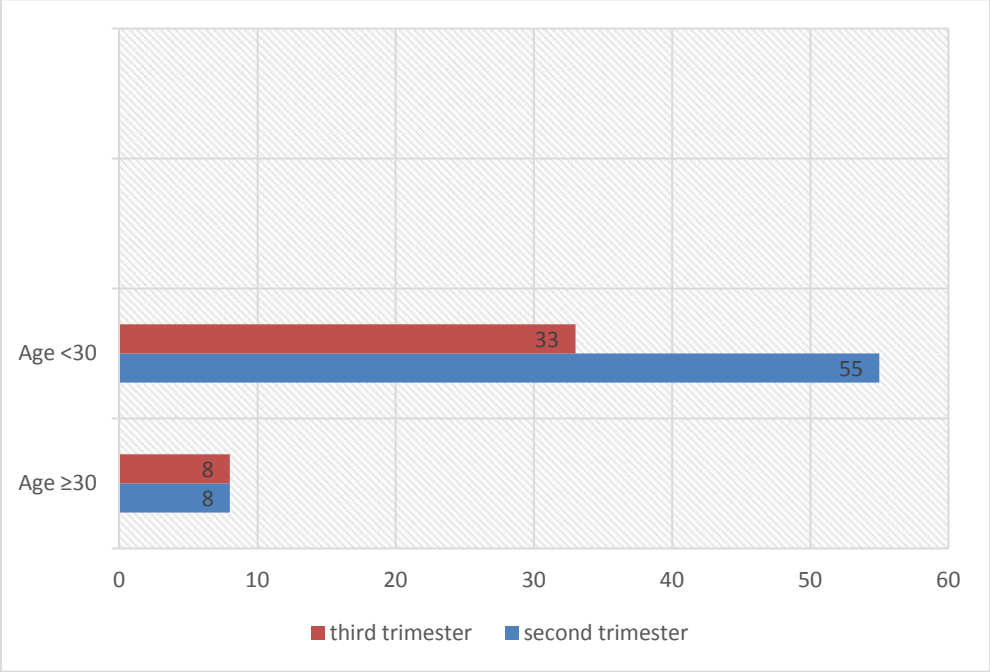


FIGURE.14

Table.14 and Figure.14 show the association of age with trimester. 8(50%)pregnant women 30 years and above were in their second trimester and 8(50%) were in their third trimester.55(63%)pregnant women below 30 were in their second trimester and 33(25%) in their third trimester.

CORRELATION OF LINEA NIGRA WITH VARIOUS PARAMETERS

CORRELATION OF LINEA NIGRA WITH AGE

(TABLE.15 and FIGURE.15)

		LINEA NIGRA			
		Absent	Present	Total	P value
Age <30		39(44%)	49(56%)	88	
Age ≥30		6(38%)	10(10%)	16	0.256
Total		45	59	104	

TABLE.15

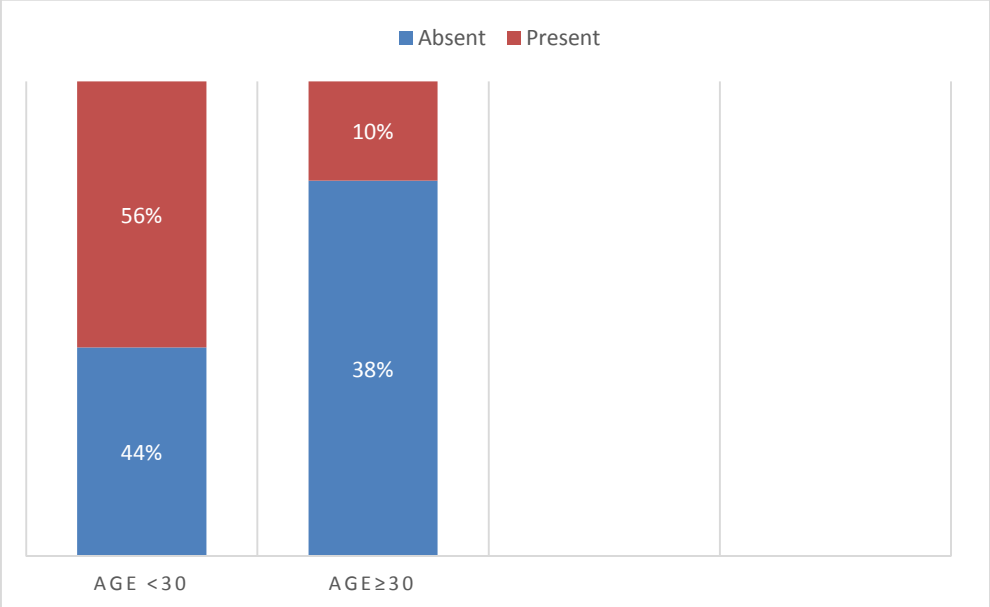


FIGURE.15

Table.15 and Figure.15 shows the correlation of linea nigra with age.49(56%) women below 30 years and 10(10%)women 30 years and above had linea nigra.

CORRELATION OF LINEA NIGRA WITH GRAVIDITY

(TABLE.16 and FIGURE.16)

		LINEA NIGRA			
		Absent	Present	Total	P value
Gravida	G1	27(50%)	28(51%)	55	
	G2	4(21%)	15(79%)	19	
	G3	9(45%)	11(55%)	20	4.78
	G4	5(50%)	5(50%)	10	
Total		45	59	104	

TABLE.16

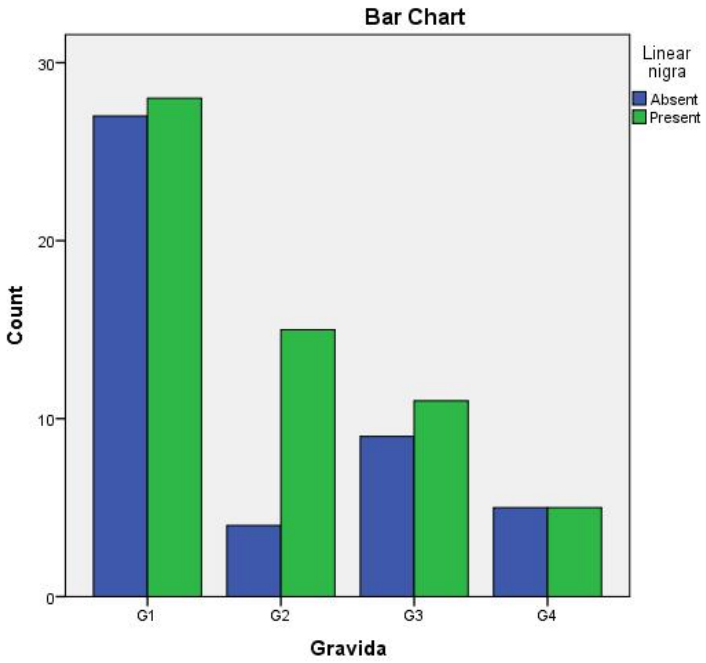


FIGURE.16

Table.16 and Figure.16 show the correlation of linea nigra with gravida.28(51%) women in gravida one(G1), 15(79%)in gravida two(G2), 11(55%)women in gravida 3 (G3) and 5(50%)women in gravida four(G4)had linea nigra.

CORRELATION OF LINEA NIGRA WITH PRIMIGRAVIDAE /MULTIGRAVIDAE

(TABLE.17 and FIGURE.17)

		LINEA NIGRA			
		Absent	Present	Total	P value
Multigravida Primigravida		18(37%)	31(63%)	49	
		27(49%)	28(51%)	55	1.06
Total		45	59	104	

TABLE.17

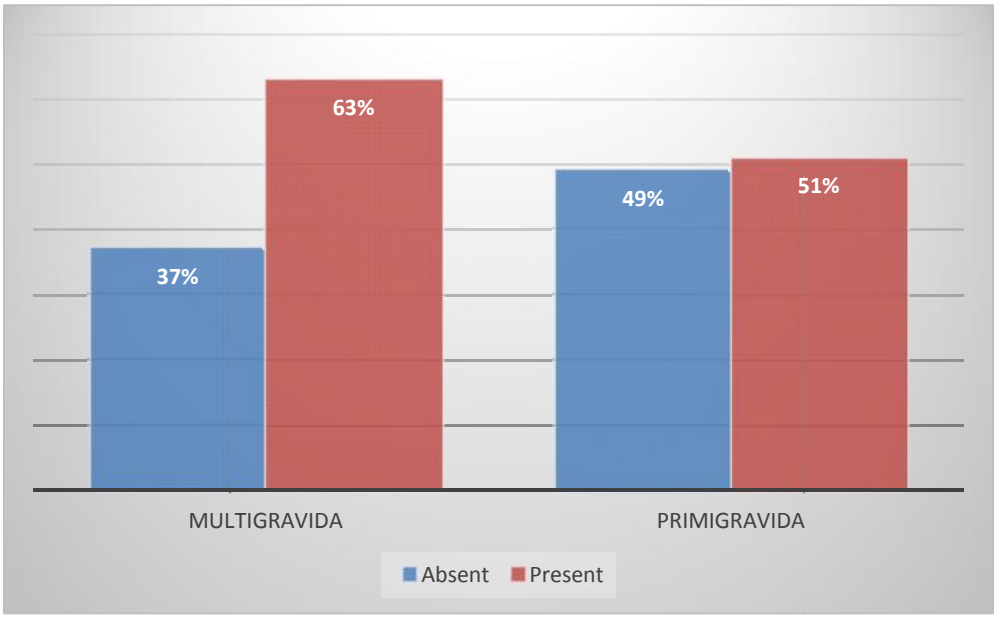


FIGURE.17

Table.17 and Figure.17 show the correlation of linea nigra with primigravidae/multigravidae.31(63%)primigravidae and 28(51%) multigravidae had linea nigra.

CORRELATION OF LINEA NIGRA WITH TRIMESTER

(TABLE.18 and FIGURE.18)

Trimester		LINEA NIGRA		Total	P value
		Absent	Present		
	Second	41(65%)	22(35%)	63	
	Third	4(10%)	37(90%)	41	30
Total		45	59	104	

TABLE.18

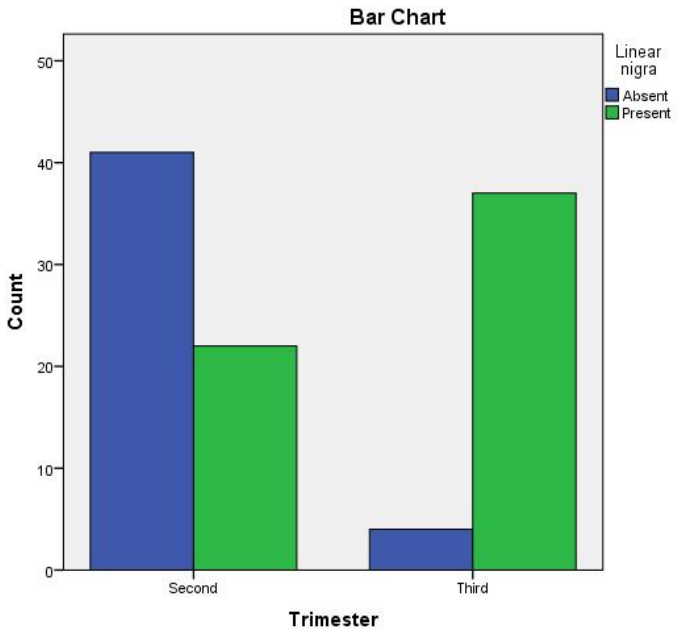


FIGURE 18

Table.18 and Figure.18 shows the correlation of linea nigra with trimester.22(35%) women in second trimester and 37(90%)in third trimester had linea nigra.

CORRELATION OF HYPERPIGMENTATION OF AREOLA WITH VARIOUS PARAMETERS

CORRELATION OF HYPERPIGMENTATION OF AREOLA WITH AGE

(TABLE.19 and FIGURE.19A ,19B)

		HYPERPIGMENTATION OF AREOLA			
		Absent	Present	Total	P value
Age <30		62(70%)	26(30%)	88	0.39
Age ≥30		10(63%)	6(37%)	16	
Total		72	32	104	

TABLE 19

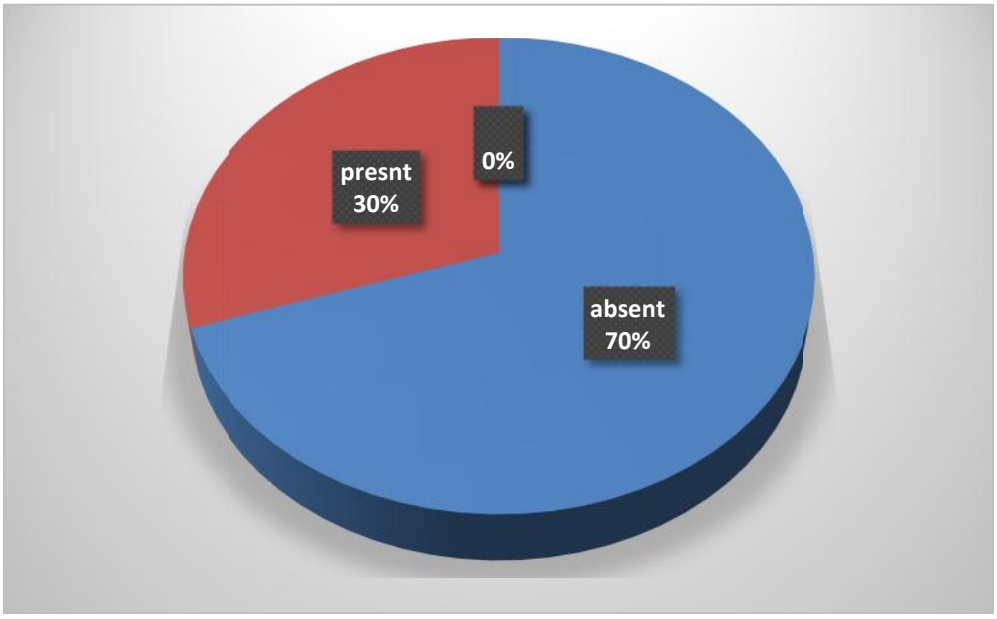


FIGURE.19A

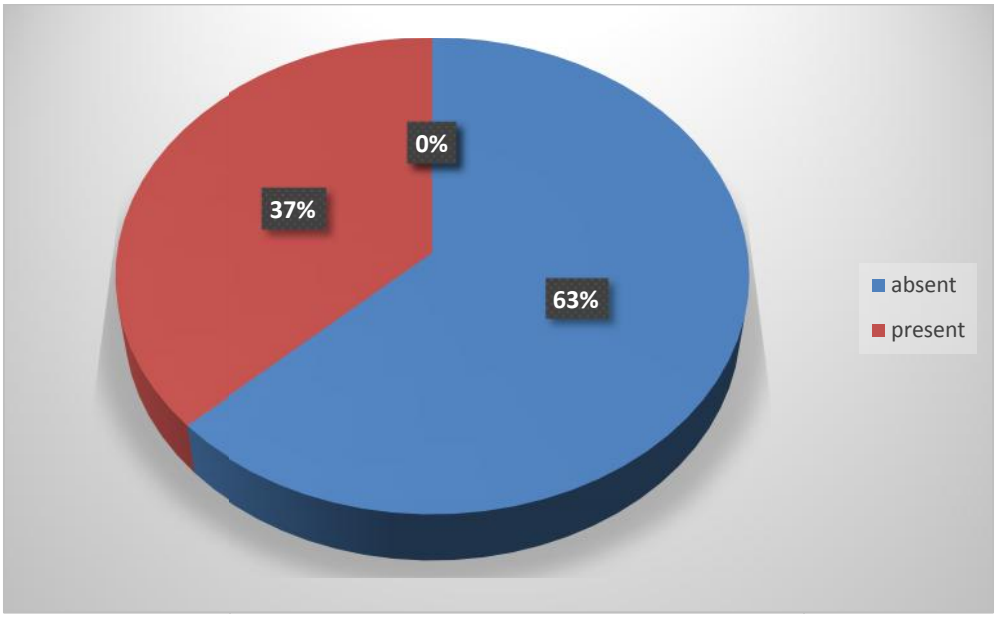


FIGURE. 19B

Table.19 and Figure.19A and 19B show the correlation of hyperpigmentation of the areola with age.26(30%)of pregnant women below 30 years and 6(37%)30 years and above had hyperpigmentation of the areola.

CORRELATION OF HYPERPIGMENTATION OF AREOLA WITH GRAVIDITY

(TABLE.20 and FIGURE.20)

		HYPERPIGMENTATION OF AREOLA			
		Absent	Present	Total	P value
Gravida	G1	37(67%)	18(33%)	55	
	G2	10(53%)	9(47%)	19	6.60
	G3	18(90%)	2(10%)	20	
	G4	7(70%)	3(30%)	10	
Total		72	32	104	

TABL. 20

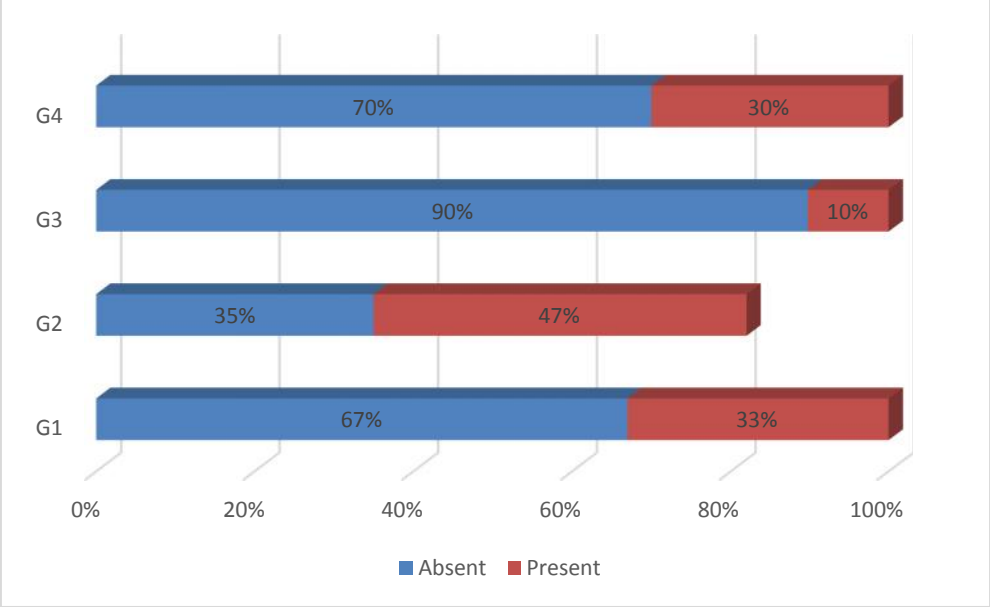


FIGURE.20

Table.20 and Figure.20 show the correlation of hyperpigmentation of areola with gravidity.18(33%)in gravida one(G1) 9(47%) in gravida two(G2)2(10%)in gravida three(G3) and 3(30%)in gravida (G4) had hyperpigmentation of the areola.

CORRELATION OF HYPERPIGMENTATION OF AREOLA WITH PRIMIGRAVIDAE /MULTIGRAVIDAE

(TABLE.21 and FIGURE .21)

		HYPERPIGMENTATION OF AREOLA			
		Absent	Present	Total	P value
Multigravida		35(71%)	14(29%)	49	
Primigravida		37(67%)	18(33%)	55	0.21
Total		72	32	104	

TABLE.21

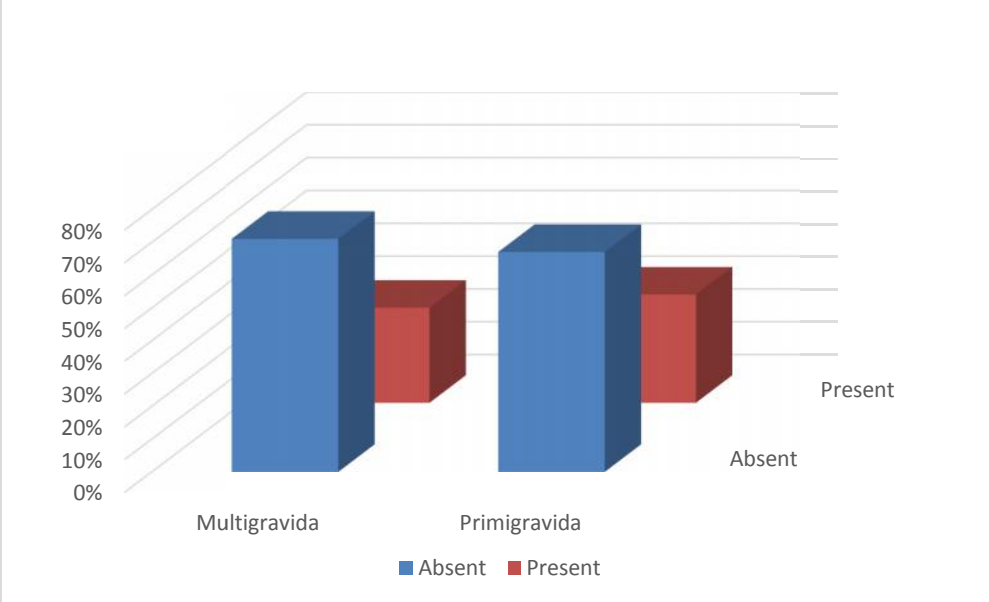


FIGURE.21

Table.21 and Figure.21 show the correlation of hyperpigmentation of areola with primigravidae /multigravidae.14(29%)primigravidae and 18(33%)multigravidae had hyperpigmentation of areola.

CORRELATION OF HPERPIGMENTATION OF AREOLA WITH TRIMESTER

(TABLE.22 and FIGURE.22)

Trimester	HYPERPIGMENTATION OF AREOLA		Total	
	Absent	Present		P value
Second Third	46(73%)	17(27%)	63	
	26(63%)	15(37%)	41	1.07
Total	72	32	104	

TABLE.22

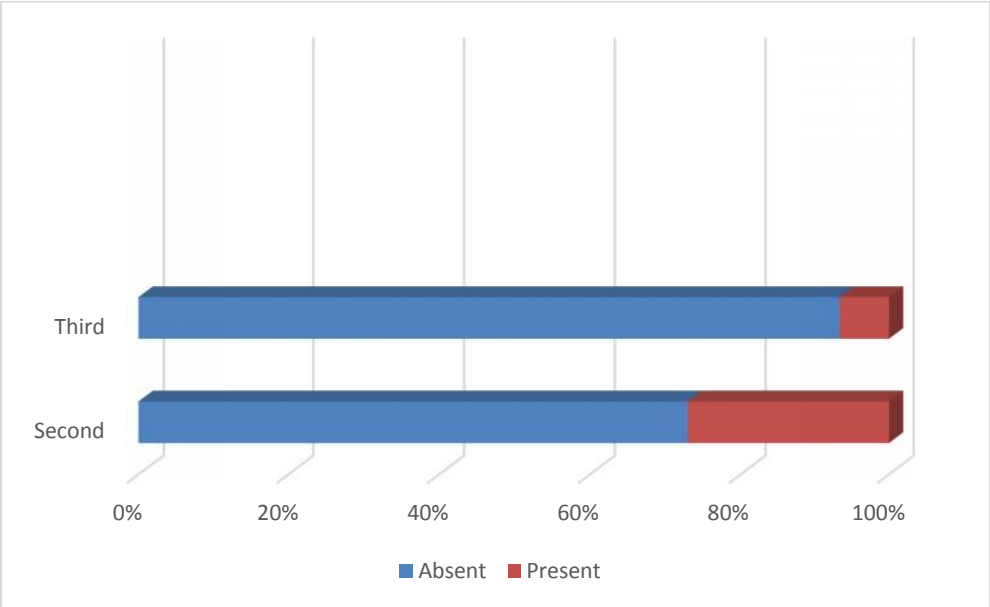


FIGURE.22

Table.22 and Figure.22 show the correlation of hyperpigmentation of areola with trimester.17(27%)in second trimester and 15(37%)in third trimester had hyperpigmentation of areola.

CORRELATION OF STRAIE GRAVIDARUM WITH VARIOUS PARAMETERS

CORRELATION OF STRAIE GRAVIDARUM WITH AGE

(TABLE.23 and FIGURE.23)

Age		STRAIE GRAVIDARUM		Total	P value
		Absent	Present		
Age<30		48(55%)	40(45%)	88	
Age ≥30		10(62%)	6(38%)	16	0.4
Total		58	46	104	

TABLE.23

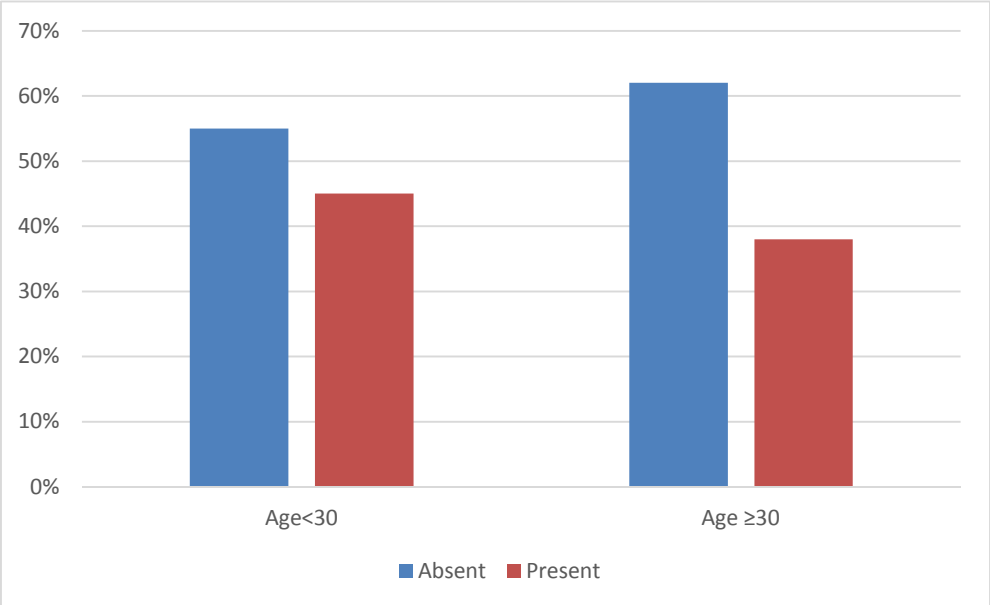


FIGURE.23

Table.23 and Figure.23 show the correlation of straie gravidarum with age.40 (45%)pregnant women below 30 years and 6(48%)pregnant women 30 years and above had straie gravidarum.

CORRELATION OF STRAIE GRAVIDARUM WITH GRAVIDITY

(TABLE.24 and FIGURE.24)

		STRAIE GRAVIDARUM			
		Absent	Present	Total	P value
Gravida	G1	34(62%)	21(38%)	55	
	G2	8(42%)	11(58%)	19	
	G3	11(55%)	9(45%)	20	2.37
	G4	5(50%)	5(50%)	10	
Total		58	46	104	

TABLE.24

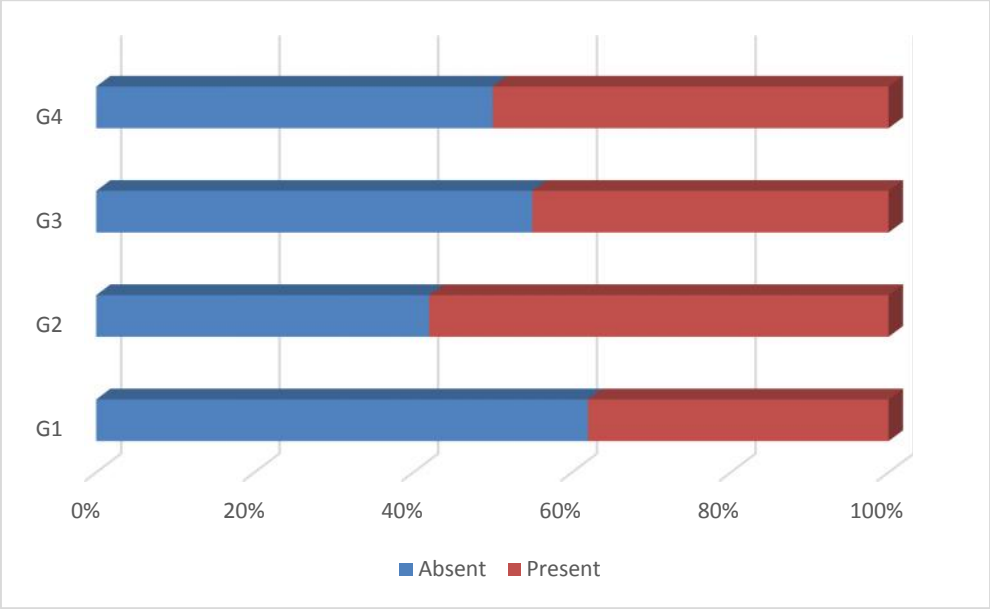


FIGURE.24

Table.24 and Figure.24 show the correlation of straie gravidarum with gravidity.21(38%)in gravida one(G1),11(58%)in gravida two(G2),9(45%)in gravida 3(G3) and 5(50%) in gravida four(G4) had straie gravidarum.

CORRELATION OF STRAIE GRAVIDARUM WITH PRIMIGRAVIDAE MULTIGRAVIDAE

(TABLE.25 AND FIGURE.25)

		STRAIE GRAVIDARUM			
		Absent	Present	Total	P value
Multigravida		24(49%)	25(51%)	49	
Primigravida		34(62%)	21(38%)	55	1.73
Total		58	46	104	

TABLE.25

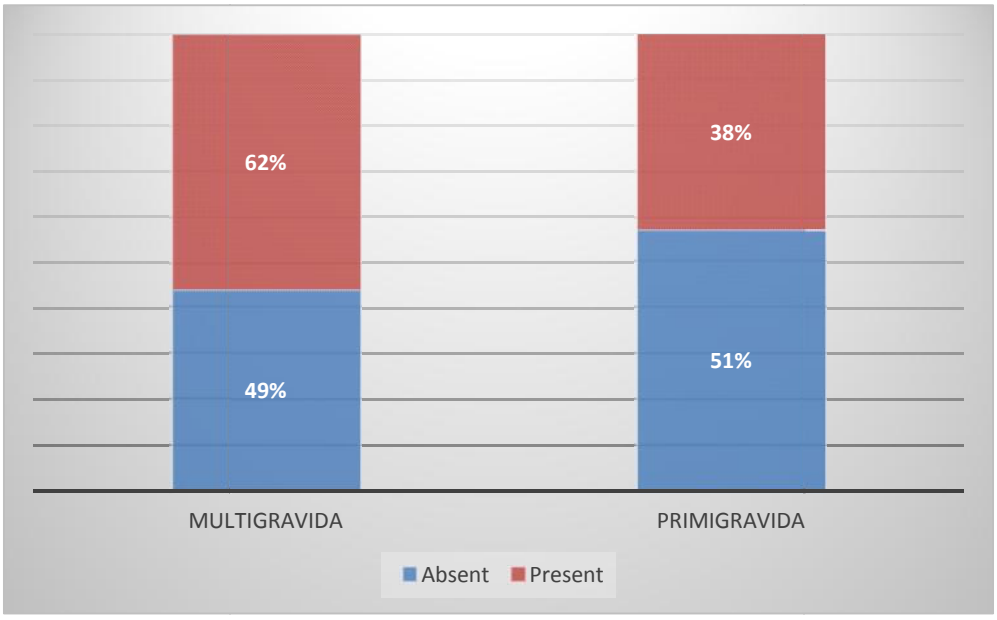


FIGURE.25

Table.25 and Figure.25 show the correlation of straie gravidarum with primigravidae/multigravidae.25(51%) mutigravidae and 21(38%)primigravidae had straie gravidarum.

CORRELATION OF STRAIE GRAVIDARUM WITH TRIMESTER

(TABLE .26 and FIGURE.26)

Trimester	STRAIE GRAVIDARUM		Total	P value
	Absent	Present		
Second	42(67%)	21(33%)	63	
Third	16(39%)	25(61%)	41	7.1
Total	58	46	104	

TABLE.26

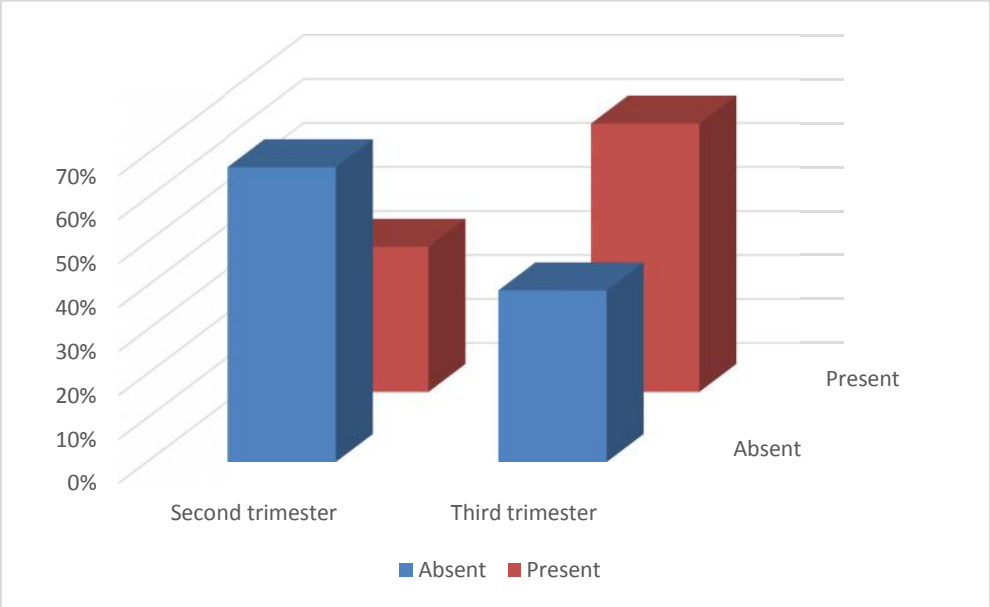


FIGURE.26

Table.26 and Figure.26 show the correlation of straie gravidarum with trimester.21(33%)of pregnant women in second trimester and 25(61%)women in third trimester had straie gravidarum.

CORRELATION OF CHLOASMA WITH VARIOUS PARAMETERS

CORRELATION OF CHLOASMA WITH AGE

(TABLE.27 and FIGURE.27)

		CHLOASMA			
		Absent	Present	Total	P value
Age<30		73(83%)	15(17%)	88	
Age≥30		14(88%)	2(12%)	16	0.20
Total		87	17	104	

TABLE.27

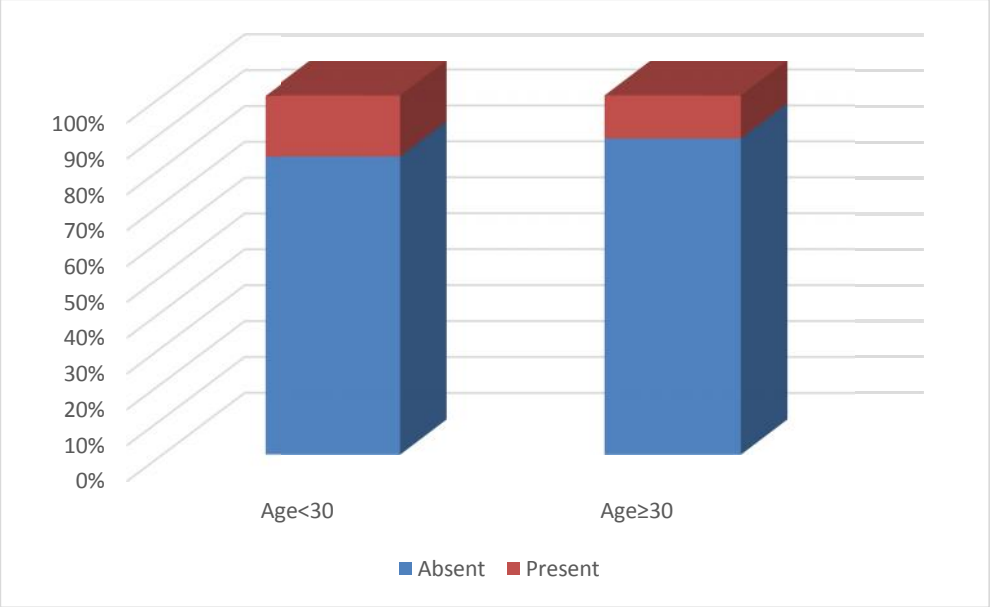


FIGURE.27

Table.27 and Figure.27 show the co relation of chloasma with age.15(17%)of pregnant women below 30 years of age and 2(12%)of pregnant women 30 years and above had chloasma.

CORRELATION OF CHLOASMA WITH GRAVIDITY

(TABLE.28 and FIGURE.28)

		CHLOASMA			
		Absent	Present	Total	P value
Gravida	G1	45(82%)	10(18%)	55	
	G2	15(79%)	4(21%)	19	2.42
	G3	19(95%)	1(5%)	20	
	G4	8(80%)	2(20%)	10	
Total		87	17	104	

TABLE .28

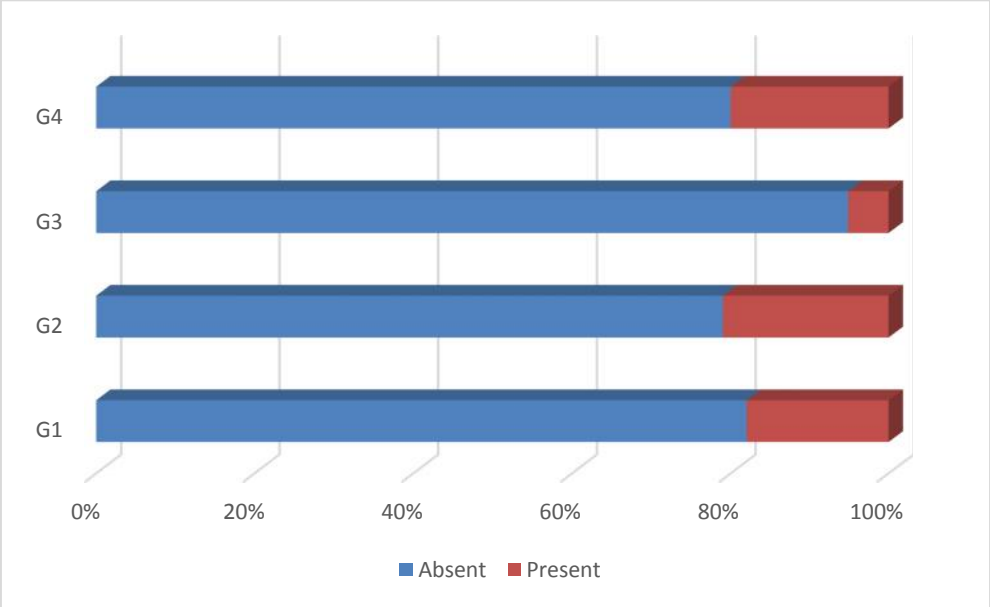


FIGURE.28

Table.28 and Figure.28 show the correlation of chloasma with gravidity.10(18%)in gravida one(G1),4(21%)in gravida two(G2),1(5%)in gravida three(G3) and 2(20%)in gravida four(G4).

CORRELATION OF CHLOASMA WITH PRIMIGRAVIDAE /MULTIGRAVIDAE

(TABLE.29 and FIGURE.29A ,29B)

		CHLOASMA			
		Absent	Present	Total	P value
Multigravida		42(86%)	7(14%)	49	
Primigravida		45(82%)	10(18%)	55	0.24
Total		87	17	104	

TABLE.29

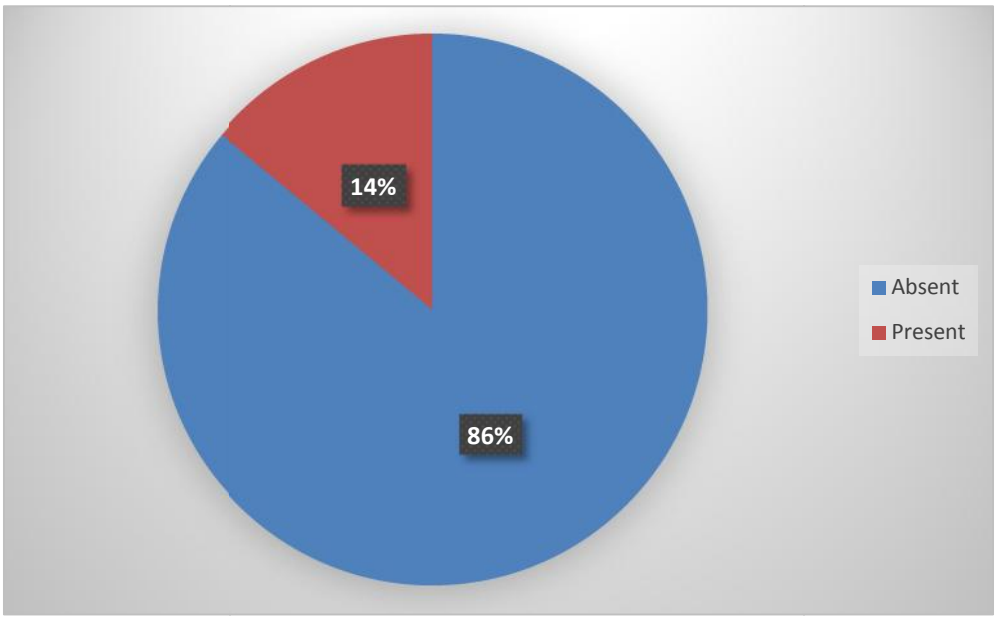


FIGURE.29A

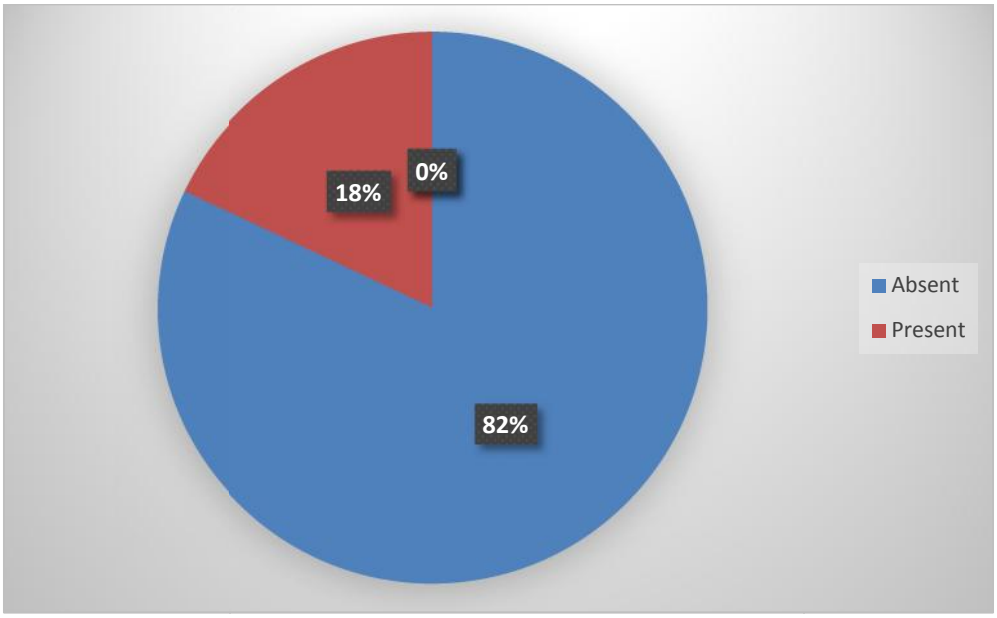


FIGURE.29B

Table.29 and Figure.29A and 29B show the correlation of chloasma with primigravidae/multigravidae.7(14%)multigravidae and 10(18%)primigravidae had chloasma.

CORRELATION OF OEDMA WITH VARIOUS PARAMETERS

CORRELATION OF ODEMA WITH AGE

(TABLE.30 and FIGURE.30)

		OEDEMA			
		Absent	Present	Total	P value
Age <30		82(93%)	6(17%)	88	
Age ≥30		11(68%)	5(31%)	16	8.5
Total		93	11	104	

TABLE.30

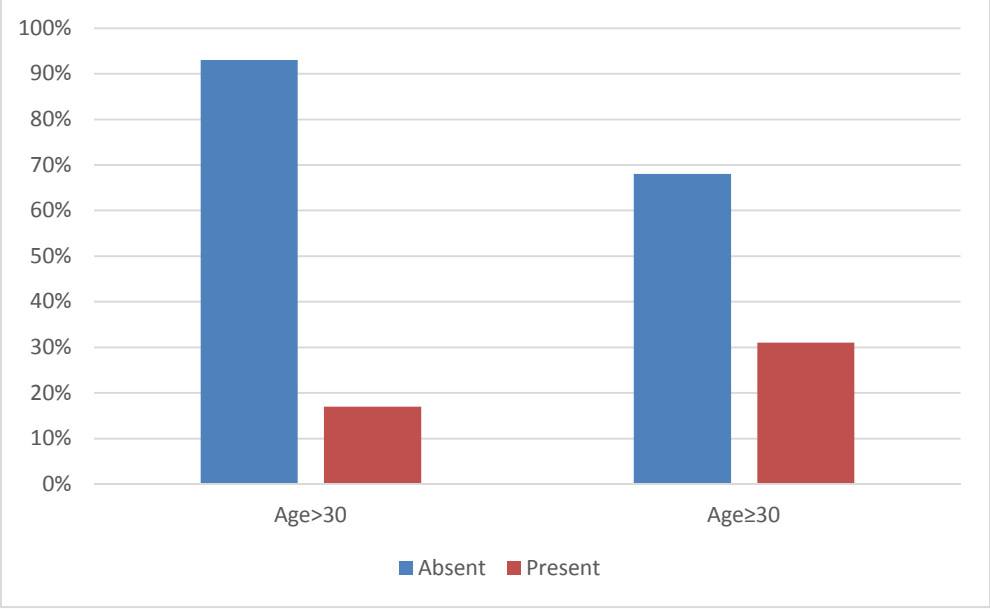


FIGURE.30

Table.30 and Figure.30 shows the correlation of oedma with age.6(17%)women below the age of 30 and 5(31%)30 years and above have oedema.

CORRELATION OF OEDEMA WITH GRAVIDITY

(TABLE.31 and FIGURE.31)

		OEDEMA			
		Absent	Present	Total	P value
Gravida	G1	53(96%)	2(4%)	55	
	G2	15(79%)	4(21%)	19	
	G3	16(80%)	4(20%)	20	6.8
	G4	9(90%)	1(10%)	10	
Total		93	11	104	

TABLE.31

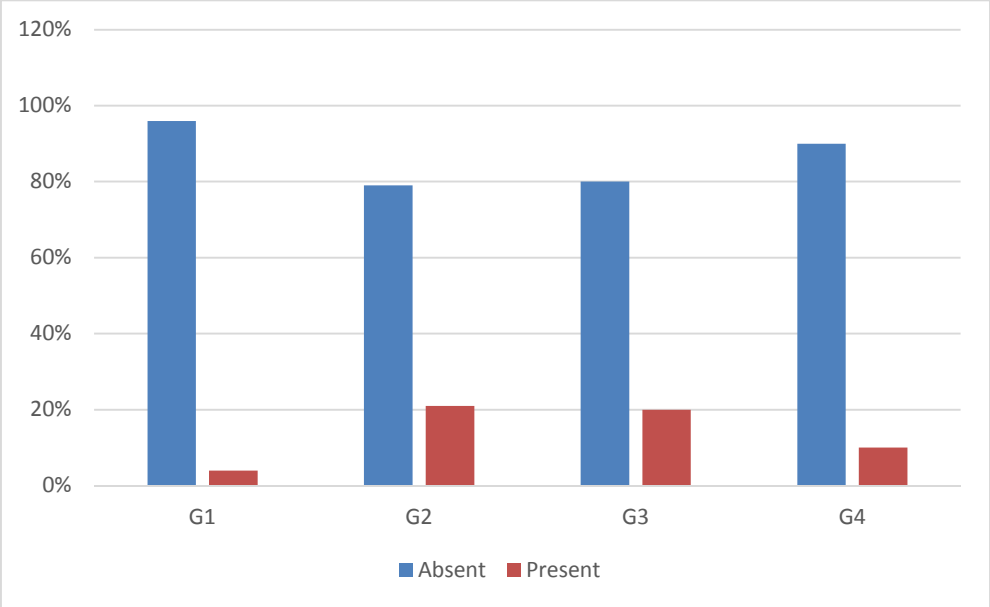


FIGURE.31

Table.31 and Figure.31 shows the correlation of oedema with gravidity.2(4%)women in gravida one(G1),4(21%)in gravida two(G2), 4(20%)in gravida three(G3) and 1(10%)in gravida four(G4) had oedema.

CORRELATION OF OEDEMA WITH PRIMIGRAVIDAE /MULTIGRAVIDAE

(TABLE.32 and FIGURE.32)

		Oedema			
		Absent	Present	Total	P value
Multigravida Primigravidae		40(82%)	9(18%)	49	5.88
		53(96%)	2(4%)	55	
Total		93	11	104	

TABLE.32

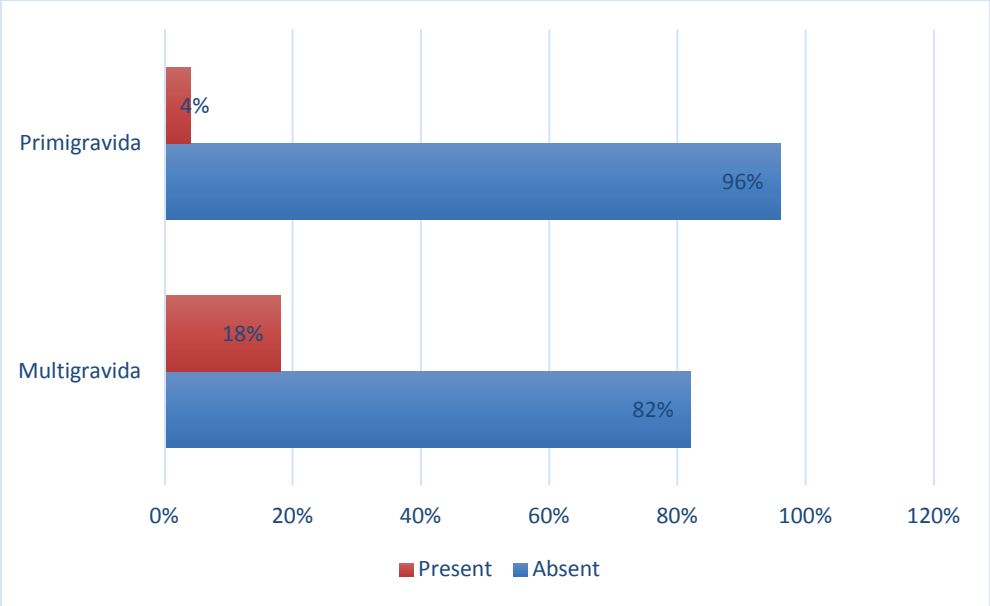


FIGURE.32

Table.32 and Figure.32 shows the correlation of odema with primigravidae/multigravidae.9(18%)multigravidae and 2(4%) primigravidae had odema.

CORRELATION OF OEDEMA WITH TRIMESTER

(TABLE .33 and FIGURE.33)

Trimester		Oedema		Total	P vlaue
		Absent	Present		
Second		63(100%)	0	63	
Third		30(73%)	11(27%)	41	18.72
Total		93	11	104	

TABLE.33

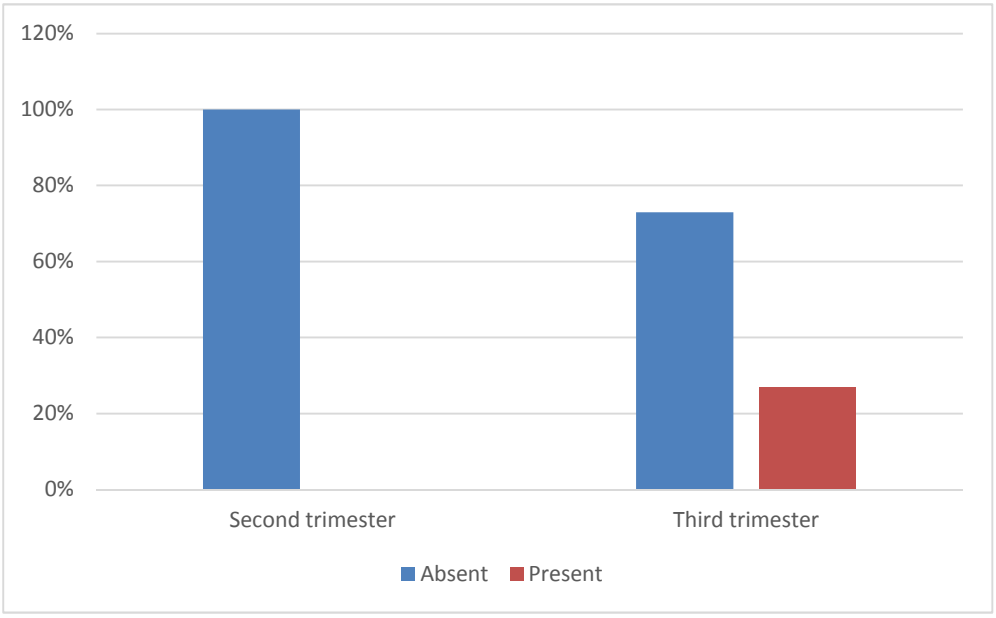


FIGURE.33

Table.33 and Figure.33 shows the correlation of odema with trimester.No pregnamt women in second trimester had odema,11(27%) women in third trimester had oedema.

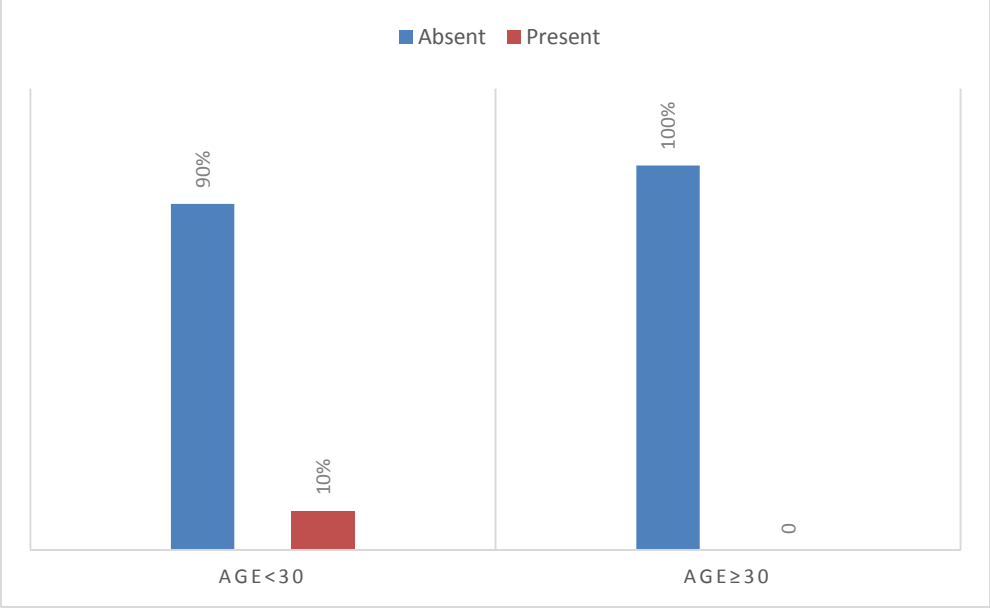
CORRELATION OF VARICOSITIES WITH VARIOUS PARAMETERS.

CORRELATION OF VARIC0SITIES WITH AGE

(TABLE.34 and FIGURE.34)

Age		VARICOSITIES		Total	P value
		Absent	Present		
Age <30		79(90%)	9(10%)	88	
Age≥30		16(100%)	0	16	1.77
Total		95	9	104	

TABLE.34



FIGURRE.34

Table.34 and Figure.34 shows the correlation of varicosities with age.9(10%)pregnant women below the age of 30 had varicosities,no pregnant women of 30 years and above had varicosities.

CORRELATION OF VARICOSITIES WITH GRAVIDITY

(TABLE.35 and FIGURE.35)

		VARICOSITIES			
		Absent	Present	Total	P value
Gravida	G1	53(96%)	2(4%)	55	
	G2	17(90%)	2(10%)	19	4.48
	G3	17(85%)	3(15%)	20	
	G4	8(80%)	2(20%)	10	
Total		95	9	104	

TABLE.35

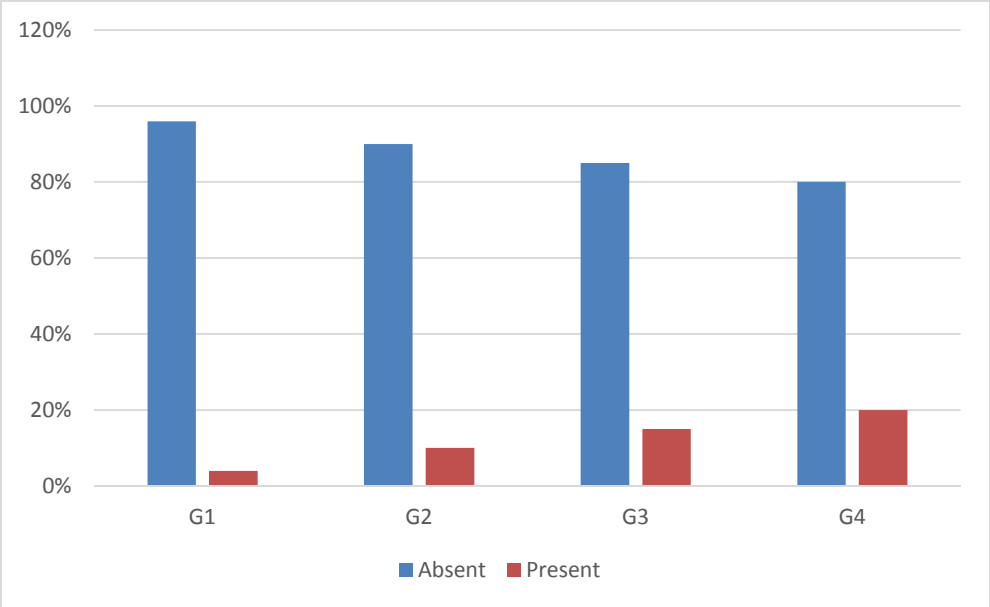


FIGURE.35

Table.35 and Figure.35 shows the correlation of varicosities with gravida.2(4%)in gravida one (G1),2(10%)in gravida two(G2),3(15%)in gravida three(G3) and 2(20%)in gravida four(G4) had varicosities.

CORRELATION OF VARICOSITIES WITH PRIMIGRAVIDAE MULTIGRAVIDAE

(TABLE.36 and FIGURE.36)

		VARICOSITIES			
		Absent	Present	Total	P value
Multigravida		42(86%)	7(14%)	49	
Primigravida		53(96%)	2(4%)	55	3.71
Total		95	9	104	

TABLE.36

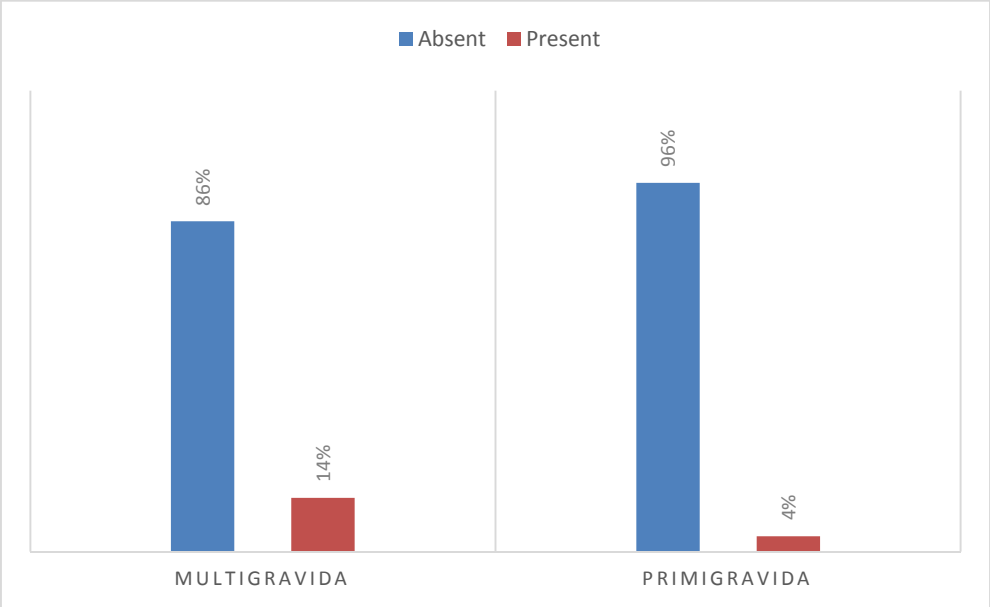


FIGURE.36

Table.36 and Figure.36 shows the correlation of varicosities with primigravidae/multigravidae.7(14%)multigravidae and 2(4%)primigravidae had varicosities.

CORRELATION OF VARICOSITIES WITH TRIMESTER

(TABLE.37 and FIGURE.37)

Trimester		VARICOSITIES		Total	P value
		Absent	Present		
Second		63(100%)	0	63	
Third		32(78%)	9(22%)	41	15.13
Total		95	9	104	

TABLE.37

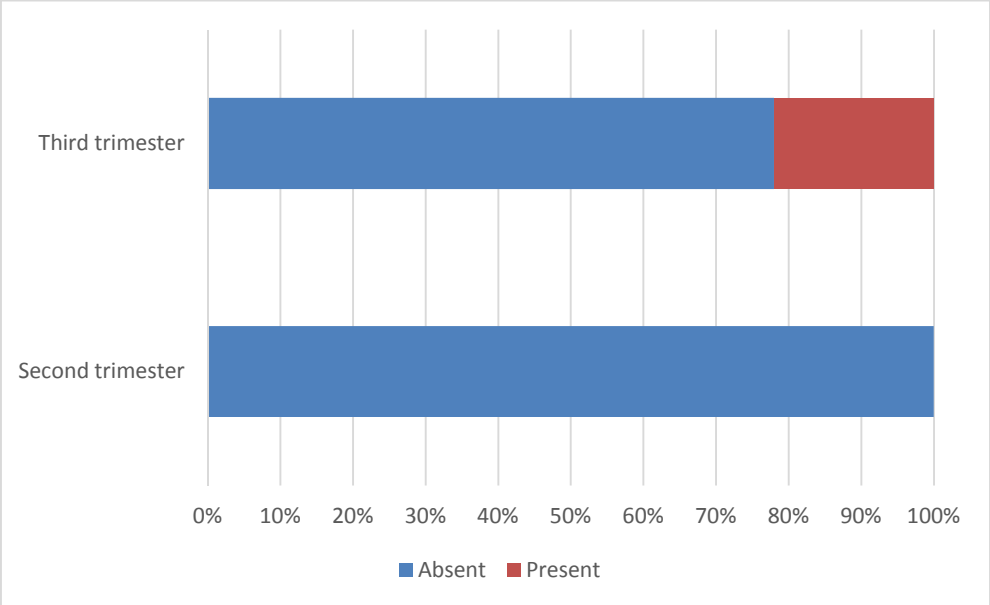


FIGURE.37

Table.37and Figure.37 shows the correlation of varicosities with trimester.No pregnant women in second trimester had any varicosities.9(22%)women in third trimester had varicosities.

CORRELATION OF PALMAR ERYTHEMA WITH VARIOUS PARAMETERS

CORRELATION OF PALMAR ERYTHEMA WITH AGE

(TABLE.38 and FIGURE.38)

Age		Palmar erythema		Total	P value
		Absent	Present		
Age <30		84(95%)	4(5%)	88	
Age ≥30		16(100%)	0	16	0.75
Total		100	4	104	

TABLE.38

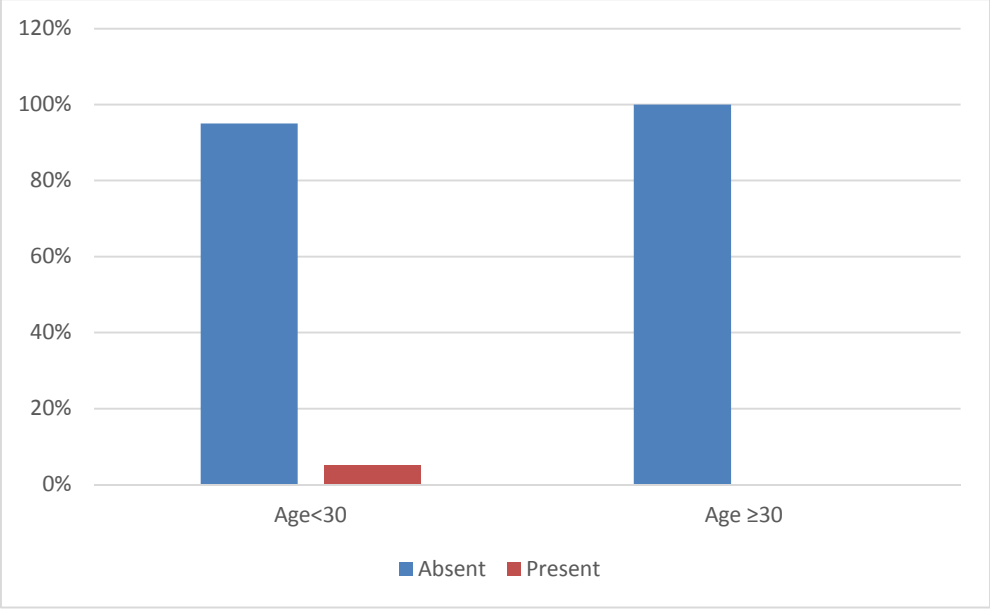


FIGURE.38

Table.38 and Figure.38 shows the co relation of palmar erythema with age.4(5%)pregnanat women belw 30 years had palmar erythema.No pregnant women 30 years and above had palmar erythema.

CORRELATION OF PALMAR ERYTHEMA WITH GRAVIDITY

(TABLE.39 and FIGURE.39)

		PALMAR ERYTHEMA			
		Absent	Present	Total	P value
Gravida	G1	53(96%)	2(4%)	55	
	G2	19(100%)	0	19	
	G3	18(90%)	2(10%)	20	3.71
	G4	10(100%)	0	10	
Total		100	4	104	

TABLE.39

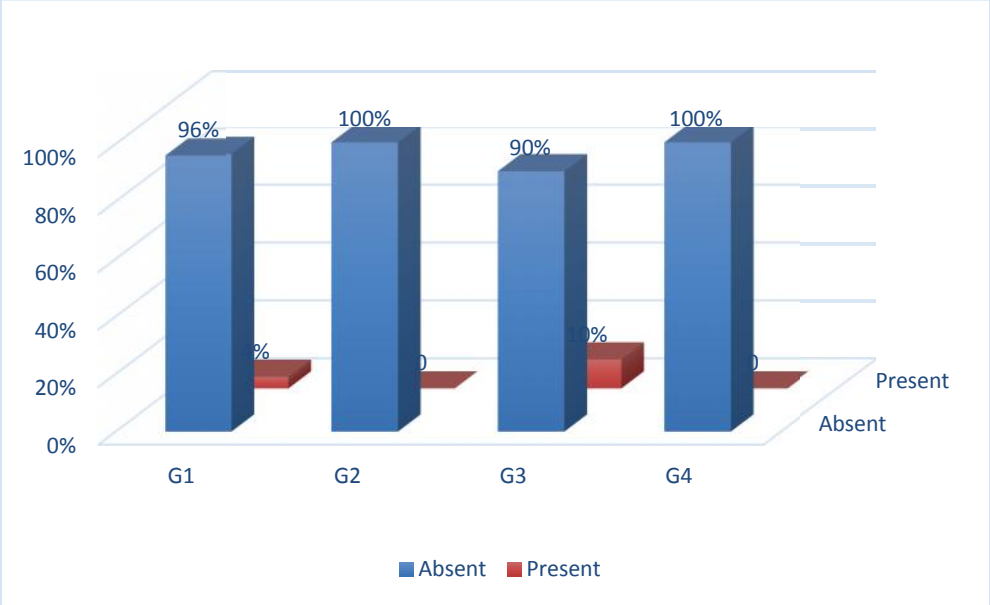


FIGURE.39

Table.39 and Figure.39 shows the correlation of palmar erythema with gravidity.2(4%)in gravida one(G1), no pregnant women with gravida two(G2) and four(G4) had palmar erythema.2(10%)pregnant women with gravida three(G3) had palmar erythema.

CORRELATION OF PALMAR ERYTHEMA WITHPRIMIGRAVIDAE /MULTIGRAVIDAE

(TABLE.40 and FIGURE.40)

		Palmar erythema			
		Absent	Present	Total	P value
Multigravida		47(96%)	2(4%)	49	
Primigravida		53(96%)	2(4%)	55	0.014
Total		100	4	104	

TABLE.40

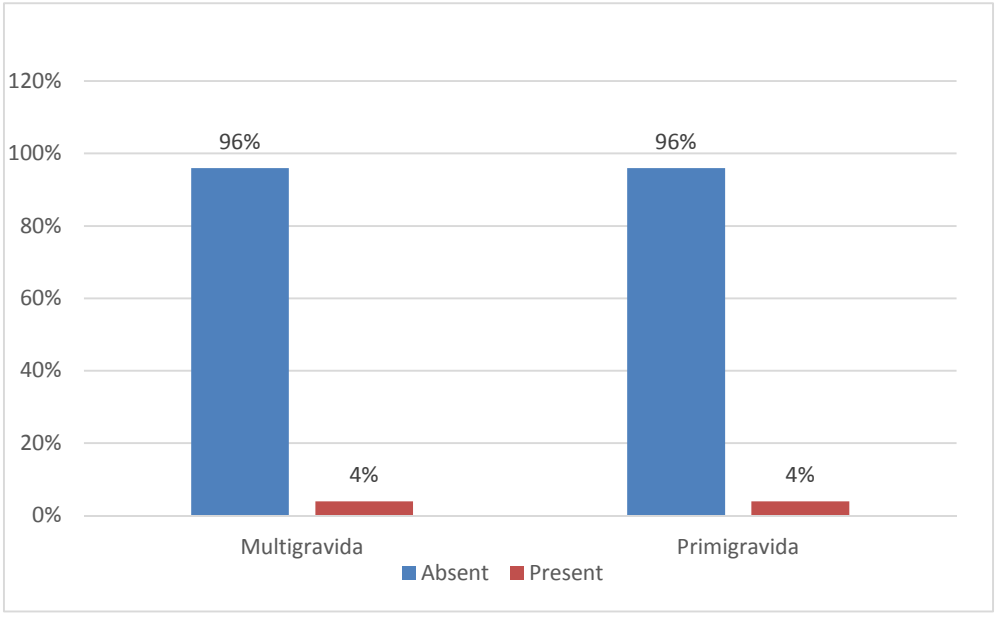


FIGURE.40

Table.40 and Figure.40 shows the correlation of palmar erythema with primigravidae/multigravidae .2(4%)multigravidae and 2(4%)primigravidae had palmar erythema.

CORRELATION OF PALMAR ERYTHEMA WITH TRIMESTER

(TABLE.41and FIGURE.41)

Trimester		PALMAR ERYTHEMA		Total	P value
		Absent	Present		
Second		60(95%)	3(5%)	63	
Third		40(98%)	1(2%)	41	0.35
Total		100	4	104	

TABLE.41

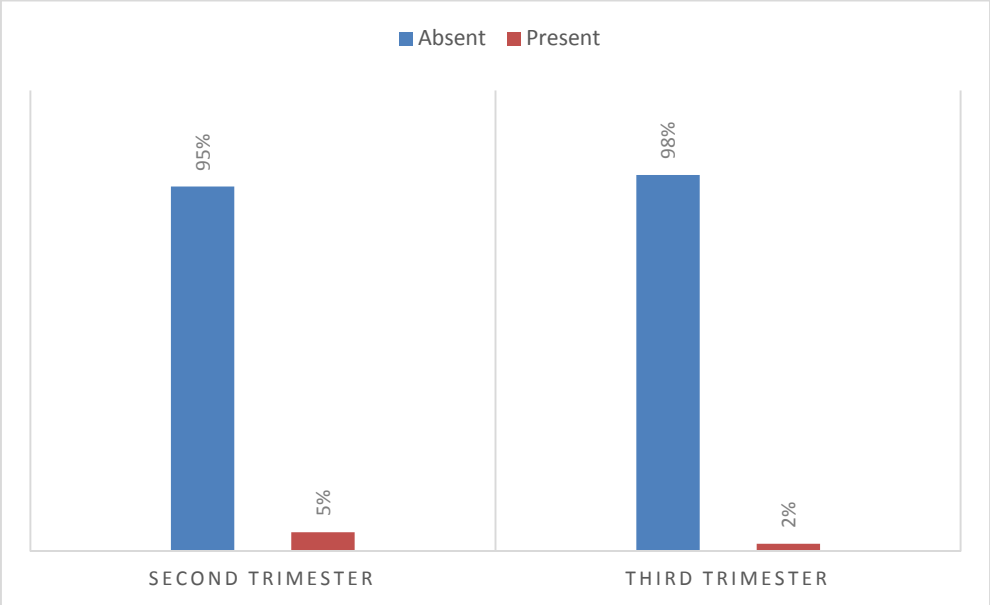


FIGURE.41

Table.41 and Figure.41shows the co relation of palmar erythema with trimester.3(5%)in second trimester and 1(2%) in third trimester had palmar erythema.

CORRELATION OF VARIOUS PARAMETERS WITH PYOGENIC GRANULOMA

CORRELATION OF PYOGENIC GRANULOMA WITH GRAVIDITY

(TABLE.42 and FIGURE.42)

		PYOGENIC GRANULOMA			
		Absent	Present	Total	P value
Gravida	G1	52(95%)	3(5%)	55	
	G2	19(100%)	0	19	2.75
	G3	20(100%)	0	20	
	G4	10(100%)	0	10	
Total		101	3	104	

TABLE.42

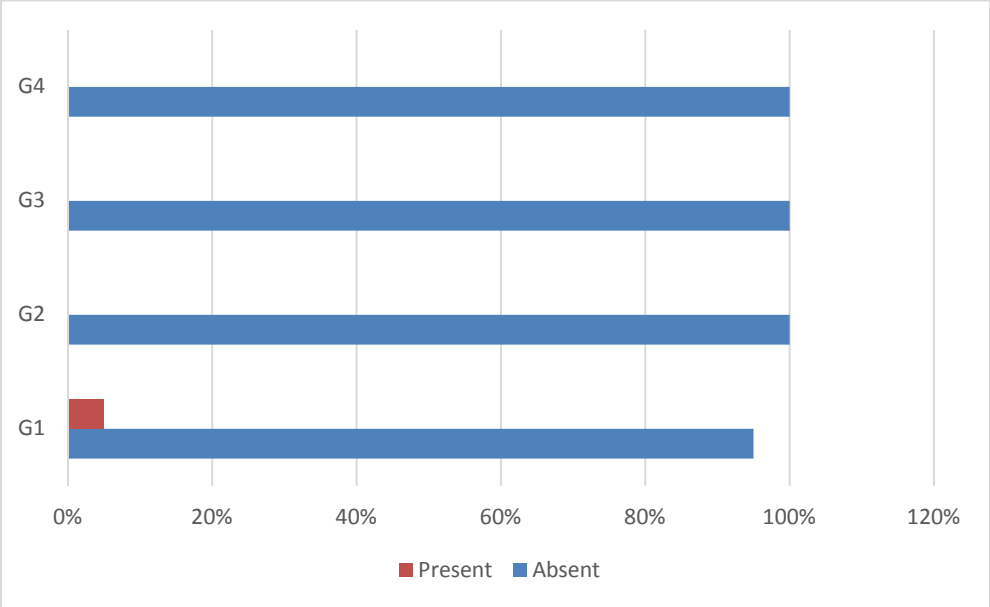


FIGURE.42

Table.42 and Figure.42 shows the correlation of pyogenic granuloma with gravidity.3(5%)in gravida one(G1) had pyogenic granuloma.No pregnant women with gravida two(G2) ,gravida three(G3) and gravida four (G4) had pyogenic granuloma.

CORRELATION OF PYOGENIC GRANULOMA WITH PRIMIGRAVIDAE /MULTIGRAVIDAE.

(TABLE.43 and FIGURE.43)

		PYOGENIC GRANULOMA			
		Absent	Present	Total	P value
Multigravida		49(100%)	0	49	
Primigravida		52(95%)	3(5%)	55	2.75
Total		101	3	104	

TABLE .43

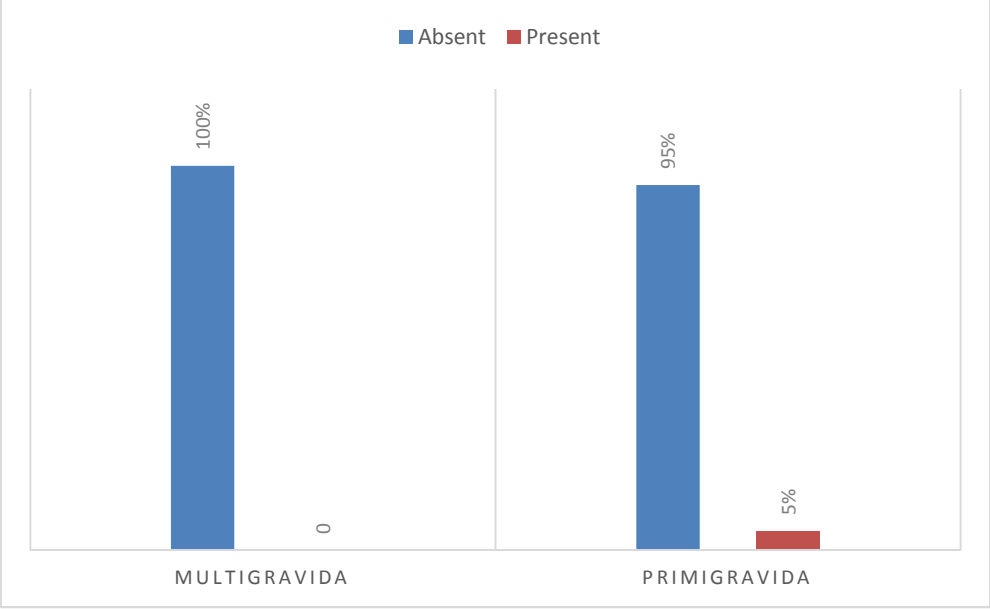


FIGURE.43

Table.43 and Figure.43 shows the correlation of pyogenic granuloma with primigravidae/multigravidae.No multigravidae had pyogenic granuloma 3(5%)primigravidae had pyogenic granuloma.

CORRELATION OF PYOGENIC GRANULOMA WITH TRIMESTER

(TABLE .44 and FIGURE.44)

Trimester		PYOGENIC GRANULOMA		Total	
		Absent	Present		P value
Second		61(97%)	2(3%)	63	
Third		40(98%)	1(2%)	41	0.048
Total		101	3	104	

TABLE.44

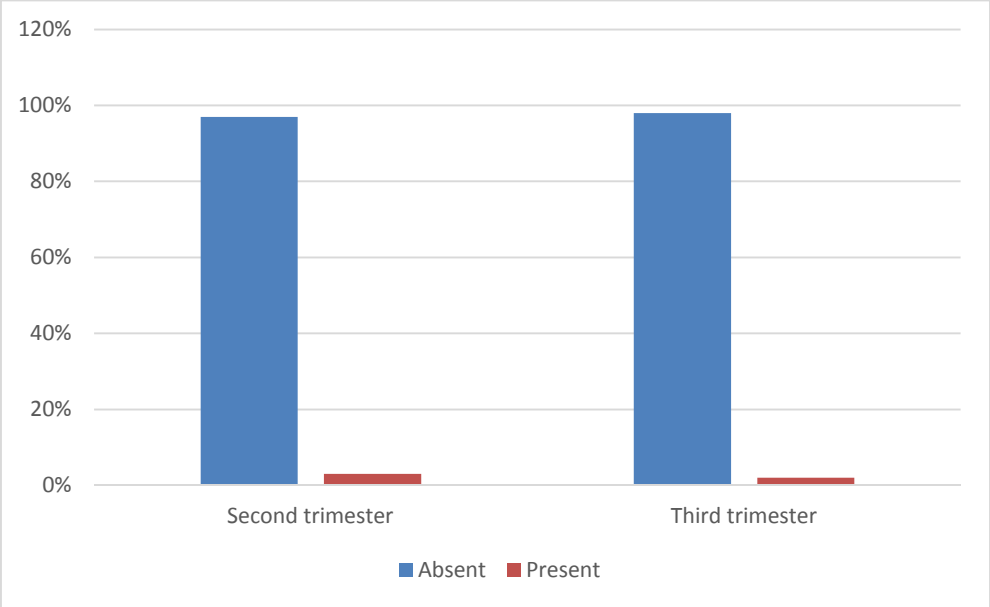


FIGURE.44

Table.44 and Figure.44 shows the correlation of pyogenic granuloma with trimester.2(3%)multigravidae and 1(2%)primigravidae had pyogenic granuloma.

CORRELATIONS OF GINGIVAL HYPERPLASIA WITH VARIOUS PARAMETERS

CORRELATION OF GINGIVAL HYPERPLASIA WITH GRAVIDITY

(TABLE.45 and FIGURE.45)

		GINGIVAL HYPERPLASIA		Total	P value
		Absent	Present		
Gravida	G1	54(98%)	1(2%)	55	
	G2	19(100%)	0	19	
	G3	20(100%)	0	20	0.90
	G4	10(100%)	0	10	
Total		103	1	104	

TABLE.45

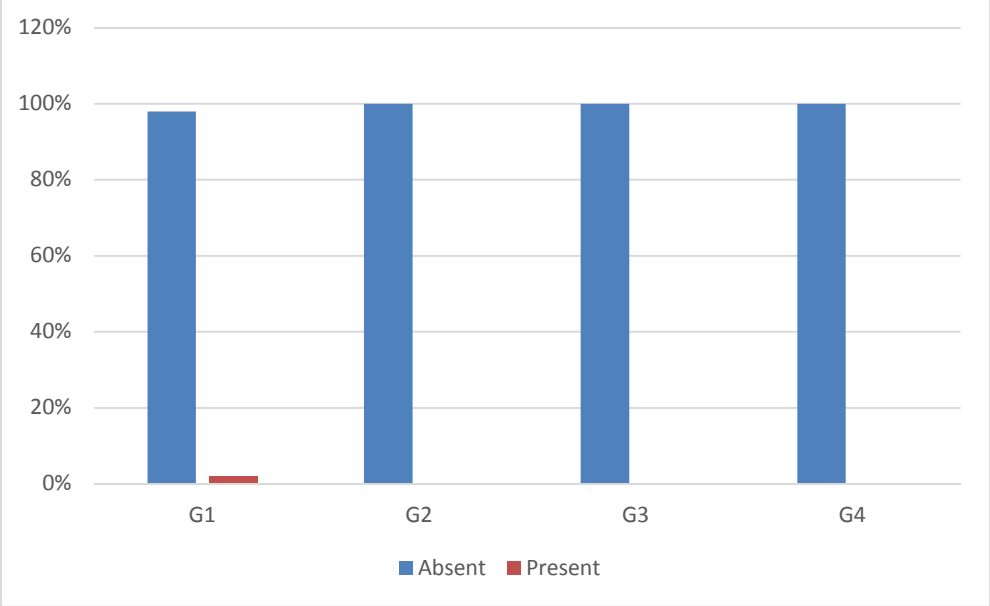


FIGURE .45

Table.45 and Figure.45 shows the correlation of gingival hyperplasia with gravidity.1(2%) pregnant woman with gravida one(G1) had gingival hyperplasia.No women with gravida two(G2),gravida three(G3) and gravida four(G4) had gingival hyperplasia.

CORRELATION OF GINGIVAL HYPERPLASIA WITH PRIMIGRAVIDAE / MULTIGRAVIDAE.

(TABLE.46 and FIGURE.46)

	GINGIVAL HYPERPLASIA			
	Absent	Present	Total	P value
Multigravida	49(100%)	0	49	
Primigravida	54(98%)	1(2%)	55	0.90
Total	103	1	104	

TABLE.46

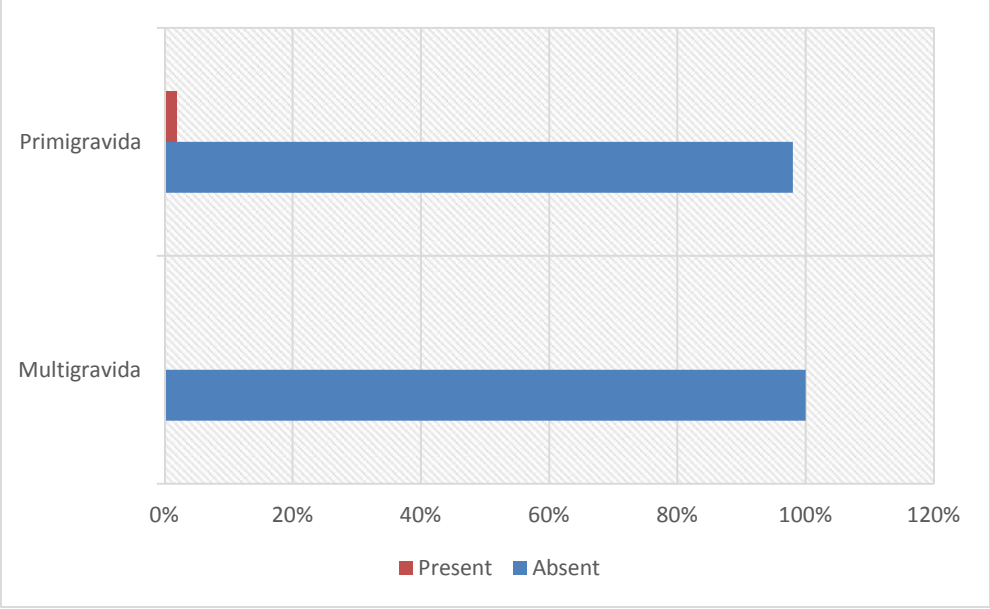


FIGURE.46

Table.46 and Figure.46 shows the correlation of gingival hyperplasia with primigravidae/ multigravidae. No multigravidae had gingival hyperplasia.1 (2%) primigravidae had gingival hyperplasia.

CORRELATION OF GINGIVAL HYPERPLASIA WITH TRIMESTER

(TABLE.47 and FIGURE .47)

Trimester		GINGIVAL HYPERPLASIA		Total	
		Absent	Present		P value
Second		62(98%)	1(2%)	63	
Third		41(100%)	0	41	0.65
Total		103	1	104	

TABLE .47

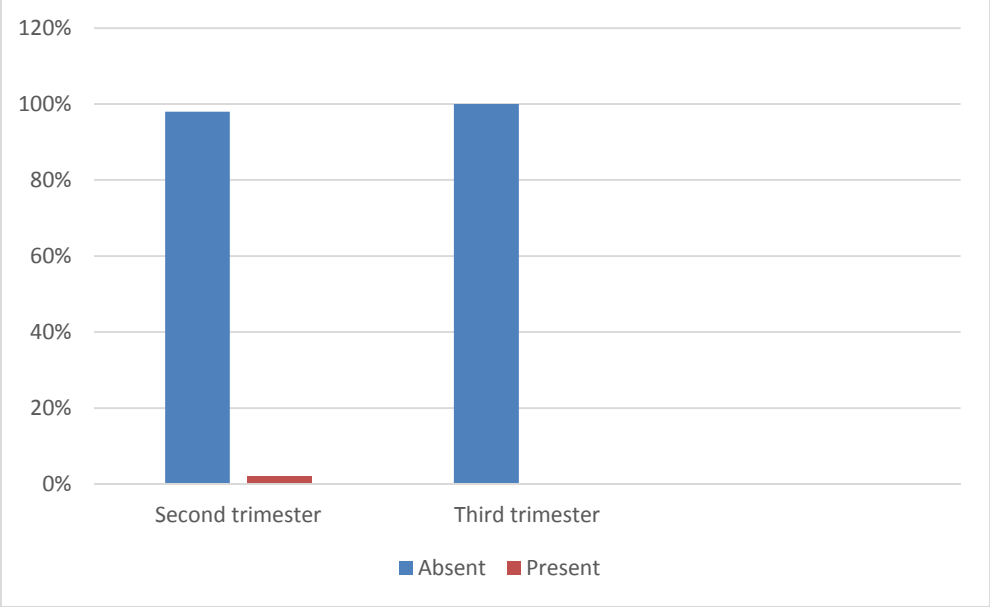


FIGURE.47

Table.47 and Figure.47 shows the correlation of gingival hyperplasia with trimester.1(2%)women in second trimester had gingival hyperplasia, no women in third trimester had gingival hyperplasia.

CORRELATION OF HIRSUTISM WITH VARIOUS PARAMETERS

CORRELATION OF HIRSUTISM WITH AGE

(TABLE.48 and FIGURE.48)

Age		HIRSUTISM		Total	
		Absent	Present		P value
Age <30		85(97%)	3(3%)	88	
Age ≥30		16(100%)	0	16	0.56
Total		101	3	104	

TABLE.48

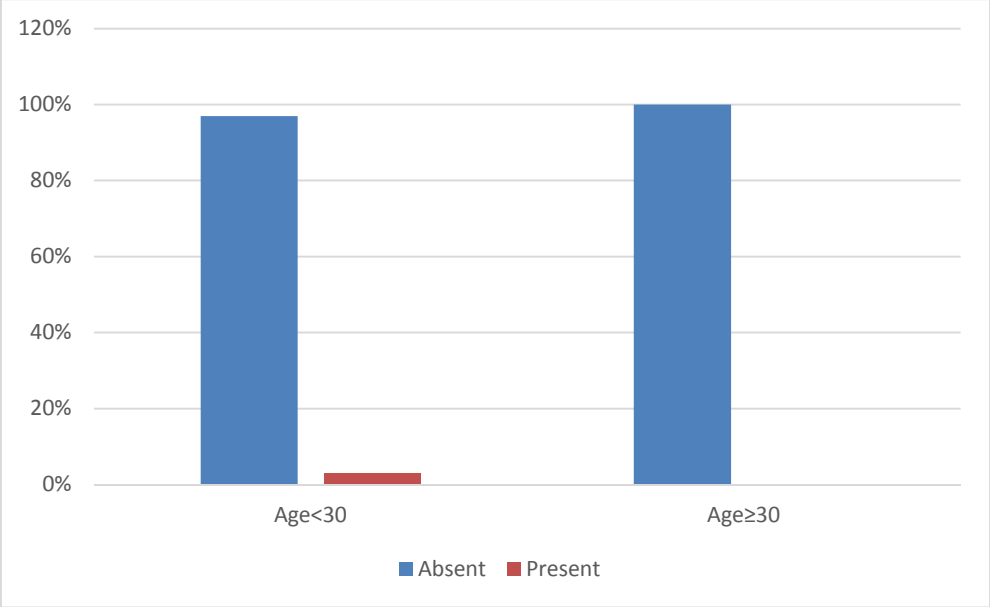


FIGURE.48

Table.48 and Figure.48 shows the correlation of hirsutism with age, 3(3%) pregnant women below the age of 30 had hirsutism and no women of 30 years and above had hirsutism.

CORRELATION OF HIRSUTISM WITH GRAVIDITY

(TABLE.49 and FIGURE.49)

		HIRSUTISM		Total	P value
		Absent	Present		
Gravida	G1	53(96%)	2(4%)	55	
	G2	18(95%)	1(5%)	19	1.38
	G3	20(100%)	0	20	
	G4	10(100%)	0	10	
Total		101	3	104	

TABLE.49

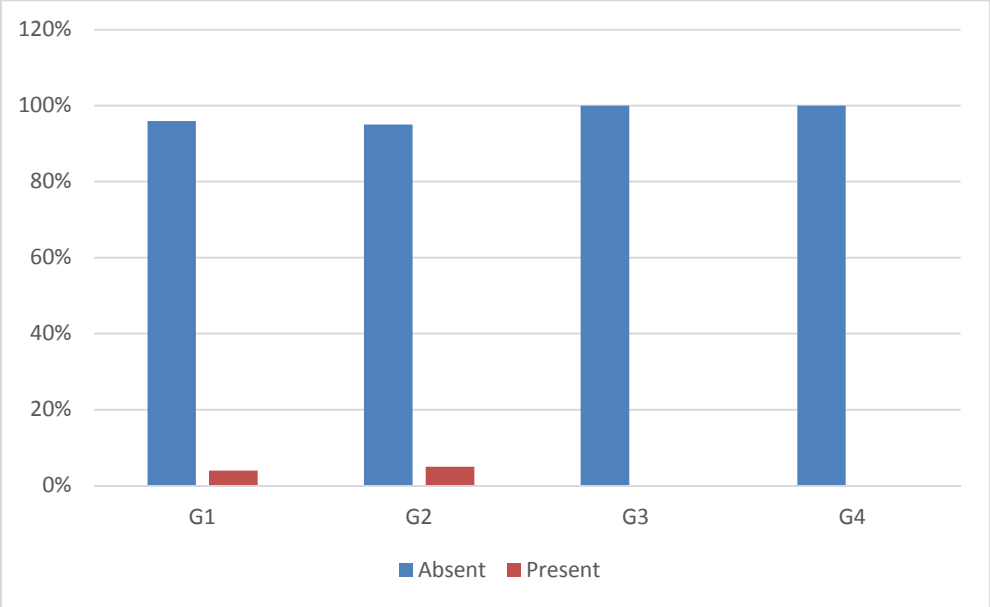


FIGURE.49

Table.49 and Figure.49 shows the correlation of hirsutism with gravidity.2 (4%)women gravida one (G),1(5%)in gravida two(G2) had hirsutism.No women in gravida three(G3) and gravida four(G4) had hirsutism.

CORRELATION OF HIRSUTISM WITH PRIMIGRAVIDAE /MULTIGRAVIDAE

(TABLE.50 and FIGURE.50)

		HIRSUTISM			
		Absent	Present	Total	P value
Multigravida Primigravidae		48(98%)	1(2%)	49	
		53(96%)	2(4%)	55	0.23
Total		101	3	104	

TABLE .50

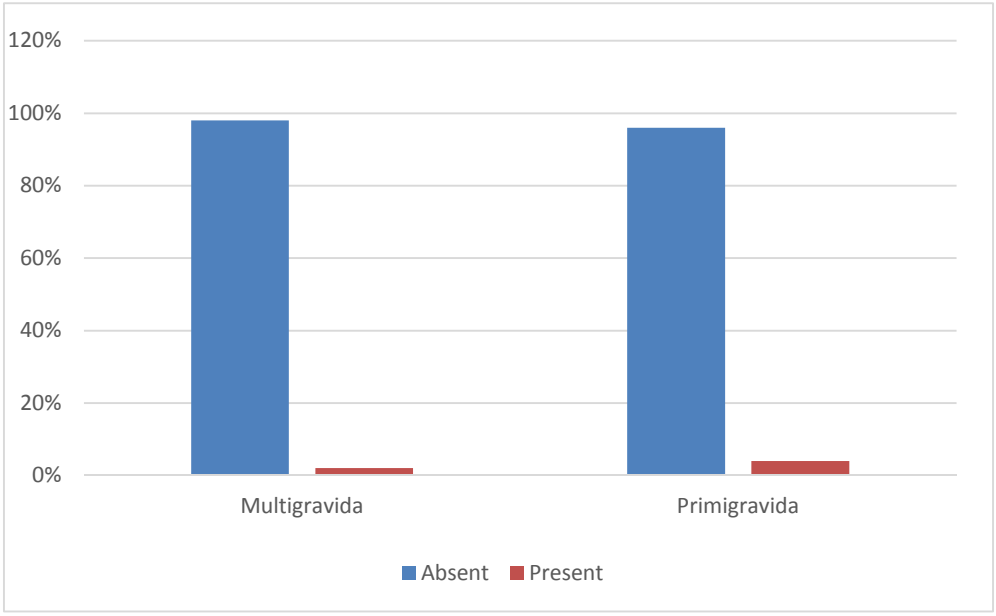


TABLE.50

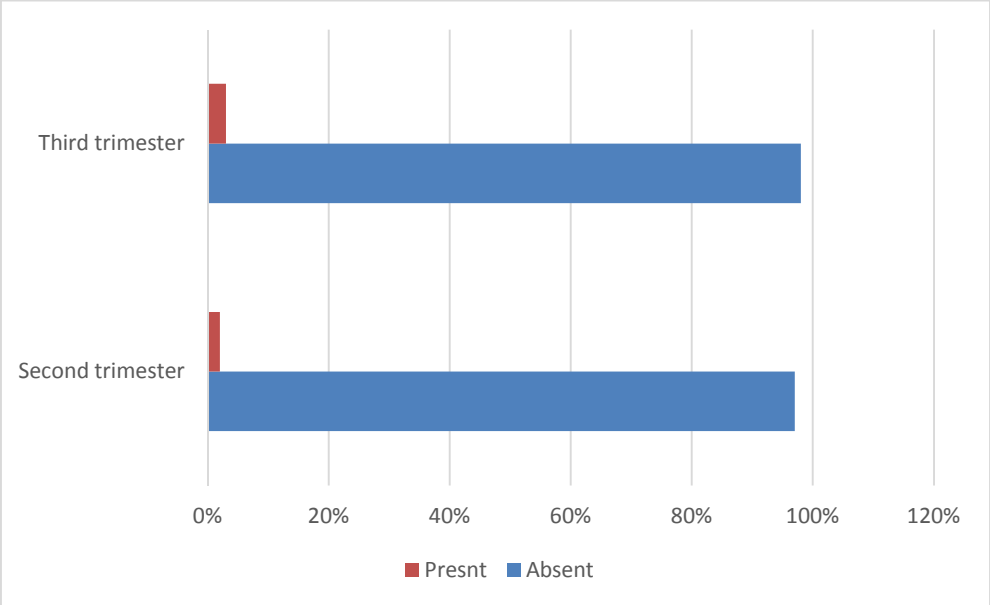
Table.50 and Figure.50 shows the correlation of hirsutism with primigravidae/multigravidae.1(2%)multigravidas and 2(4%)primigravidas had hirsutism.

CORRELATION OF HIRSUTISM WITH TRIMESTER

(TABLE.51 and FIGURE.51)

Trimester		HIRSUTISM		Total	
		Absent	Present		P value
Second		61(97%)	2(3%)	63	
Third		40(98%)	1(2%)	41	0.04
Total		101	3	104	

TABLE.51



FIGURURE.51

Table.51 and Figure.51 shows the correlation of hirsutism with trimester.2(3%)of women in second trimester and 1(2%)in third trimester had hirsutism.

CORRELATION OF SUBUNGUAL HYPERKERATOSES WITH VARIOUS PARAMETERS

CORRELATION OF SUBUNGUAL HYPERKERATOSES WITH AGE

(TABLE.52 and FIGURE.52)

Age		SUBUNGUAL HYPERKERATOSIS		Total	
		Absent	Present		P value
Age <30		83(94%)	5(6%)	88	
Age ≥30		15(94%)	1(6%)	16	0.008
Total		98	6	104	

TABLE.52

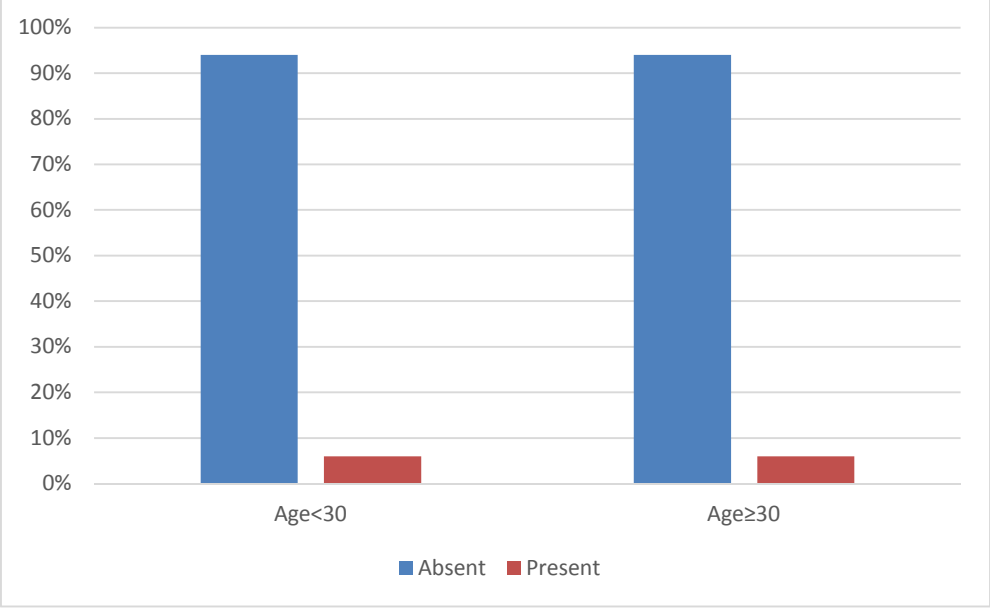


FIGURE.52

Table.52 and Figure.52 show the correlation of subungual hyperkeratoses with age.5 (6%)women below 30 years and 1(6%)30 years and above had subungual hyperkeratoses.

CORRELATION OF SUBUNGUAL HYPERKERATOSES WITH PRIMIGRAVIDAE /MULTIGRAVIDAE.

(TABLE.53 and FIGURE.53)

		SUBUNGUAL HYPERKERATOSIS		Total	
		Absent	Present		P value
Multigravida Primigravidae		46(94%)	3(6%)	49	0.021
		52(95%)	3(5%)	55	
Total		98	6	104	

TABLE.53

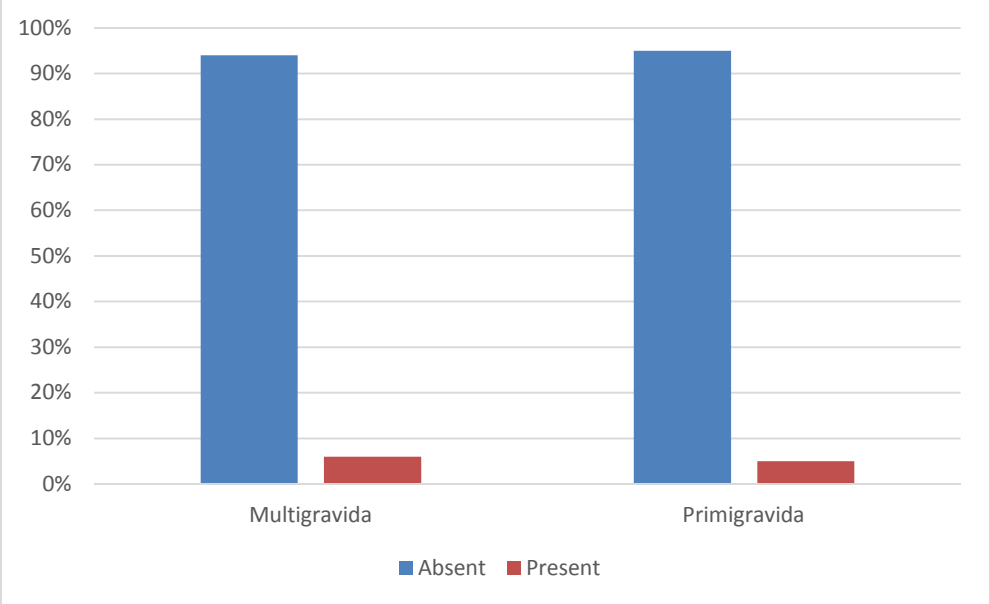


FIGURE.53

Table.53 and Figure.53 shows the correlation of subungual hyperkeratoses with primigravidae /multigravidae.3(6%)multigravidas and 3(5%)primigravidas had subungual hyperkeratoses.

CORRELATION OF SUBUNGUAL HYPERKERATOSES WITH TRIMESTER

(TABLE.54 and FIGURE.54)

Trimester		SUBUNGUAL HYPERKERATOSIS		Total	
		Absent	Present		P value
Second		57(90%)	6(10%)	63	
Third		41(100%)	0	41	4.14
Total		98	6	104	

TABLE.54

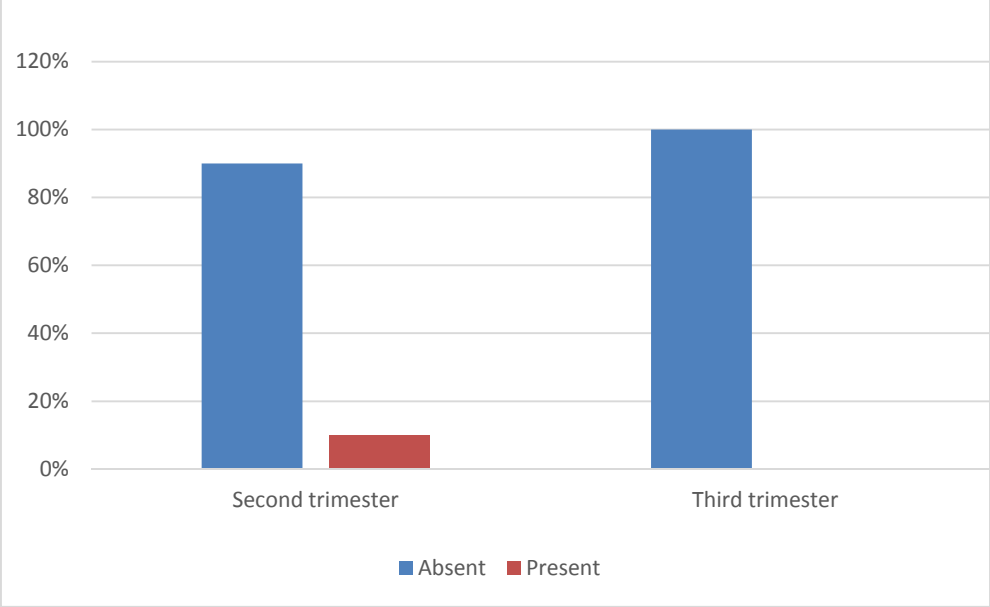


FIGURE.54

Table.54 and Figure.54 shows the correlation of subungual hyperkeratoses with trimester.6(10%) in second trimester had subungual hyperkeratoses.No women in third trimester had subungual hyperkeratoses.

CORRELATION OF VARIOUS PARAMETERS WITH DISTAL ONYCHOLYSIS

CORRELAION OF DISTAL ONYCHOLYSIS WITH AGE

(TABLE.55 and FIGURE.55)

		DISTAL ONYCHOLYSIS			
		Absent	Present	Total	P value
Age <30		86(98%)	2(2%)	88	
Age ≥30		16(100%)	0	16	0.37
Total		102	2	104	

TABLE.55

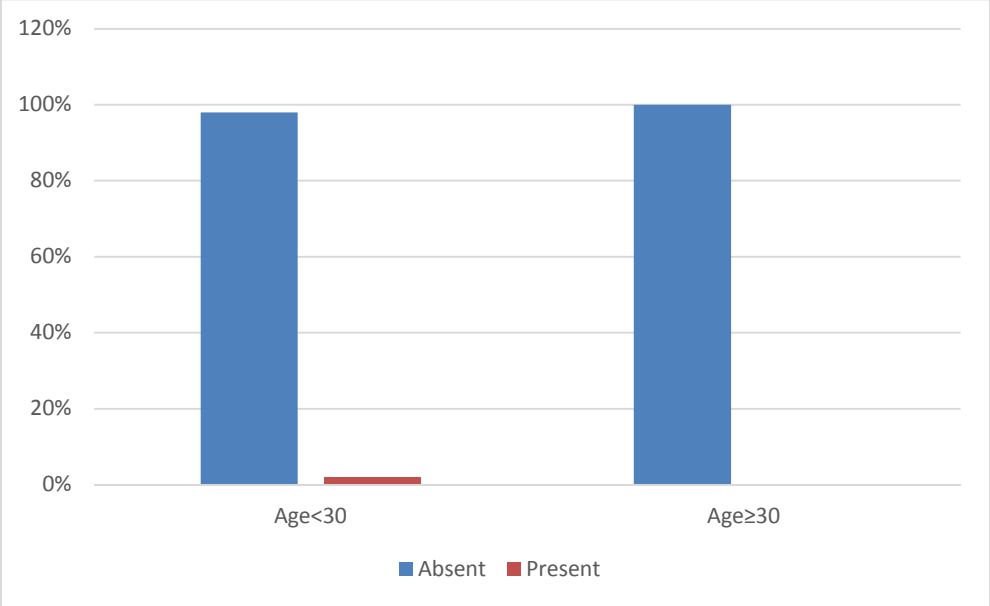


FIGURE.55

Table.55 and Figure.55 shows the correlation of distal onycholysis with age.2(2%) below the age of 30 and no patient 30 years and above had distal onycholysis.

CORRELATION OF DISTAL ONYCHOLYSIS WITH GRAVIDITY

(TABLE.56 and FIGURE.56)

		DISTAL ONYCHOLYSIS			
		Absent	Present	Total	P value
Gravida	G1	54(98%)	1(2%)	55	
	G2	19(100%)	0	19	
	G3	19(95%)	1(5%)	20	1.57
	G4	10(100%)	0	10	
Total		102	2	104	

TABLE.56

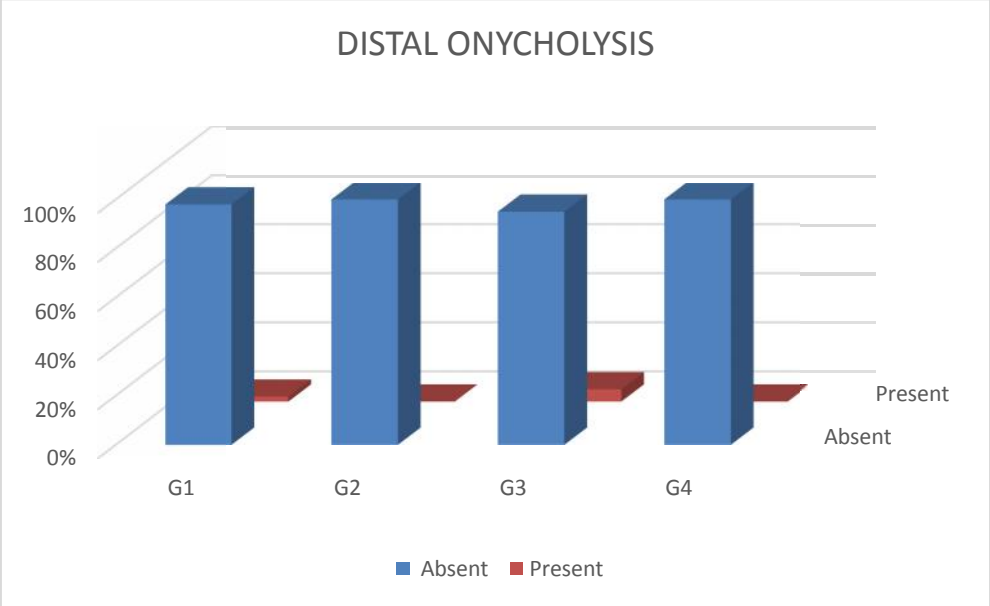


FIGURE.56

Table.56 and Figure.56 shows the correlation of distal onycholysis with gravidity.1(2%)in gravida one(G1),1(5%) in gravida three(G3) had distal onycholysis.No women with gravida two(G2) and gravida four(G4) had distal onycholysis.

CORRELATION OF DISTAL ONYCHOLYSIS WITH PRIMIGRAVIDAE/ MULTIGRAVIDAE

(TABLE.57 and FIGURE.57)

		DISTAL ONYCHOLYSIS			
		Absent	Present	Total	P value
Multigravida		48(98%)	1(2%)	49	
Primigravidae		54(98%)	1(2%)	55	0.007
Total		102	2	104	

TABLE.57

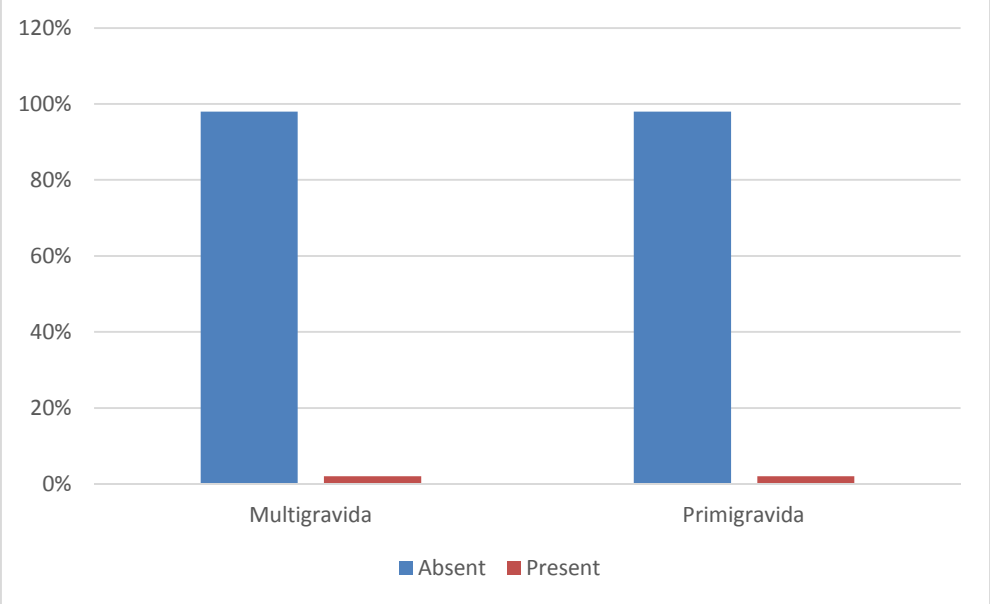


TABLE.57

Table.57 and Figure.57 shows the co relation of distal onycholysis with primigravidae/multigravidae.1(2%)multigravidas and 1(2%)primigravidas had distal onycholysis.

CORRELATION OF PRURITUS OF PREGNANCY WITH VARIOUS PARAMETERS

CORRELATION OF PRURITUS OF PREGNANCY WITH AGE

(TABLE .58 and FIGURE.58)

Age		PRURITUS OF PREGNANCY		Total	
		Absent	Present		P value
Age <30		86(98%)	2(2%)	88	
Age ≥30		14(88%)	2(12%)	16	3.82
Total		100	4	104	

TABLE.58

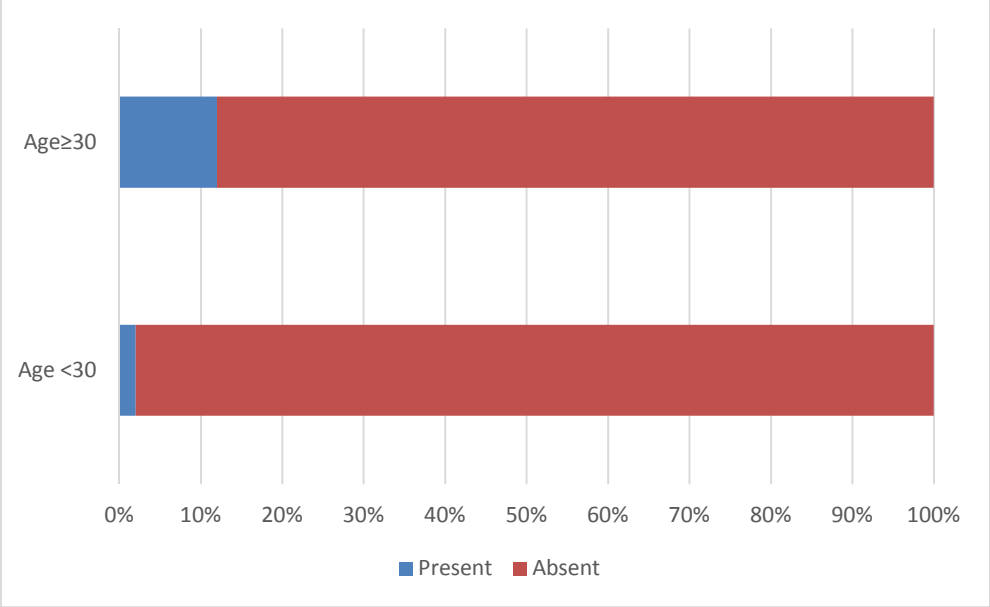


FIGURE.58

Table.58 and Figure.58 shows the correlation of pruritus of pregnancy with age.2(2%) below the age of 30 and 2(12%) 30 years and above had pruritus of pregnancy.

CORRELATION OF PRURITUS OF PREGNANCY WITH GRAVIDITY

(TABLE.59 and FIGURE.59)

		PRURITUS OF PREGNANCY			
		Absent	Present	Total	P value
Gravida	G1	53(96%)	2(4%)	55	
	G2	18(95%)	1(5%)	19	1.93
	G3	20(100%)	0	20	
	G4	9(90%)	1(10%)	10	
Total		100	4	104	

TABLE .59

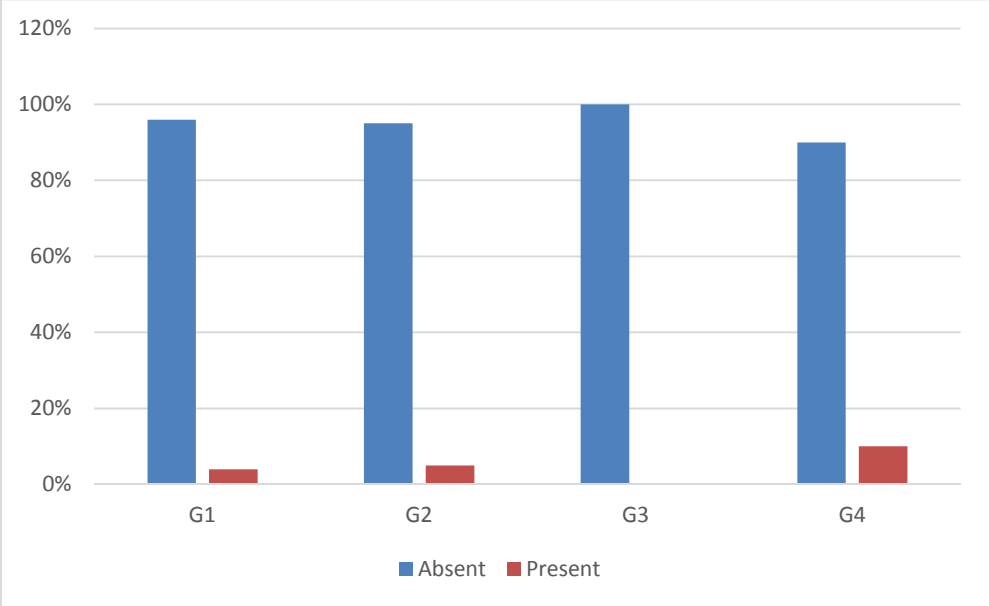


FIGURE.59

Table.59 and Figure.59 shows the co relation of pruritus of pregnancy with gravidity. 2(4%) in gravida one (G1), 1(5%)in gravida two(G2),1 (10%)in gravida four(G4) had pruritus of pregnancy.No women in gravida three(G3) had pruritus of pregnancy.

CORRELATION OF PRURITUS OF PREGNANCY WITH PRIMIGRAVIDAE /MULTIGRAVIDAE

(TABLE.60 and FIGURE.60)

		PRURITUS OF PREGNANCY			
		Absent	Present	Total	P value
Multigravida		47(96%)	2(4%)	49	
Primigravidae		53(96%)	2(4%)	55	0.014
Total		100	4	104	

TABLE.60

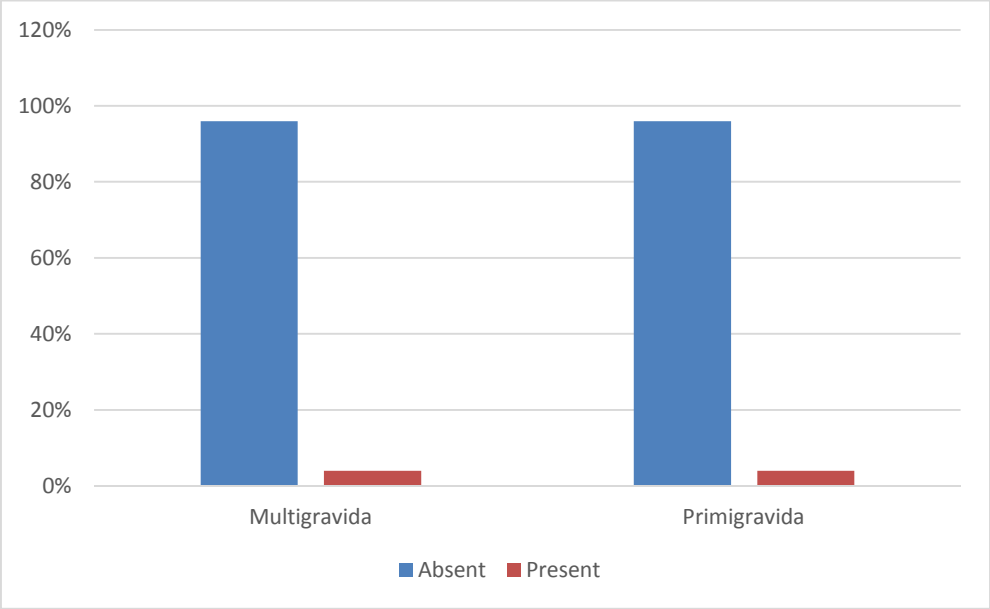


FIGURE.60

Table.60 and Figure.60 shows the correlation of pruritus with pregnancy with primigravidae/multigravidae.2(4%) multigravidas and (4%) primigravidas had pruritus of pregnancy.

CORRELATION OF PRURITUS OF PREGNANCY WITH TRIMESTER

(TABLE .61 and FIGURE.61)

Trimester		PRURITUS OF PREGNANCY		Total	
		Absent	Present		P value
Second		60(95%)	3(5%)	63	
Third		40(98%)	1(2%)	41	0.36
Total		100	4	104	

TABLE.61

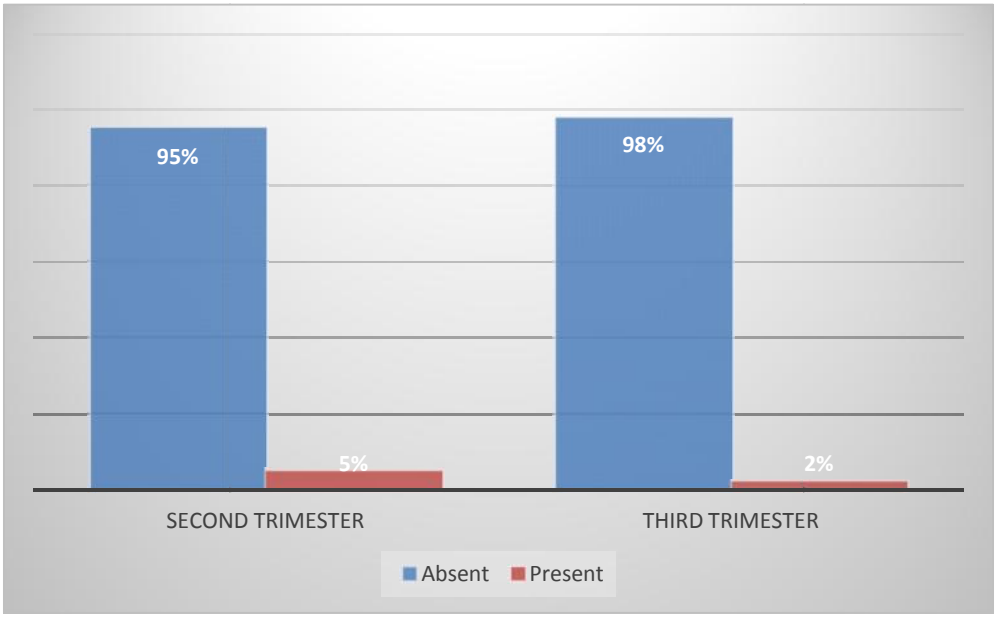


FIGURE.61

Table.61 and Figure.61 shows the correlation of pruritus of pregnancy with trimester.3(5%)in second trimester and 1(2%)in third trimester had pruritus of pregnancy.

CORRELATION OF ECZEMA IN PREGNANCY WITH VARIOUS PARAMETERS

CORRELATION OF ECZEMA IN PREGNANCY WITH AGE

(TABLE.62 and FIGURE.62)

Age		ECZEMA IN PREGNANCY		Total	
		Absent	Present		P value
Age <30		85(98%)	2(2%)	87	
Age≥30		14(88%)	2(12%)	16	3.76
Total		99	4	103	

TABLE.62

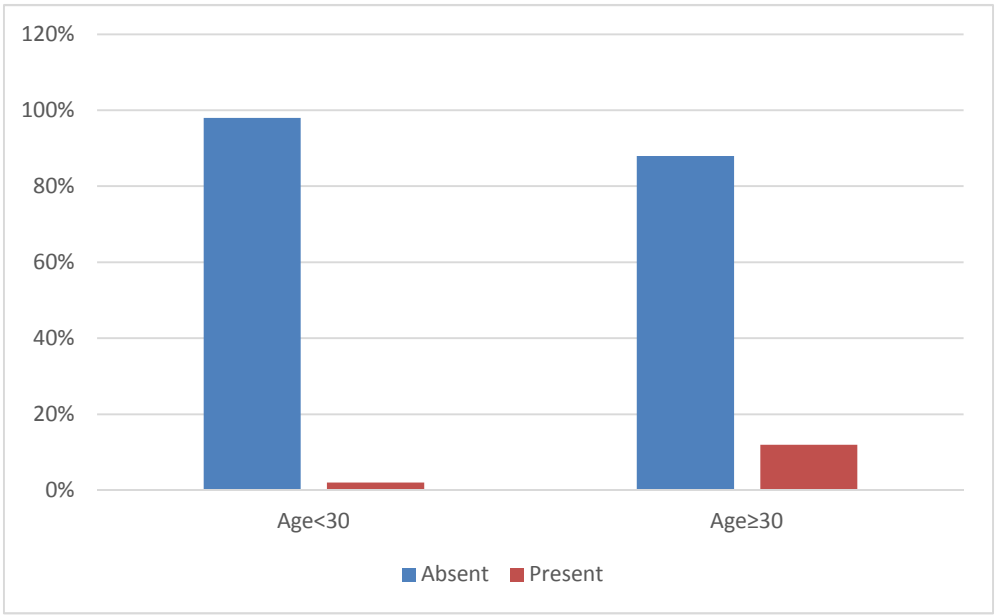


FIGURE.62

Table.62 and Figure.62 shows the correlation of eczema in pregnancy with age.2(2%) below 30 years and 2(12%) 30 years and above had eczema in pregnancy.

CORRELATION OF ECZEMA IN PREGNANCY WITH GRAVIDITY

(TABLE.63 and FIGURE.63)

		ECZEMA IN PREGNANCY			
		Absent	Present	Total	P value
Gravida	G1	52(96%)	2(4%)	54	
	G2	19(100%)	0	19	1.84
	G3	19(95%)	1(5%)	20	
	G4	9(90%)	1(10%)	10	
Total		99	4	103	

TABLE.63

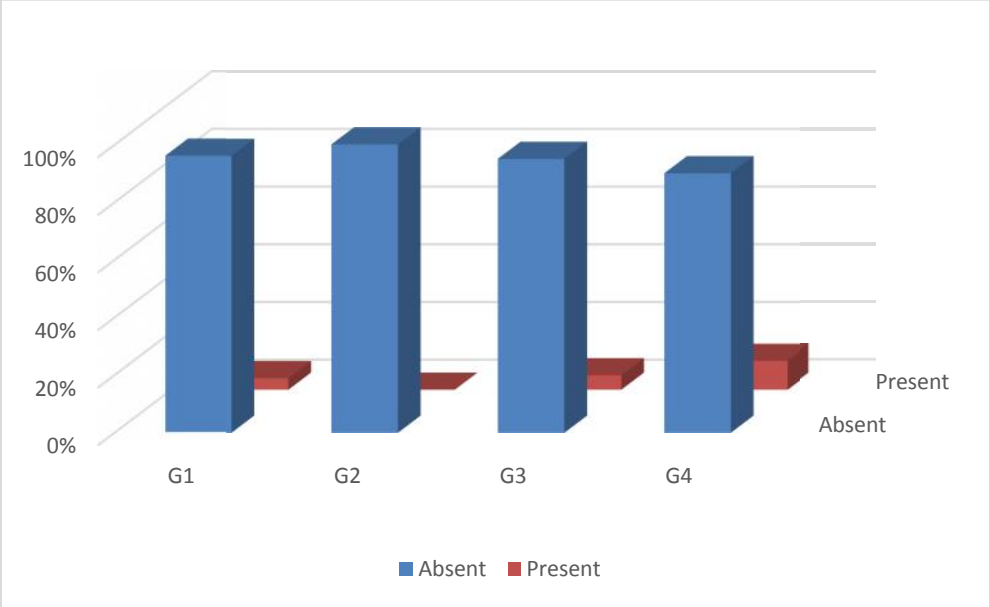


FIGURE.63

Table.63 and Figure.63 shows the correlation of eczema in pregnancy with gravidity.2(4%)in gravida one(G1) ,1(5%)in gravida three(G3) ,1(10%)in gravida four (G4) and no patient in gravida two(G2) had eczema in pregnancy.

CORRELATION OF ECZEMA IN PREGNANCY WITH PRIMIGRAVIDAE /MULTIGRAVIDAE.

(TABLE.64 and FIGURE.64)

	ECZEMA IN PREGNANCY			
	Absent	Present	Total	P value
Multigravida	47(96%)	2(4%)	49	
Primigravida	52(96%)	2(4%)	54	0.01
Total	99	4	103	

TABLE.64

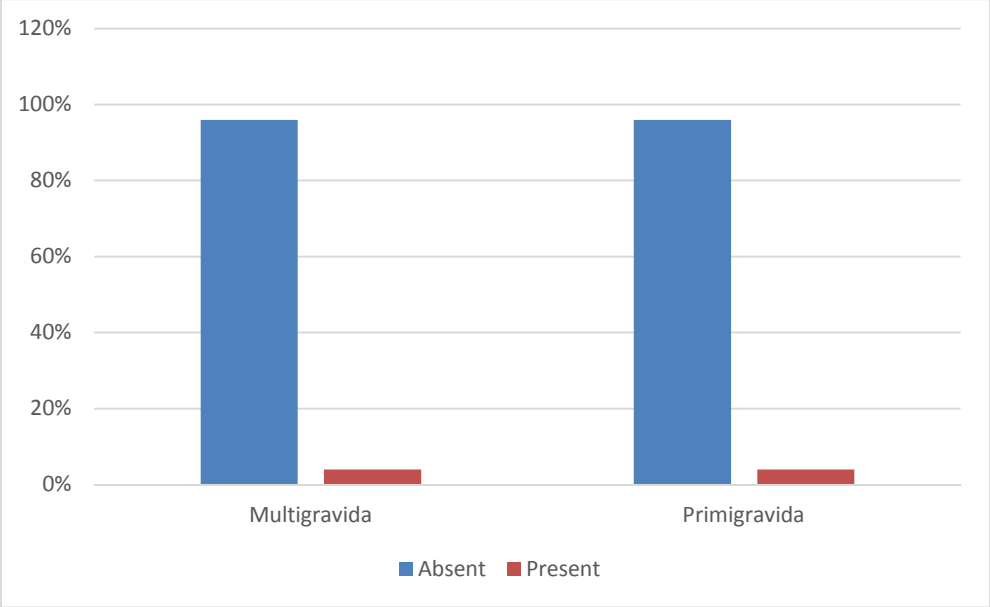


FIGURE.64

Table.64 and Figure.64 shows the correlation of eczema in pregnancy with primigravidae/multigravidae.2(4%)multigravidas and 2(4%)primigravidas had eczema in pregnancy.

CORRELATION OF ECZEMA IN PREGNANCY WITH TRIMESTER

(TABLE.65 and FIGURE.65)

Trimester		ECZEMA IN PREGNANCY		Total	P value
		Absent	Present		
Second		58(94%)	4(6%)	62	
Third		41(100%)	0	41	2.75
Total		99	4	103	

TABLE.65

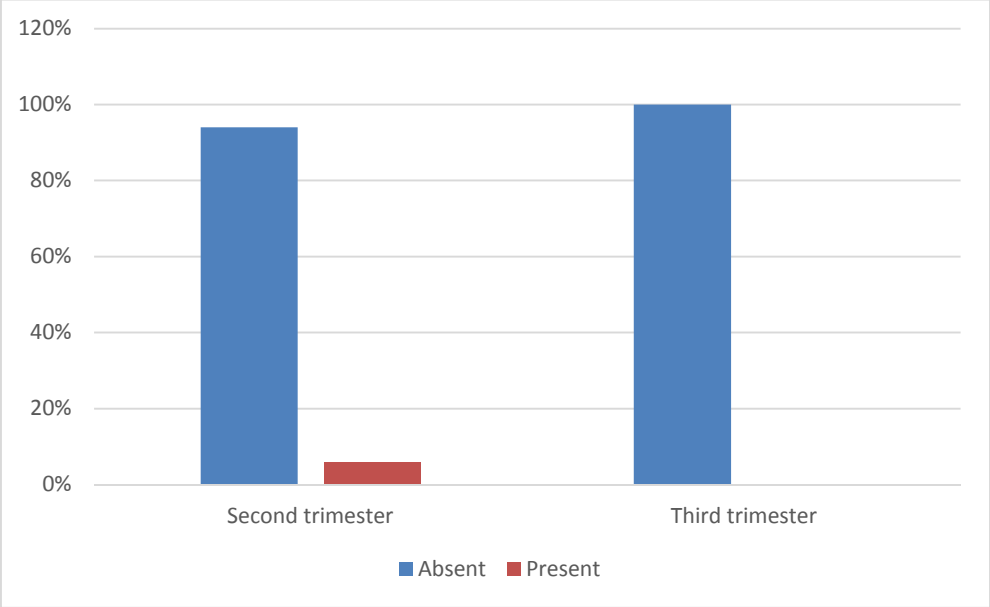


FIGURE.65

Table.65 and Figure.65 shows the correlation of eczema in pregnancy with trimester.4(6%) in second trimester had eczema in pregnancy.No women in our study had eczema in third trimester.

CORRELATION OF PRURITIC FOLLICULITIS OF PREGNANCY WITH VARIOUS PARAMETERS

CORRELATION OF PRURITIC FOLLICULITIS OF PREGNANCY WITH AGE

(TABLE.66 and FIGURE.66)

Age	PRURITIC FOLLICULITIS OF PREGNANCY		Total	P value
	Absent	Present		
Age <30	86(98%)	2(2%)	88	
Age ≥30	16(100%)	0	16	0.37
Total	102	2	104	

TABLE.66

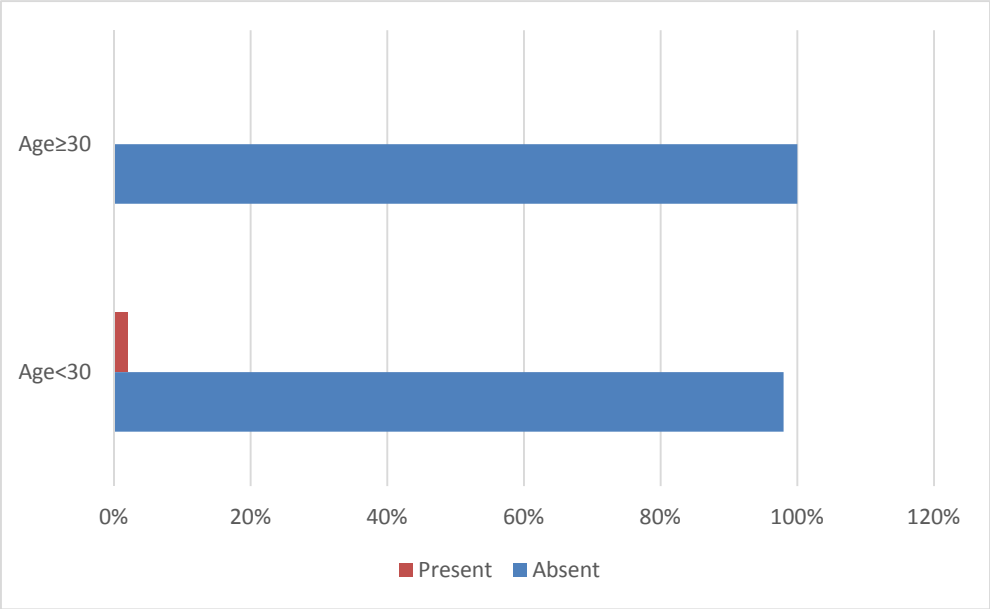


FIGURE.66

Table.66 and Figure.66 shows the correlation of pruritic folliculitis of pregnancy with age.2(2%) below the age of 30 and no patient 30 years and above had pruritic folliculitis of pregnancy.

CORRELATION OF PRURITIC FOLLICULITIS OF PREGNANCY WITHGRAVIDITY

(TABLE.67 and FIGURE.67)

		PRURITIC FOLLICULITIS OF PREGNANCY		Total	P value
		Absent	Present		
Gravida	G1	54(98%)	1(2%)	55	
	G2	19(100%)	0	19	1.57
	G3	19(95%)	1(5%)	20	
	G4	10(100%)	0	10	
Total		102	2	104	

TABLE.67

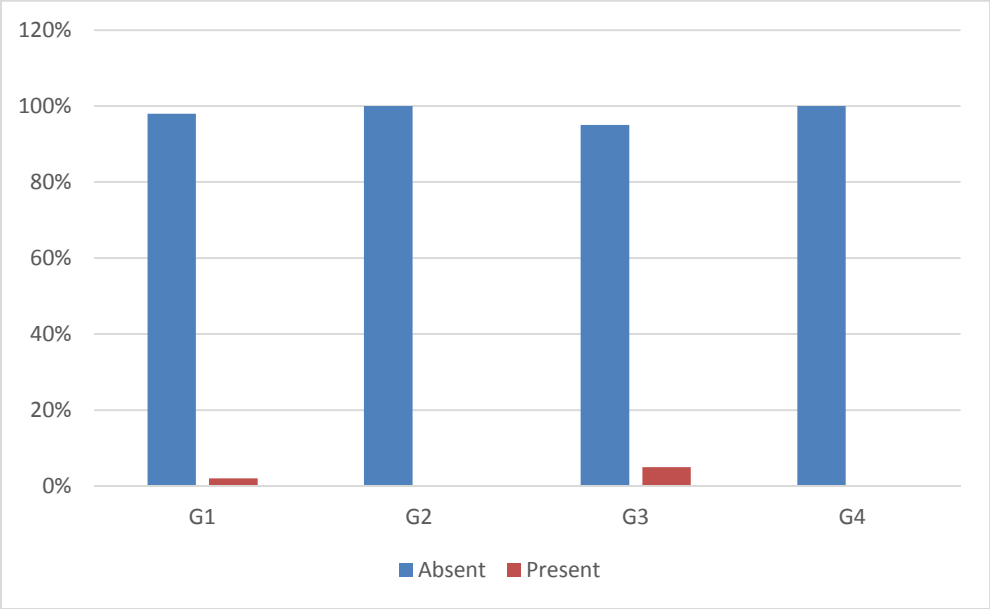


FIGURE.67

Table.67 and Figure.67 shows the correlation of pruritic folliculitis of pregnancy with gravidity.1(2%)in gravida one(G1) and 1(5%)in gravida three(G3) had pruritic folliculitis of pregnancy.No women with gravida two(G2) and gravida (G4) had pruritic folliculitis of pregnancy.

CORRELATION OF PRURITIC FOLLICULITIS OF PREGNANCY WITH PRIMIGRAVIDAE/MULTIGRAVIDAE.

(TABLE.68 and FIGURE.68)

		PFP			
		Absent	Present	Total	P value
Multigravida		48(98%)	1(2%)	49	
Primigravidae		54(98%)	1(2%)	55	0.007
Total		102	2	104	

TABLE.68

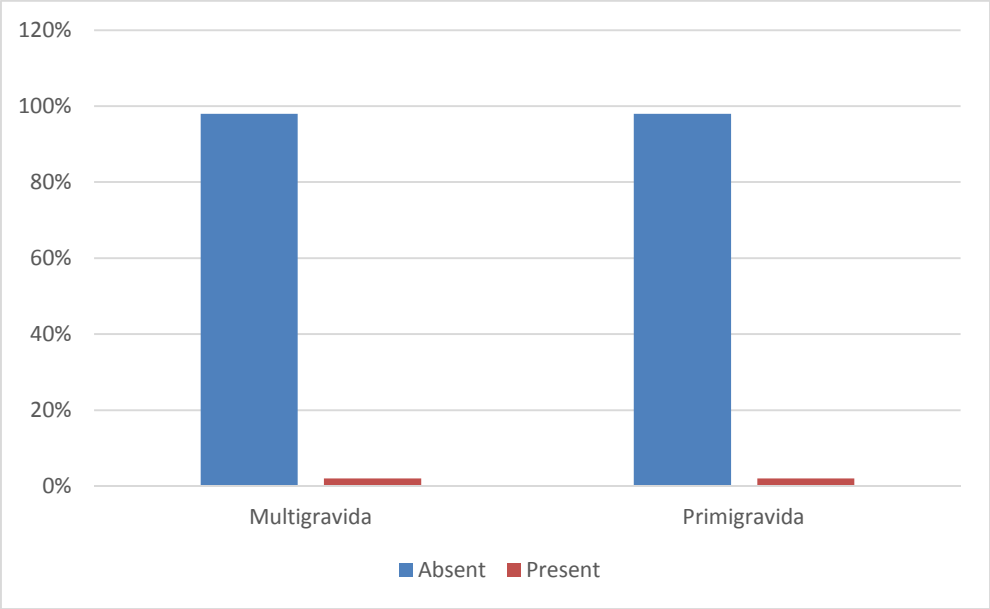


FIGURE.68

Table.68 and Figure.68 shows the correlation of pruritic folliculitis of pregnancy with primigravidae/multigravidae.1(2%)multigravida and 1(2%) primigravida had pruritic folliculitis of pregnancy.

CORRELATION OF PRURITIC FOLLICULITIS OF PREGNANCY WITH TRIMESTER

(TABLE .69 and FIGURE.69)

Trimester		PRURITIC FOLLICULITIS OF PREGNANCY		Total	
		Absent	Present		P value
Second		62(98%)	1(2%)	63	
Third		40(98%)	1(2%)	41	0.096
Total		102	2	104	

TABLE.69

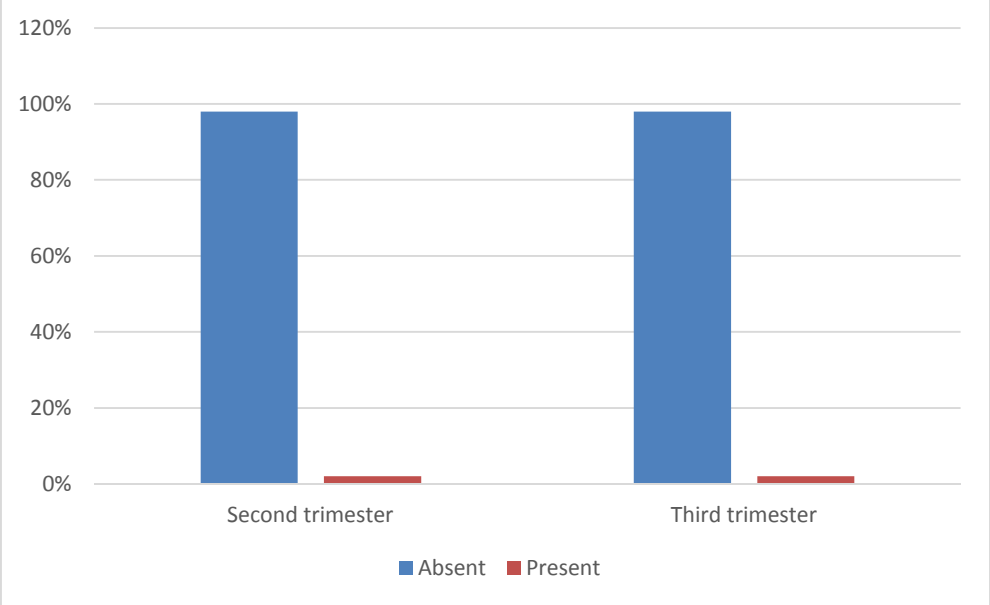


FIGURE.69

Table.69 and Figure.69 shows the correlation of pruritic folliculitis f pregnancy with trimester.1(2%) in second trimester and 1(2%) in third trimester had eczema in pregnancy.

CORRELATION OF PRURIGO OF PREGNANCY WITH VARIOUS PARAMETERS

CORRELATION OF PRURIGO OF PREGNANCY WITH AGE

(TABLE.70 and FIGURE .70)

Age		PRURIGO OF PREGNANCY		Total	
		Absent	Present		P value
Age <30		80(91%)	8(9%)	88	
Age ≥30		15(94%)	1(6%)	16	0.13
Total		95	9	104	

TABLE.70

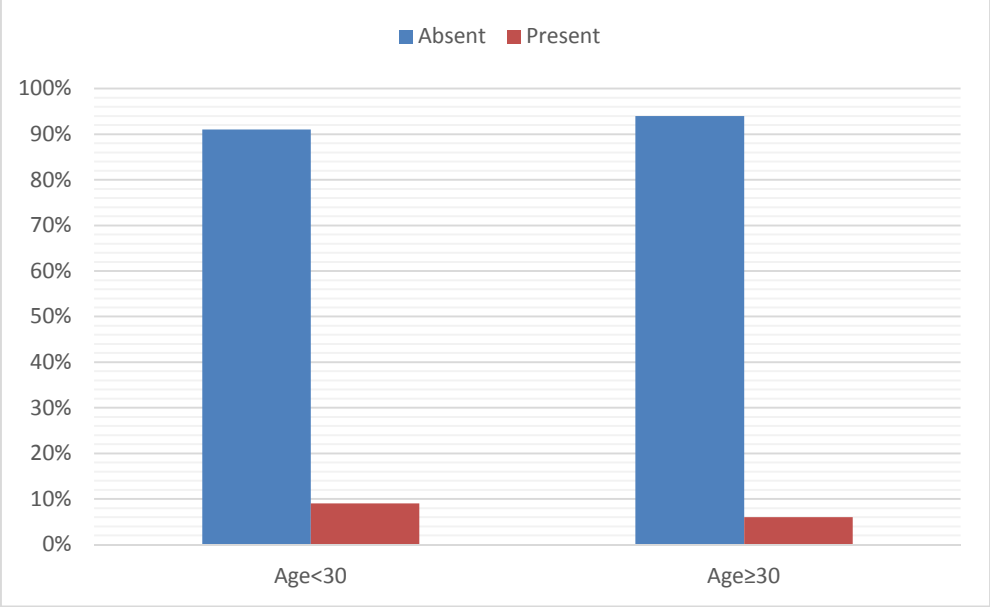


FIGURE.70

Table.70 and Figure.70 shows the correlation of prurigo of pregnancy with age.8(9%) below the age of 30 and 1(6%) 30 years and above had prurigo of pregnancy.

CORRELATION OF PRURIGO OF PREGNANCY WITH GRAVIDITY

(TABLE .71 and FIGURE.71)

		PRURIGO OF PREGNANCY			
		Absent	Present	Total	P value
Gravida	G1	50(91%)	5(9%)	55	
	G2	17(89%)	2(11%)	19	0.45
	G3	19(95%)	1(5%)	20	
	G4	9(90%)	1(10%)	10	
Total		95	9	104	

TABLE.71

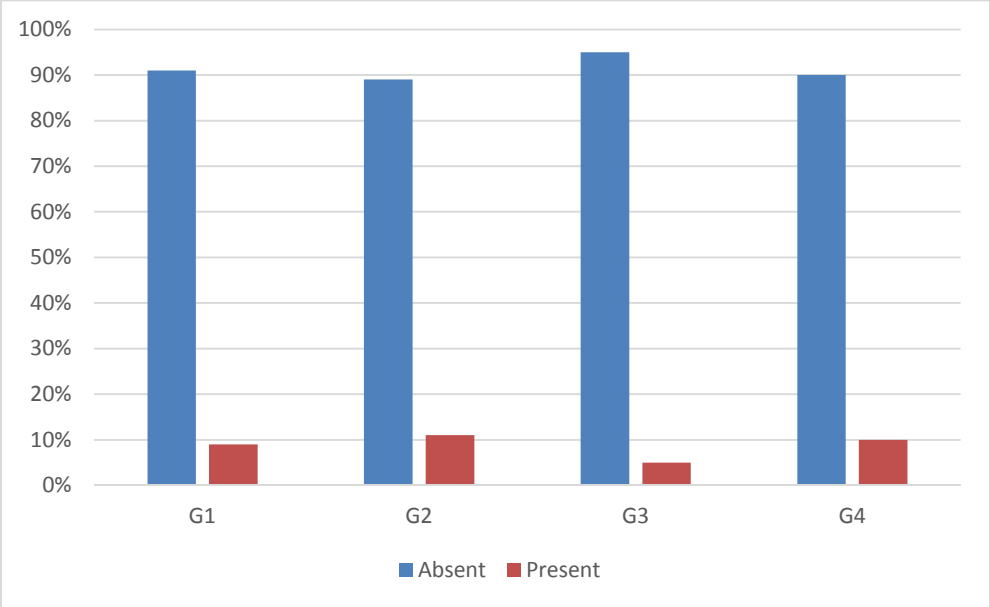


FIGURE.71

Table.71 and Figure.71shows the correlation of prurigo of pregnancy with gravidity.5(9%) in gravida one (G1),2(11%) in gravida two(G2),1(5%)in gravida three(G3) and 1(10%) in gravida four (G4) had prurigo of pregnancy.

CORRELATION OF PRURIGO OF PREGNANCY WITH PRIMIGRAVIDzE/MULTIGRAVIDAE.

(TABLE.72 and FIGURE.72)

		PRURIGO OF PREGNANCY			
		Absent	Present	Total	P value
Multigravida		45(92%)	4(8%)	49	
Primigravidae		50(91%)	5(9%)	55	0.028
Total		95	9	104	

TABLE.72

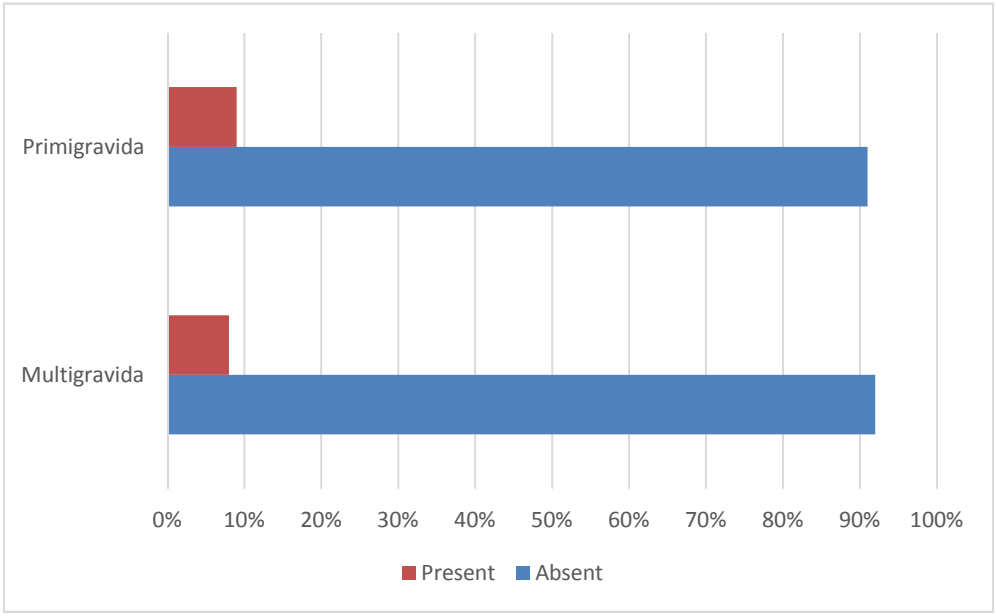


FIGURE.72

Table.72 and Figure.72 shows the correlation of prurigo of pregnancy with primigravidae/multigravidae.4(8%) multigravidas and 5(9%)primigravidas had prurigo of pregnancy.

CORRELATION OF PRURIGO OF PREGNANCY WITHTRIMESTER

(TABLE.73 and FIGURE.73)

Trimester		PRURIGO OF PREGNANCY		Total	P value
		Absent	Present		
Second		58(92%)	5(8%)	63	
Third		37(90%)	4(10%)	41	0.10
Total		95	9	104	

TABLE.73

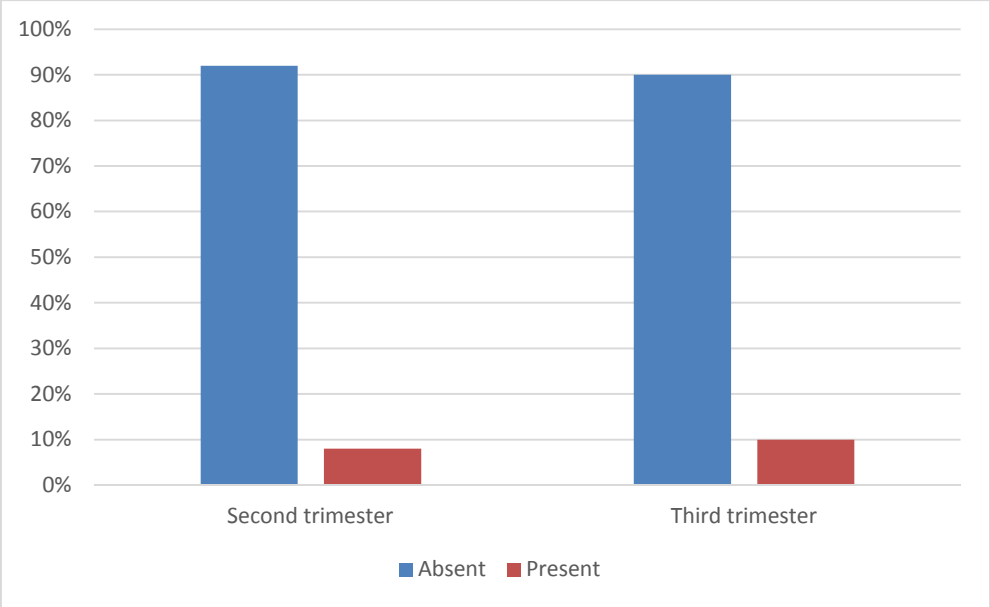


FIGURE.73

Table.73 and Figure.73 shows the correlation of prurigo of pregnancy with trimester.5(8%)in second trimester and 4(10%)in third trimester had prurigo of pregnancy.

CORRELATION OF PRURITIC URTICARIAL PAPULES AND PLAQUES OF PREGNANCY(PUPPP)WITH VARIOUS PARAMETERS

CORRELATION OF PUPPP WITH AGE

(TABLE.74 and FIGURE.74)

Age		PUPPP		Total	
		Absent	Present		P value
Age<30		80(91%)	8(9%)	88	
Age≥30		15(94%)	1(6%)	16	0.13
Total		95	9	104	

TABLE.74

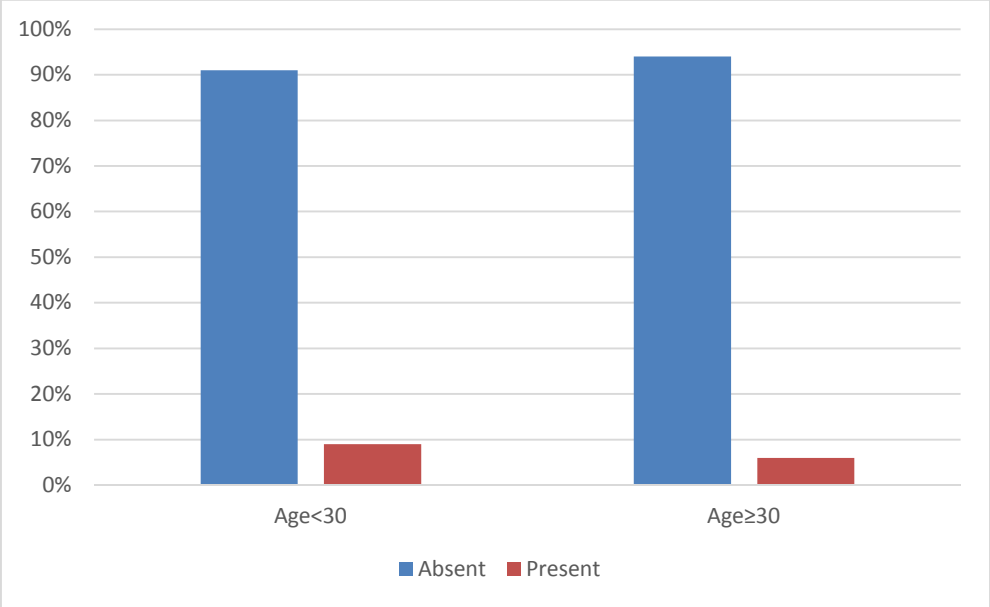


FIGURE.74

Table.74 and Figure.74 shows the correlation of PUPPP with age.8(9%) below 30 years and 1(6%)30 years and above had PUPPP.

CORRELATION OF PUPPP WITH GRAVIDITY

(TABLE.75 and FIGURE.75)

		PUPPP			
		Absent	Present	Total	P value
Gravida	G1	51(93%)	4(7%)	55	
	G2	19(100%)	0	19	5.21
	G3	16(80%)	4(20%)	20	
	G4	9(90%)	1(10%)	10	
Total		95	9	104	

TABLE.75

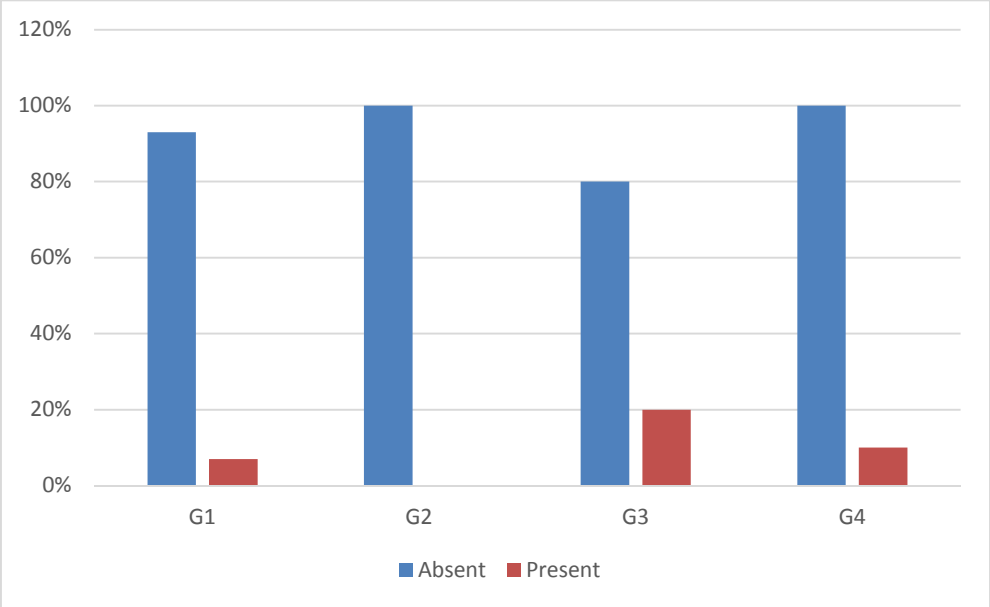


FIGURE.75

Table.75 and Figure.75shows the correlation of PUPPP with gravidity.4(7%)in gravida one(G1),4(20%)in gravida three(G3) ,1(10%)in gravida four(G4) had PUPPP .No women in gravida two(G2) had PUPPP.

CORRELATION OF PUPPP WITH PRIMIGRAVIDAE /MULTIGRAVIDAE.

(TABLE.76 and FIGURE.76)

		PUPPP		Total	P value
		Absent	Present		
Multigravida		44(90%)	5(10%)	49	
Primigravida		51(93%)	4(7%)	55	0.28
Total		95	9	104	

TABLE.76

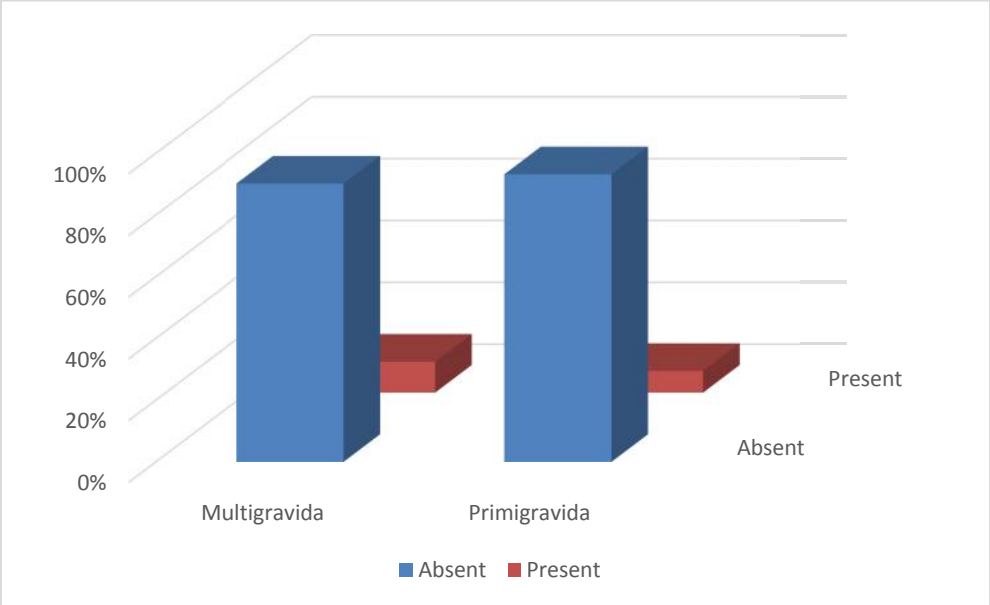


FIGURE.76

Table.76 and Figure.76 shows the correlation of PUPPP with primigravidae/ multigravidae.5(10%)multigravidas and 4(7%)primigravidas had PUPPP.

CORRELATION OF PUPPP WITH TRIMESTER

(TABLE.77 and FIGURE.77)

Trimester		PUPPP		Total	P value
		Absent	Present		
Second		58(92%)	5(8%)	63	
Third		37(90%)	4(10%)	41	0.10
Total		95	9	104	

TABLE.77

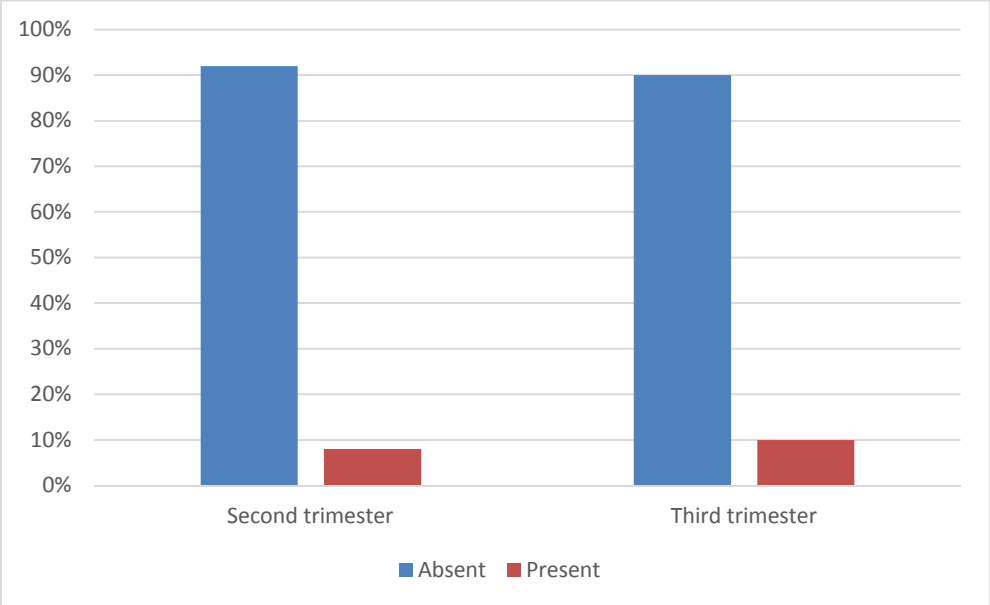


FIGURE.77

Table.77 and Figure.77 shows the correlation of PUPPP with trimester.5 (8%)in second trimester and 4(10%)in third trimester had PUPPP.

DISCUSSION

The present study was conducted on pregnant women in the second and third trimester attending a tertiary care hospital in Belagavi during the period January 2016 to December 2016.A total of 104 pregnant women were included in this cross sectional randomised study.Our observations are discussed below and compared with similar studies done in India and abroad.

PHYSIOLOGICAL SKIN CHANGES

In our study physiological skin changes were seen in 94(90%) of pregnant women out of 104 and the most common physiological skin change was linear nigra which was seen in 59 (57%) women out of 104.This findings were similar to a study done by Kumari et al.[123] where linea nigra was the most common physiological changeand was found in 555 women out of 607 (incidence 91.4%).

The other physiological skin changes found in our study were hyperpigmentation of the areola 32(31%),straie gravidarum 46(44%),chloasma 17(16%),oedema 11(10%),varicosities 9(9%),palmar erythema 4(4%),pyogenic granuloma 3(3%),gingival hyperplasia 1(1%),hirsutism 3(3%),subungual hyperkerstoses6(6%) and distal onycholysis 2(2%) in comparison to the study done by Kumari et al.[123] Where melasma was seen in 15/607 (2.5%) of cases. It has been reported to occur in 50-75% of pregnant women,[123] striae distensae (striae gravidarum) develop in up to 90% of women during the sixth and seventh month of pregnancy. In their study, striae were seen in 484 (79.7%) cases. Non pitting edema of legs, eyelids, face and hands is present in about 50% of women during the third

trimester Vascular changes seen in our study include nonpitting edema of feet in 59 (9.8%) cases and abdominal wall edema in 3 cases.[123]

LINEA NIGRA

In our study 59(57%) pregnant women had linea nigra which was the most common physiological change in comparisonto a study done by kumari et al.[123] where linea nigrawas seen in 91.4% cases and in a study done by Hassan et al[126]where pigmentary changes were seen in 89% of cases, of which linea nigra was the most common, seen in 80% of cases, and in a study done by Panickar et al.[l27] where linea nigra was found in 81.6%of the cases.

The incidence of linea nigra is91% in the total Indian population.

Correlation of linea nigra with age has not been studied in any earlier studies and in our study we did not find any statistical significance(P value 0.256)

Correlation of linea nigra with gravidity in our study did not have any statistical significance(P value 4.78)This correlation has not been done in any previous studies.

Correlation of linea nigra with primigravidae/multigravidae did not have any statistical significance (P value 1.06) This correlation has not been done in any previous studies.

Correlation of linea nigra with trimester did not show any statistical significance in our study(P value 30),however in a study done by Panicker et al.[127] Pigment changes were seen in 65.27% of the cases during the second trimester and in 99.5% of the cases during the third trimester. The increase in the incidence during the

second and third trimesters was because the placenta—the source of estrogen and progesterone, which are strong melanogenic stimulants.[127]

HYPERPIGMENTATION OF AREOLA

Hyperpigmentation of areola was found in 59(57%)out of 104 pregnant women in our study which was the third most common physiological change observed which is less than the study done by Hassan et al. where hyperpigmentation of the areola was the second most common physiological change and was seen in 75% of the cases.[126]

Correlation of hyperpigmentation of areola with age in our study did not show any statistical significance (P value 0.39)and there are no previous studies to compare.

Correlation of hyperpigmentation of areola with gravidity.In our study did not show any statistical insignificance(P value 6.60).No previous studies are available to compare these parameters.

Correlation of hyperpigmentation of areola with primigravidae/multigravidae did not show any statistical insignificance(P value 0.21). No similar previous studies have been done.

Correlation of hyperpigmentation of areola with trimester,did not show any statistical significance (P value 1.07).In our study 17(27%) in second trimester and 15(37%) in third trimester had hyperpigmentation of areola. However in a study done by Panicker et al.[127] pigment changes were seen in 65.27% of the cases during the second trimester and in 99.5% of the cases during the third trimester.

STRAIE GRAVIDARUM

In our study out of 104 pregnant women 46(44%)had straie gravidarum the incidence of which is less than the study done by Kumari et al.[123] where it was was found 72.8%of cases and Panicker et al. [127]where it was found in 79.9%of cases. Chang et al.[128]found incidence of striae gravidarum in their survey to be 55% which was similar to the findings in our study.

Correlation of straie gravidarum with age was statistically insignificant(P value 0.4)and no previous studies have been done.

Correlation of straie gravidarum with gravidity was statisticaly insignificant (P value 2.37).In our study 25(51%)multigravidas and 21(38%)primigravidas developed straie gravidarum which is similar to studies done Kumari et al.[123] where 267 (55.2%) were multi gravidas 217 (44.8%) were primigravidas and in a study done by Panicker et al.[127] where it was found in multigravidas (76.95%) and primigravidas (45.67%).

Correlation of straie gravidarum with primigravidae/multigravidae was not statistically significant(P value 1.73). In our study 25(51%)multigravidas and 21(38%)primigravidas developed straie gravidarum which is similar to the incidence in studies done by Kumari et al.[123] where 267 (55.2%) were multi gravidas 217 (44.8%) were primi gravidas and by the study done by Panicker et al.[127] where it was found in multigravidas (76.95%) and primigravidas (45.67%).

Correlation of straie gravidarum with trimester was statistically insignificant(P value 7.1).In our study we found 21(33%)in second trimester and 25(61%)in third trimester however in the study done by Kumari et al.[123] straie gravidarum was found to be more common during the second trimester.

CHLOASMA

In our study out of 104 women 17(16%)had chloasma. In a study done by Kumari et al.[123]chloasma was reported a very low incidence of 2.5% of cases,[123]and in a study by Hassan et al.[126] chloasma was reported a very high incidenceof 64%of cases . This difference may be due to the fact that pigmentary changes are more discernible in the fair skinned individuals. The incidence has been reported in Indian population to be 50%.[123]

Correlation of chloasma with age had no statistical significance(P value 0.20).No previous studies showed such correlation.

Correlation of chloasma with gravidity had no statistical significance (P value 2.42).No previous studies are available for comparison.

Correlation of chloasma with primigravidae/multigravidae had no statistical significance (P value 0.24). No previous studies have been done to compare this parameter.

OEDEMA

In our study out of 104 pregnant women 11 (10%) had oedema which is similar to the study done by Panicker et al [127] where nonpitting edema of feet was seen in 10.5% of the cases.However the incidence of oedema in pregnancy is 50% in Indian population.[123]

Correlation of oedema with age was not statistically significant (P value 8.5) No previous studies have evaluated this parameter.

Correlation of oedema with gravidity did not show any statistical significance (P value 6.8)No previous studies have evaluated this parameter.

Correlation of oedema with primigravidae/multigravidae had no statistical significance(P value 5.88)No previous studies have evaluated this parameter.

Correlation of oedema with trimester in our study did not show any statistical significance (P value 18.72).In our study 11(27%) of all the women who had oedema were in their third trimester, in a study done by Kroumpouz et al.[1] nonpitting edema of the legs, eyelids,face, and hands was present in about 50% of women during the third trimester .

VARICOSITIES

In our study 9(9%)pregnant women out of 104 had varicosities.However in a recent study by Muzaffar et al.[25]varicosities of lower legs was seen in 2.8% of cases.

Correlation of varicosities with age did not show any statistical significance(P value1.77)No previous studies have been done.

Correlation of varicosities with gravidity did not show any statistical significance (P value 4.48).No previous studies have evaluated this parameter.

Correlation of varicosities with primigravidae/multigravidae did not have any statistical significance (P value 3.71).No previous studies have evaluated this parameter.

Correlation of varicosities with trimester did not have any statistical significance(P value 15.1).In our study all the 9(9%)cases were in their third

trimester. Varicosities are most common in anus and legs, appearing in 40% of pregnant women during the 3rd trimester.[39]

PALMAR ERYTHEMA

In our study 4(4%)pregnant women out of 104 had palmar erythema which issimilar to a study done by Hassan et al.[126]where the incidence was reported to be 6.3%.Raj et al.[129]in their study reported the prevalence of palmar erythema to be 33.3%. Bean et al. [130] reported the prevalence to be 62.5% in pregnant white women and 35% in black women.

Correlation of palmar erythema with age did not show any statistical significance (P value 0.75) There are no previous studies which have compared this parameter.

Correlation with gravidity did not show any statistical significance(P value 3.71). No such previous studies done.

Correlation of palmar erythema with primigravidae and multigravidae showed a statistical significance with (P value 0.01) where out of 49 multigravidae 2(4%)had palmar erythema and out of 55 primigravidae 2(4%)had palmar erythema.However there are no previous similar correlations done.

Correlation with trimester did not show any statistical significance.(P value 0.35). No previous studies have evaluated this parameter.

PYOGENIC GRANULOMA

In our study 3(3%)out of 104 pregnant women had pyogenic granuloma which is similar to a study done by Raj et al [131] where 3 cases of pyogenic granulomas were noted in their study.

No previous studies have been done to correlate pyogenic granulomas with gravidity,primigravidas/multigravidas and trimester.

Correlation with gravidity did not show any statistical significance(P value 2.75) Correlation with primigravidae/multigravidae did not show any statistical significance(P value 2.75)

Correlation of pyogenic granuloma with trimester(second trimester) showed a statistical significance with (P value of 0.04).Out of 63 women in their second trimester 2(3%)had pyogenic granuloma, out of 41 women in their third trimester 1(2%)had pyogenic granuloma.

GINGIVAL HYPERPLASIA

In our study gingival hyperplasia was observed in only 1(1%)pregnant women out of 104 which issimilar to a study done by Panicker et al.[127] where gingival hypertrophy was found in 11/600 patients(1.8%).In a study by Muzaffar et al.[25]higherincidence of 23/140 (16.4%) of gingival edema and redness was noted.

No studies in the past have compared gingival hyperplasia with gravidity, primigravidas/ multigravidas and trimester.

Correlation with gravidity did not show any statistical significance. (P value 0.90) Correlation with primigrvidae/multigravidae did not show any statistical significance(P value 0.90).

Correlation with trimester showed no statistical significance.(P value0.65)

HIRSUTISM

In our study 3(3%)women had hirsutism out of 104 pregnant women. The definition of hirsutism states that an increase in terminal hair that commonly appears in a male pattern in women. It is associated with hyperandrogenemia.Hirsutism occurs inapproximately seven percent of women in the world.[132]

No previously done studies have compared hirsutism with age,gravidity, primigravidas/multigravidas and trimester.

Correlation with age had no statistical significance.(P value 0.56)

Correlation with gravidity showed no statistical significance.(P value 1.38)

Correlation with primigravidae/multigravidae had no statistical significance.(P value 0.23)

Correlation of hirsutism with trimester(second trimester)in our study showed a statistical significance with (P value of 0.04) where out of 63 women in their second trimester 2(3%)had hirsutism and out of 41 women in their third trimester 1(2%)had hirsutism.

SUBUNGUAL HYPERKERATOSES

In our study out of 104 pregnant women 6(6%)had subungual hyperkeratosis which is similar to a study done by Erpolat S et al.[133] where subungual hyperkeratoses has been observed in 6%of cases.

KOH was done in our study to rule out any fungal foci.

Correlation of subungual hyperkeratosis with age in our study showed a statistical significance with(P value 0.008). Out of 88 pregnant women below 30 years of age 5(6%)had subungual hyperkeratosis,and out of 16 pregnant women 30 years and above 1(6%)had subungual hyperkeratoses.

No known study has been done to compare subungual hyperkeratoses with age, primigravidae and trimester.

Correlation of subungual hyperkeratosis with primigravidae/multigravidae showed a statistical significance with(P value of 0.02). Out of 49 multigravidae,3(6%) had subungual hyperkerstosis and out of 55 primigravidae, 3(5%)had subungual hyperkeratosis.

Correlation with trimester did not have any statistical significance.(P value 4.14)

DISTAL ONYCHOLYSIS

In our study out of 104 pregnant women 2(2%)had distal onycholysis.The exact incidence of distal onycholysis is unknown

No previous studies have been carried out to correlate with age, gravidity and primigravidae/multigravidae .

Correlation of distal onycholysis with age had no statistical significance.(P value 0.37) Correlation of distal onycholysis with gravidity did not have any statistical significance.(P value 1.57)

Correlation with primigravidae and multigravidaeshowed a statistical significance with(P value of 0.007) Out of 49 multigravidae 1(2%)had distal onycholysis and out of 55 primigravidae 1(2%)had distal onycholysis.

SPECIFIC DERMATOSES OF PREGNANCY

In our study specific dermatoses of pregnancy was seen in 28(27%) of pregnant women out of 104 and the most common specific dermatoses of pregnancy observed was pruritic urticarial papules and plaques of pregnancy(PUPPP) 9(9%) and prurigo of pregnancy 9(9%) out of 104 women,followed by pruritus of pregnancy 4(4%),eczema in pregnancy 4(4%) and pruritic folliculitis of pregnancy 2(2%).

Specific dermatoses of pregnancy is seen in 0.5 to 3.0% of the cases.[124] In a study by Kumari et al.[123]of 607 pregnant women, 22 (3.6%) cases of specific dermatoses of pregnancy were seen. Of these the most common was PUPPP (also known as polymorphic eruption of pregnancy) with a total of 63.6% (14/22) cases followed by 5 (22.7%) cases of pruritus gravidarum. In an Indian study done by Shivakumar et al.[125]pruritus was found to be the commonest symptom (58.82%).

PRURIGO OF PREGNANCY

In our study prurigo of pregnancy was one of the most common specific dermatoses of pregnancy, found in 9(9%)of pregnant women out of 104 which is similar to an Indian study done by Shivakumar et al.[125] where 16 (9.41%) had prurigo of pregnancy.

Prurigo of pregnancy, previously described as Besnier's prurigo gestationis is seen in about 1 in 300 pregnancies and is reported in all trimesters of pregnancy.[135]

Correlation with age showed no statistical significance (P value 0.13). No previous studies have evaluated this parameter.

On Correlation with gravidity no statistical significance was found (P value 0.45). In our study 4(8%) multigravidae and 5(9%)primigravidae had prurigo of pregnancy. However in a study done by Hassan et al.[126] prurigo of pregnancy was present in 27% of multigravidae, which was much higher than in our study.

Correlation of prurigo of pregnancy with primigravidae and multigravidae had a statistical significance with a (P value of 0.02).Out of 49 multigravidae 4(8%)had prurigo of pregnancy and out of 55 primigravidae 5(9%)had prurigo of pregnancy. In a study done by Hassan et al.[126]prurigo of pregnancy was present in a higher incidence of 50% of all the cases.

Correlating with trimester, no statistical significance was found. (P value 0.10)In our study 5(8%)were in the second trimester and 4(10%) were in the third trimester.Prurigo of pregnancy, previously described as Besnier's prurigo gestationis is seen in a study done by Black et al[135] in about 1 in 300 pregnancies and is reported in all trimesters of pregnancy.

PRURITIC URTICARIAL PAPULES AND PLAQUES OF PREGNANCY(PUPPP)

In our study PUPPP was one of the most common specific dermatoses of pregnancy, found in 9(9%)of pregnant women out of 104.In an Indian study done by Kumari et al.[123]the incidence of PUPPP was reportedto be low in 14/604 cases (2.3%). PUPPP occurs in 1 of 160-240 pregnancies and is more common in white women.[136]

On Correlation with age, no statistical significance was found. (P value 0.13)No previous studies have evaluated these parameters.

Correlation with gravidity showed no statistical significance in our study. (P value 5.21)but the incidence of PEP is reported to be higher in multiple gestations [126]

On Corelating with primigravidae/multigravidae no statistical significance was found. (P value 0.28)In our study 5(10%) multigravidae and 4(7%)primigravidae had PUPPP but in a study done by Hassan et al.[126] All the affected patients were primigravidae.

Correlation with trimester no statistical significance was found.(P value 0.10)In our study 5(8%) women were in their second trimester and 4(10%) were in their third trimester,however it has been reported to occurs classically in primigravidae during the third trimester of pregnancy .[126]

PRURITUS OF PREGNANCY

In our study was found in 4(4%) out of 104 pregnant women which is similar to a study done by Panicker et al.[127] where six (3.52%) had pruritus gravidarum.

Correlation of pruritus of pregnancy with age had no statistical significance.(P value 3.82)No similar previous studies have been done.

Correlation with gravidity had no statistical significance.(P value 1.93)No studies are available to compare this parameter.

Correlation of pruritus of pregnancy with primigravidae/multigravidae in our study had a statistical significance with (P value of 0.01) Out of 49 multigravidae 2(4%)had pruritus of pregnancy and out of 55 primigravidae 2(4%) had pruritus of pregnancy.However there are no previous studies done to compare this parameter.

Correlation with trimester had no statistical significance.(P value 0.36)In our study 3(5%) in second trimester and 1(2%)in third trimester had pruritus of pregnancy,however in a study done by Panicker et al.[127] it was more common in the third trimester.

ECZEMA IN PREGNANCY

Was found in 4(4%)of pregnant women out of 104. Eczema is the most common dermatosis of pregnancy, accounting for between a third and a half of all cases. Only 20-40% of patients are estimated to have a pre-existing history of eczema; the rest develop symptoms for the first time during pregnancy.Three quarters of these patients develop symptoms within the first two trimesters. The total prevalence of eczema in pregnancy is unknown.[4]

Correlation with age in our study showed no statistical significance. (P value 3.76)No previous study has evaluated eczema in pregnancy with age.

Correlation with gravidity had no statistical significance.(P value 1.84)No previous study has evaluated this parameter.

Correlation with primigravidae and multigravidae in our study showed a statistical significance with (P value of 0.01)Out of 49 multigravidae 2(4%)had eczema in pregnancy and out of 54 primigravidae 2(4%)had eczema in pregnancy. However in a study done by Ambros et al.[4]It has been mostly reported in primigravidae.

Correlation with trimester showed no statistical significance.(P value 2.75)In our study 4(6%) in second trimester and no women in third trimester had eczema in pregnancy,it has been mostly reported during the second trimester by Ambros et al.[4]

PRURITIC FOLLICULITIS OF PREGNANCY

Pruritic folliculitis of pregnancy was found in our study in 2(2%)of pregnant women out of 104 which is similar to a recent Indian study done by Puri et al. [137] where pruritic folliculitis of pregnancy was reported in (2%) of cases.

Correlation with age had no statistical significance in our study.(P value 0.37)No previous studies have evaluated this parameter.

Correlation with gravidity had no statistical significance in our study. (P value 1.57) No previous studies to correlating pruritic folliculitis of pregnancy with gravidity have been done.

Correlation with primigravidae /multigravidae had a statistical significance with (P value of 0.007) Out of 49 multigravidae 1(2%)had pruritic folliculitis of pregnancy and out of 55 primigravidae 1(2%)had pruritic folliculitis of pregnancy.No previous study has correlated this parameter.

Correlation with trimester did not have any statistical significance. (P value 0.09)In our study 1(2%)in second trimester and 1(2%)in third trimester had pruritic folliculitis of pregnancy,however in a study done by Panicker et al.[127] pruritic folliculitis of pregnancy was found to be more common (>50%) during the third trimester.[127]

An increased frequency of infection was seen in our study, which is common during pregnancy and is probably related to low cellular immunity the most common infection in our study noted was tinea corporis and tinea cruris with incidence of 14(13%) and 8(8%)respectively followed by pityriasis versicolor 6(6%). The other infective dermatitis found was 3 (3%) varicella,1(1%)had intertrigo, 1(1%)had herpes labialis. In a study done by Panicker et al.[127] the infections seen in the study group were dermatophytosis (21.6%), scabies (2.8%), pityriasis versicolor (6.16%) and molluscum contagiosum (0.3%). Increased eccrine sweating, warm climate, and depressed cellular immunity may be the reasons for higher incidence of these infections [134]

The other dermatoses found during our study was 4(4%)irritant contact dermatitis,3(3%) acne vulgaris, 1(1%) polymorphic light eruption (PMLE), 1(1%) lymphadenoma, 1(1%) lymphangitis, 1(1%) vitiligo vulgaris,1(1%) seborrheic dermatitis and 1(1%) acrochordons,respectively.

LIMITATION OF OUR STUDY

- This study lacks generalisation to the entire population of pregnant women as the study was carried out on pregnant women having dermatological chief complaints.

- The sample size was small (104 pregnant women).

- First trimester was excluded because in literature most of the dermatological manifestations commenced in the second and third trimester.

- As the study was done in a tertiary care hospital chances of over estimation of the prevalence of specific dermatoses of pregnancy is possible**.**

CONCLUSIONS

- Based on the findings in this study,the following conclusions were made:
- Itchy lesions were the most common complaint 45(43%).
- 94(90%)women had physiological skin changes of pregnancy and 28(27%)women had specific dermatoses of pregnancy.
- Linea nigra 59(57%)was the most common physiological skin changefollowed by straie gravidarum 46(44%) and hyperpigmentation of areola 32(31%).
- Pruritic urticarial papules and plaques of pregnancy 9(9%) and prurigo of pregnancy 9(9%)were the two most common specific dermatoses of pregnancy.
- Tinea corporis 14(13%) was the most common infective dermatitis.
- A statistical significance was found between Pyogenic granuloma and women in their second trimester.
- A statistical significance was found between palmar erythema and multigravidae and primigravidae.
- A statistical significance was found between hirsutism women in their second trimester.
- A statistical significance was found between subungual hyperkeratoses and age i.e pregnant women below thirty years and pregnant women thirty years and above.
- Astatistical significance was also found between subungual hyperkeratoses and multigravidae.
- A statistical significance was found between distal onycholysis and multigravidae and primigravidae.

- A statistical significance was found between pruritus of pregnancy and multigravidae and primigravidae.
- A statistical significance was found between eczema in pregnancy and multigravidae and primigravidae.
- A statistical significance was found between pruritic folliculitis of pregnancy and multigravidae and primigravidae.
- A statistical significance was found between prurigo of pregnancy and primigravidae.
- Our study emphasizes the need for a detailed and meticulous examination of pregnant women to detect various other disorders as pregnant women are more susceptible to a wide range of dermatological conditions, infections apart from the specific dermatoses of pregnancy.
- Skin changes are quite common in pregnancy though most of them are physiological in nature and need no further management.
- However,most of thepruritic eruptions of pregnancy, which are not a rare entity, can be a source of significant distress to pregnant women and need timely therapeutic intervention.

SUMMARY

This study ‘Skin changes in pregnant women in the second and third trimester is a one year cross sectional study which was conducted in a tertiary care hospital in Belagavi during the period January 2016 to December 2016.

A total of 104 randomly presenting pregnant women in the second and third trimester coming with skin changes in the outpatient and inpatient care in a tertiary care Hospital in Belagavi were selected for this study.

- 88(85%) pregnant women were below the age of thirty and 16(15%) were thirty years and above.
- 55(53%) were primigravidae and 49(47%) were multigravidae.
- Most of these women were housewives.
- 62(60%) women were in their second trimester and 42(40%) women were in their third trimester when they presented to us.
- Itchy lesions were the most common complaint 45(43%).
- 94(90%) women had physiological skin changes of pregnancy and 28(27%) women had specific dermatoses of pregnancy.
- Linea nigra 59(57%) was the most common physiological skin change followed by straie gravidarum 46(44%) followed by hyperpigmentation of areola 32(31%).
- Pruritic urticarial papules and plaques of pregnancy 9(9%) and prurigo of pregnancy 9(9%) were the two most common specific dermatoses of pregnancy.
- Tinea corporis 14(13%) was the most common infective dermatitis.

- Pyogenic granuloma was found in 2(3%) women in their second trimester and 1(2%)in their third trimester and a statistical significance was found between pyogenic granuloma and women in second trimester. (P value 0.04)
- Palmar erythema was seen in 2(4%) multigravidae and 2(4%) primigravidae and a statistical significance was found between the correlation of palmar erythema and multigravidae/primigravidae. (P value 0.01)
- Hirsutism was seen in 2(3%) of women in second trimester and 1(2%) women in third trimester and a statistical significance was found between hirsutism and women in second trimester.(P value 0.04)
- Subungual hyperkeratoses was seen in 5(6%)pregnant women below the age of thirty and 1(6%) pregnant women thirty years and above and a statistical significance was found between the both. (P value 0.008)
- Subungual hyperkeratoses was observed in 3(6%) multigravidae and 3(5%) primigravidae and a statistical significance was found between subungual hyperkeratoses and multigravidae. (P value 0.02)
- Distal onycholysis was seen in 1(2%) multigravidae and 1(2%) primigravidae and a statistical significance is found between the both. (P value 0.007)
- Pruritus of pregnancy was seen in 2(4%) multigravidae and 2(4%) primigravidae and statistical significance was found between the both. (P value 0.01)
- Eczema in pregnancy was seen in 2(4%) multigravidae and 2(4%) primigravidae and a statistical significance was found between the both. (P value 0.05)

- Pruritic folliculitis of pregnancy was present in 1(2%) multigravidae and 1(2%) primigravidae and a statistical significance was found between the both. (P value 0.007)
- Prurigo of pregnancy was seen in 4(8%) multigravidae and 5(9%) primigravidae and a statistical significance was found between prurigo of pregnancy and primigravidae. (P value 0.02)

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ANNEXURE I

CONSENT FORM

I.D.NO. _____ INFORMED CONSENT FORM

The study is conducted by _____Post-graduate student in M.D Dermatology, Venereology and Leprosy under guidance of _____, Department of Dermatology, J N Medical College, Belgaum.

Respected Madam,

We invite you to participate in our study as, you are eligible for the same. During the study you will be asked some questions in detail regarding your present complaints.

Purpose of the study:

Skin changes in pregnant women are very common. During pregnancy profound immunological, metabolic ,endocrine and vascular changes take place which are responsible for the skin changes.The concerns of the patient may range from cosmetic appearance ,to the change of recurrence of the particular problem during a subsequent pregnancy, to its potential effects on the fetus.Moreover pregnancy modifies the course of a number of pre-existing dermatological conditions. Though very common no large study to our knowledge has been done in Belgaum to know the frequency and pattern of skin changes in pregnant women.

Procedure and treatment:

If you choose to participate, you will be asked to give a detailed history of your disease, undergo a dermatological and physical examination and answer the questions which will be handed to you in the form of preformed questionnaire.

Risks and benefits:

The result of you taking part in this research would help health care providers towards a better understanding of the skin changes, and thus we will be able to provide improved patient care. There is no risk associated with the study.

Alternatives:

If you decide not to participate in this study, you will still be receiving the usual standard care.

Privacy and confidentiality:

Your privacy will be respected and all information collected about you during the course of this study will be kept confidential. Your identity will remain undisclosed.

Relations with the Institutional/ Sponser's policy:

The J N Medical College will provide, within the limitations of the laws of the State of Karnataka, facilities and medical attention to patients who suffer injuries as a result of participating in this project. However, the treatment will not be free and no reimbursement will be provided.

Financial incentives :-

You shall not be receiving any payment or any financial incentives for participating in this study.

Authorization to publish results:

The results of this study may be published for scientific purpose or presented to a scientific group. Your identity, however, will be maintained confidential at all times.

Voluntary participation:

Your participation in this study is voluntary. Your decision whether or not to participate will neither affect the care , nor your future relations with the doctor or the hospital.

Questions:

If you need any further information regarding your rights as a study participant, you may also contact Dr.Ganga S. Pilli (Mobile No.9480275601), Chairman of Institutional Ethics Committee, JNMC, and Belagavi-10

STATEMENT OF CONSENT:

I.D.NO:_____

I Mrs _____Volunteer and consent to participate in this study. I have read the consent document that has been read to me in my vernacular language. I accept to participate in the study. All the information regarding this study is provided to me and I have understood the same. I have been given the opportunity to ask questions and obtain appropriate answers.

Participant's Parents name:

Signature or left thumb print of participant & Date:

Witness name:

Signature of witness &Date :

Signature of the investigator & Date :

ANNEXURE II

PROFORMA

Sr no: Date: OPD/IPD no:

Name: Age: Occupation:

Husbands name: Occupation:

Consanguinity in marriage: YES ☐ Degree ☐ NO ☐

Address:

LMP: EDD: Gestational age:

Gravida:	Para:	Abortion/still birth:	Living:

Chief complaints:

I. Itching ☐
II. Itchy lesions ☐
III. Redness/red lesions ☐
IV. Pigmentation ☐
V. Fluid filled lesions ☐
VI. Raised lesions ☐
VII. Dry itchy skin ☐
VIII. Hairfall ☐

IX. Oral lesions ☐

X. Nail changes ☐

XI. Others ☐

History of present illness:

History of similar complaints in previous pregnancy:

YES:	NO:	FIRST PREGNANCY:

Associated illnesses:

Diabetes:	Hypertension:	Thyroid:	Anaemia:	Epilepsy:	Others:

History of:

Drugs:	Radiation:	Topical creams/ointments:	Vaginal discharge:	Vaccines	Blood transfusion:

Type of previous delivery:

Normal:	Instrumental:	C -section:	Preterm:	Term:	Post term:

General physical examination:

Weight:	Height:	BP:
Pulse:	RR:	Temp:
Pallor:	Icterus:	Cyanosis:
Clubbing:	Oedema:	Lymphadenopathy:

Mucocutaneous examination:

Skin:

Hair/scalp:

Oral cavity:Tongue:

Dental caries :

Oral mucosa:

Nails:

Systemic examination

Cardio vascular system:

Respiratory system:

Cenral nervuous system:

Per abdomen:

Investigations:

Impression:

ANNEXURE III-PHOTOGRAPHS

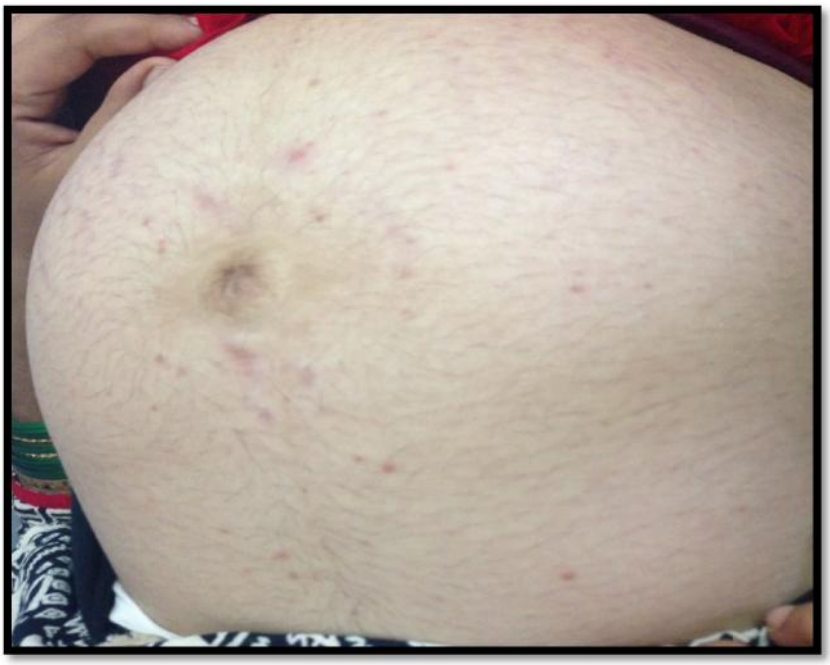
PHOTOGRAPH 1A. LINEA NIGRA

PHOTOGRAPH . 1B. LINEA NIGRA

PHOTOGRAPH 2. STRAIE GRAVIDARUM

PHOTOGRAPH 3. HYPERPIGMENTATION OF AREOLA

PHOTOGRAPH 4. PRURITIC FOLLICULITIS OF PREGNANCY



PHOTOGRAPH 5. PRURITIC URTICARIAL PAPULES AND PLAQUES OF PREGNANCY

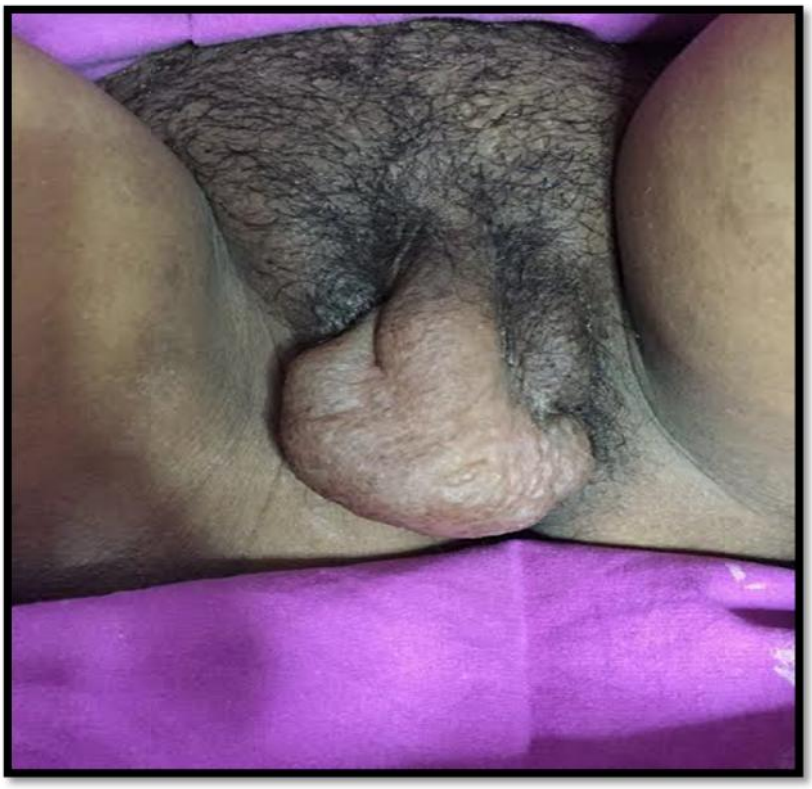
PHOTOGRAPH 6. PRURIGO OF PREGNANCY WITH STRAIE GRAVIDARUM

PHOTOGRAPH 7. IRRITANT CONTACT DERMATITIS

PHOTOGRAPH 8. TINEA CORPORIS

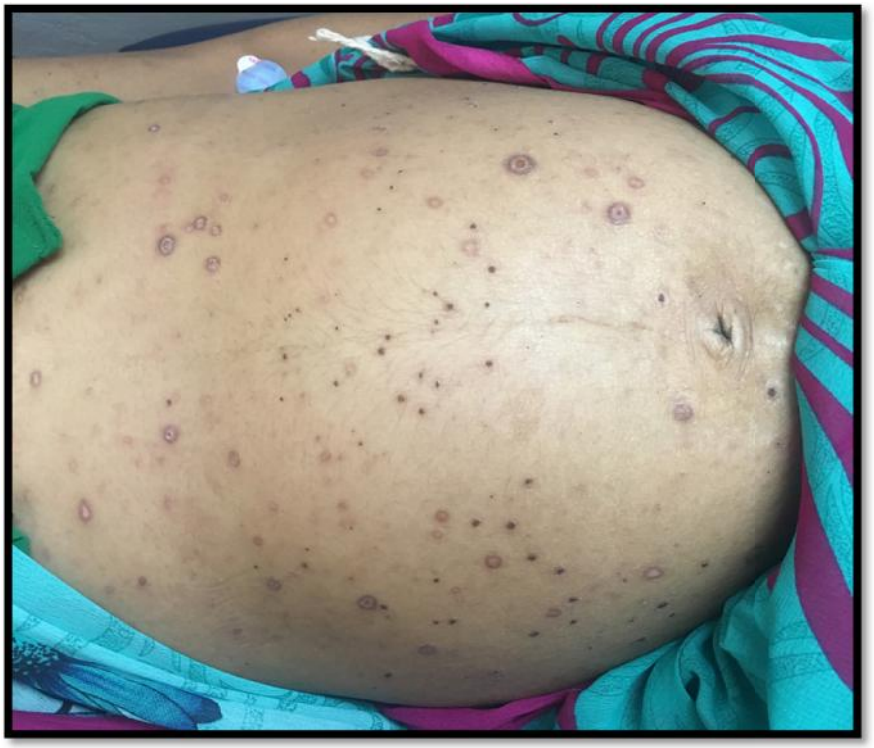
PHOTOGRAPH 9. TINEA CRURIS

PHOTOGRAPH 10. INTERTRIGO



PHOTOGRAPH 11. LYMPHANGITIS

PHOTOGRAPH 12. VITILIGO VULGARIS



PHOTOGRAPH 13. VARICELLA

ANNEXURE-IV MASTER CHART

Sr no.	Age	Occupation	Gravida	Parity	Living	Abortion	Trimester	Gestational age	Chief complaint	Investigation	Physiological skin changes	Specific dermatoses	Other skin conditions
1	23	HW	G1	P1	L0	A0	2	27weeks	Itching	-	LN+S+HA	PP	-
2	28	HW	G3	P2	L1	A1	2	24weeks	Itchy lesions	KOH	S+LN	-	T.cru
3	32	Farmer	G3	P2	L1	A1	2	24weeks	Itchy lesions	KOH	LN+HA	-	T.cru
4	21	HW	G1	P1	L0	A0	3	32weeks	Asymptomatic lesions	WL	PG+HA+S+LN	-	P.versicolor
5	22	Self emp	G1	PO	L0	A0	2	15weeks	Pigmentation	WL	Chloasma+LN+S	-	-
6	24	Factory W	G1	P1	L0	A0	2	23weeks	Itchy lesions	KOH	LN+S+SH		T.cru
7	25	HW	G1	P1	L0	A0	2	16weeks	Itchy lesions	-	PE	PUPPP	-
8	22	HW	G1	P1	L0	A0	3	29weeks	Strech marks	-	S+LN+HA	-	-
9	22	Farmer	G1	P0	L0	A0	2	13weeks	Itchy lesions	-	-	POP	-
10	24	HW	G3	P3	L2	A0	3	28weeks	Asymptomatic lesions	WL	LN	-	P.versicolor
11	22	Self emp	G2	P2	L1	A0	2	26weeks	Pigmentation	WL	chloasma+LN+SH	-	-
12	23	Farmer	G1	P1	L0	A0	2	25weeks	Itchy lesions	-	LN+S+DO	-	T.corp
13	26	HW	G4	P3	L2	A1	3	32weeks	Itching	-	S+LN+V	PUPPP	-
14	24	Office	G2	P2	L1	A0	2	26weeks	Itchy lesions	-	LN	POP	-
15	23	Self emp	G1	P1	L0	A0	2	24weeks	Itchy lesions	KOH+WL	Chloasma+HA	-	T.corp
16	28	HW	G4	P3	L2	A1	3	30weeks	Itchy lesions	-	LN+HA+V	-	ICD
17	22	HW	G1	P0	L0	A0	2	15weeks	Itchy lesions	-	HA	-	PMLE
18	29	Farmer	G4	P2	L2	A1	2	19weeks	Itchy lesions	KOH	HA	-	T.cru
19	21	HW	G1	P1	L0	A0	3	29weeks	Itchy lesions	KOH	LN+S+HA	-	T.corp
20	22	HW	G1	P1	L0	A0	2	25weeks	Pigmentation	WL	Chloasma+LN	-	-
21	25	Factory W	G2	P2	L1	A0	3	28weeks	Itchy lesions	KOH	LN+S	-	T.corp
22	32	Self emp	G3	P3	L2	A0	3	31weeks	Itching	-	LN+S+V	PUPPP	-
23	28	HW	G2	P2	L1	A0	2	24weeks	Itchy lesions	-	LN+S+SH	-	ICD
24	24	HW	G1	P1	L0	A0	2	23weeks	Itchy lesions	KOH	LN+S+PG	-	T.corp
25	28	Office	G3	P3	L2	A0	3	31weeks	Pigmentation	WL	Chloasma+LN+V	-	-
26	27	Self emp	G3	P2	L2	A0	2	18weeks	Strech marks	-	S	-	-
27	31	HW	G4	P4	L2	A1	2	21weeks	Dryness	-	HA	EP	-
28	27	Farmer	G3	P3	L2	A0	2	22weeks	Strech marks	-	S	-	-
29	28	Farmer	G2	P2	L1	A0	2	27weeks	Pigmentation	WL	Chloasma+LN+S	-	-
30	24	Self emp	G1	P1	L0	A0	3	29weeks	Itchy lesions	KOH	LN+S+O	-	T.corp

31	30	HW	G4	P4	L3	A0	3	29weeks	Pigmentation	-	LN+S	-	-
32	23	Self emp	G2	P2	L1	A0	2	25weeks	Itchy lesions	-	S	-	ICD
33	22	Farmer	G1	P1	L0	A0	2	26weeks	Itchy lesions	KOH	LN+SH	-	T.corp
34	27	HW	G3	P3	L1	A1	3	34weeks	Swelling in groin	-	LN+S+O+V	-	Lymphadenoma
35	21	Factory W	G1	P1	L0	A0	3	36weeks	Pigmentation	-	LN	-	-
36	31	HW	G3	P2	L1	A1	2	23weeks	Itchy lesions	-	LN	EP	-
37	22	HW	G1	P1	L0	A0	3	35weeks	Asymtomatic lesions	WL	LN+0	-	P.versicolor
38	31	Self emp	G4	P4	L3	A0	2	26weeks	Itching	-	LN+S	PP	-
39	24	Farmer	G1	P1	L0	A0	3	33weeks	Itchy lesions	-	LN+S+V	PFP	-
40	31	HW	G2	P2	L1	A0	3	30weeks	Pigmentation	-	LN+HA+O	-	-
41	22	HW	G1	P1	L0	A0	2	23weeks	Itchy lesions	-	HA+LN	-	ICD
42	24	Office	G1	P1	L0	A0	2	25weeks	Itchy lesions	KOH	SH	-	T.corp
43	29	Self emp	G3	P3	L2	A0	3	34weeks	Swelling in groin	-	LN+O	-	Lymphangitis
44	20	HW	G1	P1	L0	A0	3	32weeks	Pigmentation	-	LN	-	-
45	19	Farmer	G1	P1	L0	A0	2	24weeks	Itching	-	PG+LN	POP	-
46	31	HW	G3	P2	L1	A1	3	30weeks	Asymtomatic lesions	KOH	LN+O+S	-	T.corp
47	21	HW	G1	P1	L0	A0	2	25weeks	Itching	-	LN+HA	POP	-
48	23	HW	G2	P2	L1	A0	3	34weeks	Itchy lesions	KOH	LN+S+HA	-	T.corp
49	25	HW	G2	P2	L1	A0	3	31weeks	Pigmentation	-	LN+HA+V	-	-
50	20	Factory W	G1	P1	L0	A0	2	21weeks	Itchy lesions	-	Hirs+HA	-	Intertrigo
51	22	Farmer	G1	P1	L0	A0	2	22weeks	Itchy lesions	-	GH+LN	PUPPP	-
52	31	HW	G4	P3	L2	A1	3	31weeks	Pigmentation	WL	Chloasma+LN+O	-	-
53	24	Self emp	G2	P2	L1	A0	3	30weeks	Strech marks	-	S+LN	-	-
54	23	HW	G1	P1	L0	A0	2	23weeks	Pigmentation	WL	Chloasma	-	-
55	25	HW	G2	P2	L1	A0	3	30weeks	Facial hair	-	Hirs	-	-
56	29	HW	G3	P3	L2	A0	2	27weeks	Itchy lesions	-	S+PE	PUPPP	-
57	30	Office	G3	P1	L1	A1	2	14weeks	Itchy lesions	KOH	-	-	T.corp
58	27	Farmer	G3	P2	L2	A0	2	18weeks	Strech marks	-	S	-	-
59	21	Teacher	G1	P1	L0	A0	2	24weeks	Pigmentation	WL	Chloasma	-	-
60	24	HW	G1	P0	L0	A0	2	15weeks	Pigmentation	-	HA	-	-
61	32	HW	G3	P3	L2	A0	3	32weeks	Asymtomatic	-	LN+S+HA+O	POP	-

									lesions				
62	22	HW	G1	P1	L0	A0	3	29weeks	Aymptomatic lesions	WL	LN+S+V	-	P.versicolor
63	21	Self emp	G1	P0	L0	A0	2	17weeks	Pimples	-	-	-	AV II
64	24	HW	G1	P1	L0	A0	3	29weeks	Itchy lesions	KOH	HA+LN	-	T.cru
65	27	Self emp	G3	P2	L1	A1	2	25weeks	Pimples	-	LN+DO	-	AV II
66	31	Self emp	G3	P2	L2	A0	2	13weeks	Itchy lesions	KOH	-	-	T.corp
67	22	Factory W	G1	P0	L0	A0	2	15weeks	Strech marks	-	S	-	-
68	27	HW	G3	P3	L1	A1	2	22weeks	Itchy lesions	-	PE	PFP	-
69	20	HW	G1	P0	L0	A0	2	15weeks	Pigmentation	-	HA	-	-
70	30	Teacher	G2	P2	L1	A0	3	32weeks	Asymtomatic lesions	-	LN+S+HA+O	PP	-
71	24	HW	G1	P1	L0	A0	3	29weeks	Asymtomatic lesions	WL	LN+S	-	P.versicolor
72	25	Self emp	G1	P0	L0	A0	2	17weeks	Pimples	-	-	-	AV II
73	21	Farmer	G1	P1	L0	A0	2	27weeks	Fluid filled lesions	Tzanck smear	S+LN+HA	-	Varicella
74	23	HW	G1	P1	L0	A0	2	24weeks	Scaly lesions	-	LN+HA	-	Seb dermatitis
75	30	HW	G4	P4	L1	A2	2	29weeks	White patches	-	S+SH	-	Vitiligo vulgaris
76	24	Farmer	G1	P0	L0	A0	2	17weeks	Strech marks	-	S	-	-
77	25	Self emp	G1	P1	L0	A0	2	24weeks	Pigmentation	WL	Chloasma	-	-
78	22	Farmer	G1	P0	L0	A0	2	17weeks	Painful lesions	-	HA	-	Herpes labialis
79	29	HW	G2	P2	L1	A0	3	31weeks	Asymtomatic lesions	WL	LN+S+HA+O	-	P.versicolor
80	23	HW	G1	P1	L0	A0	3	28weeks	Itchy lesions	-	LN+S	PUPPP	-
81	31	Teacher	G2	P2	L1	A0	3	29weeks	Itchy lesions	KOH	LN+HA	-	T.corp
82	27	Farmer	G2	P2	L1	A0	2	20weeks	Pigmentation	WL	Chloasma	-	-
83	22	Office	G1	P0	L0	A0	2	14weeks	Asymtomatic lesions	-	-	-	Acrochordons
84	26	Factory W	G2	P1	L1	A0	2	18weeks	Fluid filled lesions	Tzanck smear	HA	-	Varicella
85	21	HW	G1	P1	L0	A0	3	29weeks	Itchy lesions	WL	LN+Chloasma	PUPPP	-
86	20	Self emp	G1	P0	L0	A0	2	13weeks	Itching	-	-	PP	-
87	24	HW	G2	P2	L1	A0	3	32weeks	Itchy lesions	-	LN+HA+S+V	POP	-
88	30	Self emp	G4	P3	L2	A1	3	29weeks	Pigmentation	WL	Chloasma	-	-
89	19	Farmer	G1	P1	L0	A0	2	22weeks	Itching	-	S	POP	-

90	21	HW	G1	P1	L0	A0	2	22weeks	Itchy lesions	-	Hirs+HA	EP	-
91	31	HW	G3	P2	L2	A0	2	13weeks	Itchy lesions	-	-	PUPPP	-
92	23	Self emp	G1	P0	L0	A0	2	15weeks	Strech marks	-	S	-	-
93	26	Farmer	G1	P1	L0	A0	3	29weeks	Itchy lesions	WL	LN+S	-	P.versicolor
94	20	Factory W	G1	P0	L0	A0	2	15weeks	Pigmentation	WL	Chloasma	-	-
95	27	HW	G2	P2	L1	A0	3	32weeks	Itchy lesions	-	LN+S+HA+O	-	Resolved Varicella
96	24	HW	G1	P0	L0	A0	3	19weeks	Itchy lesions	-	PE+Chloasma	POP	-
97	19	Self emp	G1	P1	L0	A0	2	20weeks	Strech marks	-	S	-	-
98	22	Office	G1	P1	L0	A0	2	21weeks	Pigmentation	WL	Chloasma	-	-
99	27	Farmer	G3	P2	L2	A0	2	13weeks	Itchy lesions	-	-	PUPPP	-
100	25	HW	G2	P2	L1	A0	3	31weeks	Itchy lesions	KOH+WL	LN+S+Chloasma	-	T.corp
101	28	HW	G4	P3	L2	A1	3	22weeks	Itchy lesions	-	S	POP	-
102	22	HW	G1	P0	L0	A0	2	17weeks	Itchy lesions		-	EP	-
103	24	HW	G1	P0	L0	A0	2	15weeks	Asymptomatic lesions	WL	HA	-	P.vericolor
104	26	Farmer	G1	P1	L0	A0	3	29weeks	Itchy lesions	KOH	LN+S+HA	-	T.cru

ANNEXURE -V

KEY TO MASTER CHART

Occupation	
HW	Housewife
Self emp	Self employed
Factory W	Factory worker
Investigation	
WL	Woods lamp
KOH	Potassium hydroxide
Physiological skin changes	
LN	Linear nigra
HA	Hyperpigmentation of areola
Hirs	Hirsutism
SH	Subungual hyperkeratosis
DO	Distal onycholysis
S	Striae gravidarum
PE	Palmar erythema
V	Varicosities
O	Non pitting oedema
PG	Pyogenic granuloma

GH	Gingival hyperplasia
Specific dermatoses	
PP	Pruritus of pregnancy
PUPPP	Pruritic urticarial papules and plaques of pregnancy
POP	Prurigo of pregnancy
PFP	Pruritic folliculitis of pregnancy
EP	Eczema in pregnancy
Other skin conditions	
T.corp	Tinea corporis
T.cru	Tinea cruris
P.versicolor	Pityriasis versicolor
ICD	Irritant contact dermatitis
PMLE	Polymorphous light eruption
AVII	Acne vulgaris grade II
Seb dermatitis	Seborrheic dermatitis