“A HOSPITAL-BASED CROSS SECTIONAL STUDY ON CLINICO-EPIDEMIOLOGICAL STUDY OF ACNE VULGARIS IN TERTIARY CARE HOSPITAL”

Submitted by

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DISSERTATION SUBMITTED TO THE
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In partial fulfillment of the requirements for the degree of

M. D

in

DERMATOLOGY, VENEREOLOGY AND LEPROSY

Under the guidance of

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I hereby declare that this dissertation entitled **"A HOSPITAL-BASED CROSS SECTIONAL STUDY ON CLINICO-EPIDEMIOLOGICAL STUDY OF ACNE VULGARIS IN TERTIARY CARE HOSPITAL"** is a bonafide and genuine research work carried out by me under the guidance of **DR.ARUN C. INAMADAR**M.D, D.V.D., Professor and Head, Department of Dermatology, Venereology and Leprosy at BLDE (Deemed to be university) Shri B. M. Patil Medical College Hospital and Research Centre, Vijayapur.

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LIST OF ABBREVIATIONS

IL-1	-	Interleukin-1
TNF-α	-	Tumor necrosis factor-α
LTB4	-	Leukotriene B4
PMNL	-	Polymorphonuclear leucocyte
P.acne	-	*Propionobacterium acne*
IgG	-	Immunoglobulin G
TLR2	-	Toll like receptors 2
SHBG	-	Steroid hormone binding globulin
ACTH	-	Adrenocortico trophic hormone
BP	-	Benzoyl Peroxide

ABSTRACT

Background

Acne is one of the most common skin disorder worldwide irrespective of all ethnicities and races and occurs primarily at puberty with a prevalence of almost 95%. It is a chronic inflammatory disease of pilosebaceous unit characterized by non inflammatory open and closed comedones to inflammatory papules, pustules, nodules and sinus.

Objective

To determine the epidemiological and clinical aspects of acne vulgaris in a tertiary care hospital.

Methodology

Two hundred patients have been enrolled so far. Detailed history was taken including age, age and site of onset, duration of lesions, distribution of lesion and aggravating factors and clinical examination and grade of the acne (devised by Adityan *et al*.) was recorded. Hormonal irregularities were investigated in patients signs of hyperandrogenism like menstrual irregularities, hirsutism and acnthosis nigricans.

Result:

Among 506 patients included in the study 313(61.9%) were females and 193(38.1%) were males. The mean age among males and females was 20.1years and 19.6 years respectively. Grade 2 acne was seen in majority of patients 296(58.5%), while grade 4 was least common 27(5.3%). Face was commonest site involved. Topical steroid and summer exacerbation were main aggravating factors seen in 79(15.6) and 75(14.8%) patients respectively. Among 313 female patients

premenstrual flare was seen in 116(22.9%). Family history of acne seen12.5%. Menstrual irregularities was seen in 112 patients, hirsutism in 18 patients and acanthosis nigricans in 5 patients but raised laboratory markers of hyperandrogenism were observed in only 5 patients.

Conclusion

The study brings out the epidemiological and clinical aspects of acne vulgaris in a tertiary care hospital. Acne has a multifactorial etiology. It poses a dermatological and cosmetic problem in patients of acne vulgaris which can have a negative psychosocial impact on their life. Educating the patients about the aggravating factors and counseling about the role of positive family history may lead to reassurance of the patients

Keywords: Acne vulgaris, Epidemiology

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INTRODUCTION

Acne is one of the most common skin disorder worldwide irrespective of all ethnicities, races and occurring primarily at puberty with a prevalence of almost 95%.[1] It is a chronic inflammatory disease of pilosebaceous unit characterized by pleomorphic eruptions that includes non inflammatory open and closed comedones in mild cases, inflammatory papules, pustules, nodules and sinus formation in severe forms of acne.[2]

Face, back and chest are the most common sites involved.[2] On the face, most frequently involved site is cheeks followed by forehead, nose and chin.[3] The condition usually starts in adolescence, peaks at the age of 14 to 19 years and frequently resolves by mid-twenties.[2] A mild form of acne is also seen at birth which may continue into neonatal period which is related to maternal hormones stimulating the pilosebaceous unit.[4] Although acne vulgaris develops early and lasts longer in females, it is more common and severe in males.[2]

Acne vulgaris has a multi-factorial etiology. Precipitating factors include genetics, diet, drug intake, sun exposure, seasonal variation, use of cosmetics, stress, pregnancy and exposure to industrial compounds like chlorine.[5]

The Pathogenesis of acne vulgaris include [6]:-

1. Inflammation
2. Sebaceous hyperplasia and seborrhea
3. Altered cornification and differentiation
4. Colonization of duct by microbial flora.

There is no difference in pathogenesis of acne based on the skin type of each individuals.[7] Postinflammatory hyperpigmentation is the main cause of worry among acne patients of skin of color.[7] Another important complication is scarring.

Although acne is considered to be merely a cosmetic problem, it is associated with great psychological impairment like low confidence, anxiety, depression, obsessive compulsiveness, self-conciousness, poor self image and sometimes suicidal ideation in patients as compared to same age individuals without acne, mainly due to its severity.[8,9]

Acne vulgaris is a very common disease and may lead to emotional and psychological disturbances. There are very few studies on its prevalence and epidemiology in Indian literature. The main aim of this study is to determine the epidemiological and clinical aspects of acne vulgaris in a tertiary care hospital.

OBJECTIVE

To determine the epidemiological and clinical aspects of acne vulgaris in a tertiary care hospital.

REVIEW OF LITERATURE:

Acne is one of the most commonly encountered dermatosis in general population worldwide irrespective of ethnicity and race. It primarily occurs in adolescents and young adults with a prevalence of almost 95%.[1] It is one of the easily diagnosable disorder.

Though it is a common physiological process, it is considered as a disease because of its inflammatory component and subsequent disfigurement produced on the face, which has social and psychological complications.[5]

Acne vulgaris is defined as a chronic inflammatory disease of the pilosebaceous unit characterized by non inflammatory open and closed comedones and/or inflammatory papules, pustules and nodules of varying degree of inflammation and depth.[2]

Dreno *et al* defined severity of acne:[10]

- Physiological acne → was defined as 1–4 papulo-nodules or pustules on the face.
- Clinical acne → was defined as more than 5 pustules or papulo-nodules on the face.

Face, back and chest are the most frequently involved sites.[2] On the face, most frequently involved site is cheeks, followed by forehead, nose and chin.[3] Even though comedones are non-inflammatory lesions, they exhibit subclinical signs of inflammation.[11]

The earliest acne lesion observed is a comedone that would progress into an inflamed tender papule, further progressing to pustules, nodules and cysts in severe form of acne.[11] However, some patients may only present with papules and pustules without comedones. Majority of patients will have both comedones and papules / pustules.[11]

Epidemiology:

Acne vulgaris is one of the most common skin disorder worldwide, affecting all ethnicities and races.[2] It most commonly occurs in adolescence where it is present in 95% of all teenagers.[1] The peak incidence of acne vulgaris is between 14 and 17 years in females and 16-19 years in males.[6] Prepubertal development of acne in girls may be related to the accelerated biologic adrenarche indicating hormonal abnormality.[6]

Acne observed in younger patients is mostly non-inflammatory open and closed comedones as compared to adults.[12] As age increases, there is increased sebum production and blockage of pilosabeceous units which favors bacterial colonization.[12] This is followed by triggering immune response leading to development of inflammatory lesions such as papules, pustules, and nodules.[12] In young patients there is insufficient bacterial growth, hence, they present with follicular plugs and comedones.[12]

Acne vulgaris develops early and lasts longer in females than in males; however it is more common and severe in males, probably attributed to the activity of the androgens.[2] People with positive family history of acne present with higher prevalence of moderate to severe acne as compared to those with no family history.[13]

In some studies it was observed that acne was absent among people eating in their traditional manner, but upon westernization of diet, acne prevalence has increased.[13] Foods like sweets, nuts, chocolate and oily foods were considered to have increased the risk of acne vulgaris.[13]

Postinflammatory hyperpigmentation and scarring are the main complications observed in acne vulgaris patients.[14] There is direct relationship between scarring and duration of the disease.[14] The risk of post inflammatory pigmentation is more among patients Fitzpatrick skin type III and IV.[14]

In a study conducted by Adityan *et al*[15] in 309 acne patients, majority were between the age group of 16-20 years, among whom 137 were females. Male patients had more severe grade of acne as compared to females. Face was the commonest site of involvement. Most common type of lesions were closed comedones present in all patients i.e majority of them had grade 1 acne. Post acne scarring was seen in 122 patients with most commonly involved site being cheeks with ice pick scar being the commonest type of scar.

Patil *et al*[5] analysed 120 cases in which 69 patients were females. Most of the patients were between the age group of 16-20 yrs, with maximum number of patients with disease duration of 1-2 yrs. Fifty four patients were using topical cosmetics and 60 patients had exacerbation during periods of stress. A total of 80 patients had lesions over the face followed by lesions on the chest and back. Scarring was noted in 32 patients out of whom 12 had ice pick scars.

Khunger *et al*[1] analysed 280 patients of acne vulgaris with high female preponderance and the prevalence of adult acne was 0.38%. The mean age of the patients was 30.5 yrs with a range of 26-50 years. Majority of the patients had grade 2 acne mostly distributed on the face. Topical steroid was the main aggravating factor in 11.8% of the patients. Family history of acne with first degree relative was found in 108 (38.6%) patients. Summer exacerbation was noted in 36.4% of the cases. Regular use of cosmetics (25.7%), stress (25.7%) and premenstrual flare (11.7%) were noted to aggravate acne.

Sharma *et al*[16] analysed 1032 cases in which male predominance(73.2%) was observed as compared to females (71.1%). Most of the patients were between the age group of 14-16yrs. Earlier onset of acne was seen in girls compared to boys. A total of 71.4% patients had lesions over the face followed by lesions on the chest and back. Most common type was grade 1 acne(81.9%) followed by moderate and severe acne. Summer exacerbation and premenstrual flare was seen in 21.1% and 9% respectively. Patients had exacerbation during periods of stress.

In a study conducted by Dreno *et al*[10] in 409 acne patients, majority were of the age group of 16 years, with high female preponderance. Study showed patients with positive family history had an increased risk of developing acne vulgaris. Face was the commonest site of involved. Post acne scarring was observed more commonly among people with Fitzpatrick skin type I and II.

The 1002 acne patients studied by Ghodsi *et al*[13] showed high female preponderance with majority patients between the age group of 12-20years and mean age of 16.5years. The mean age of moderate/severe acne was 16.3years which was comparatively higher than that of mild acne (15.7 years). Patients with a positive

family history of acne showed a higher prevalence (19.9%) of moderate/severe acne. Premenstrual flare and mental stress were considered to be positively associated with severity of acne. Dietary habits such as nuts, chocolates and oily foods were also associated with increased risk of development of acne.

Duquia *et al* [11] analysed 2,264 adolescents who were enlisted in the army among which individuals with acne were 1960 patients. Of these patients, only comedones were present in 161 patients, inflammatory lesions in 404patients and both in 1,395 patients. Patients with light skin type had 9% higher probability of developing acne as compared to dark skin type. Patients who consumed yogurt daily had 5% higher probability of acne compared to non-consumers.

In a study conducted by Bagatin *et al* [12] among 452 adolescents examined, 434 individulas had acne with the prevalence of 96%. There was no sex predilection observed. Prevalence of acne increased with age, majority were of age over 14 years. Most common type of lesions were comedones(61.1%) followed by mild and moderate grade acne(6.1%). Face was the commonest site of involvement. Positive family history of acne in mother or father was present in about half of the adolescents. This study revealed there was no significant difference among gender, ethinicity and smoking.

In a Chinese study by Yeung *et al*[14] analysed 552 patients of acne vulgaris with higher male preponderance(53.6%) than females(50.8%) but difference observed was negligible. Most of the patients were between the age group of 15-20yrs(55.9%) followed by of 21-25years(43.5%). Acne scarring and pigmentation were the complications observed which were more common in females than in males.

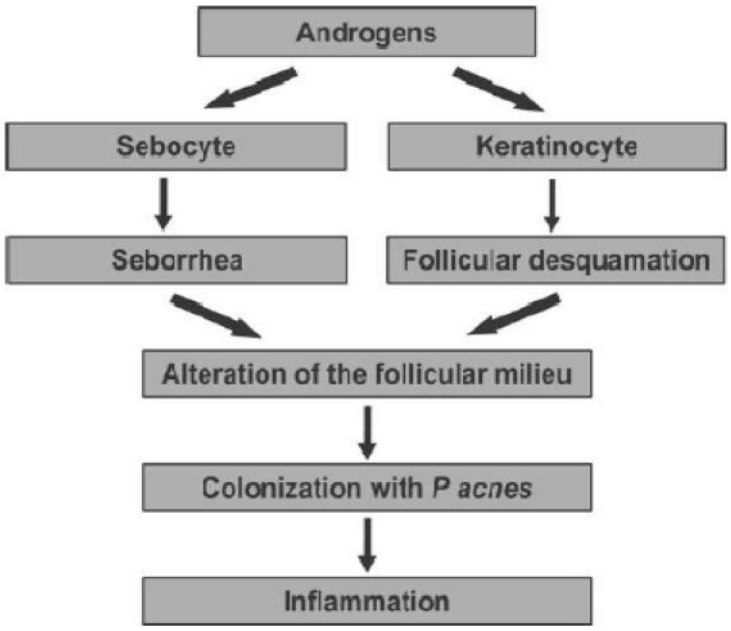
Etiopathogenesis :

Acne vulgaris is a multifactorial disease. It results from combination of various factors. They are as follows[17]:-

Increased sebaceous gland activity with seborrhea

1. Abnormal follicular differentiation with increased keratinization
2. Microbial hypercolonization of the follicular canal and
3. Increased inflammation primarily through activation of the adaptive immune system.

Pathogenesis of acne:[17]



Out of above mentioned factors, abnormal follicular differentiation with increased keratinization, increased sebaceous gland activity and seborrhoea are the most important, as they combine to produce a microcomedo, which is the primary lesion of acne.[17] The microcomedo can develop into either a non-inflammatory comedo or become inflamed and present as a papule, pustule, or nodule.[17]

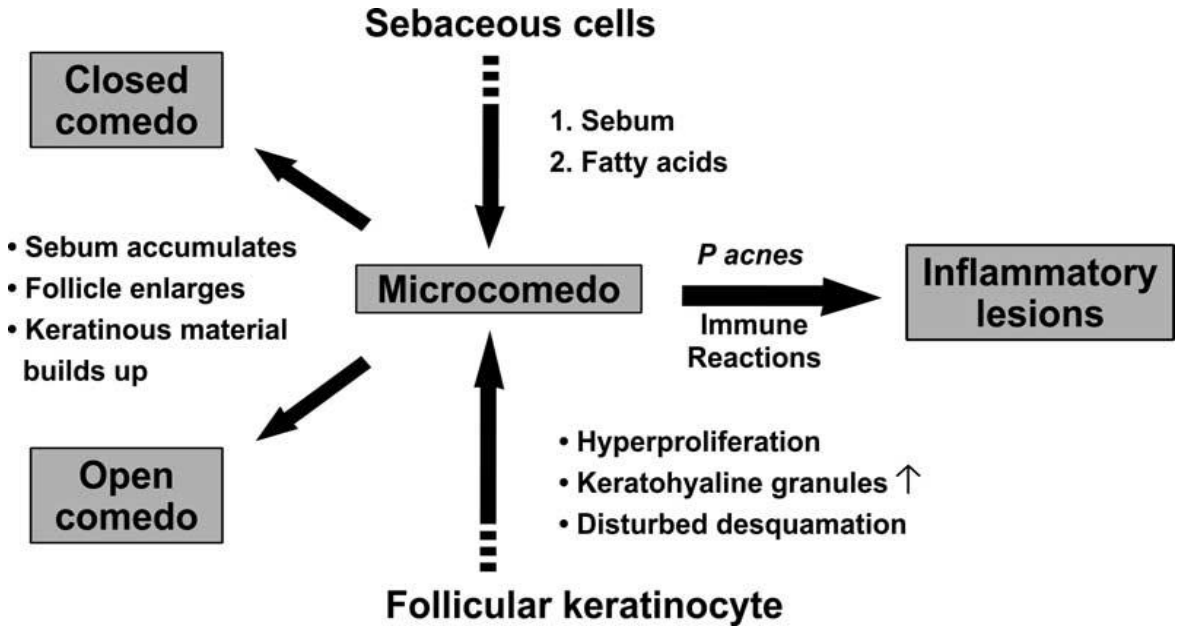
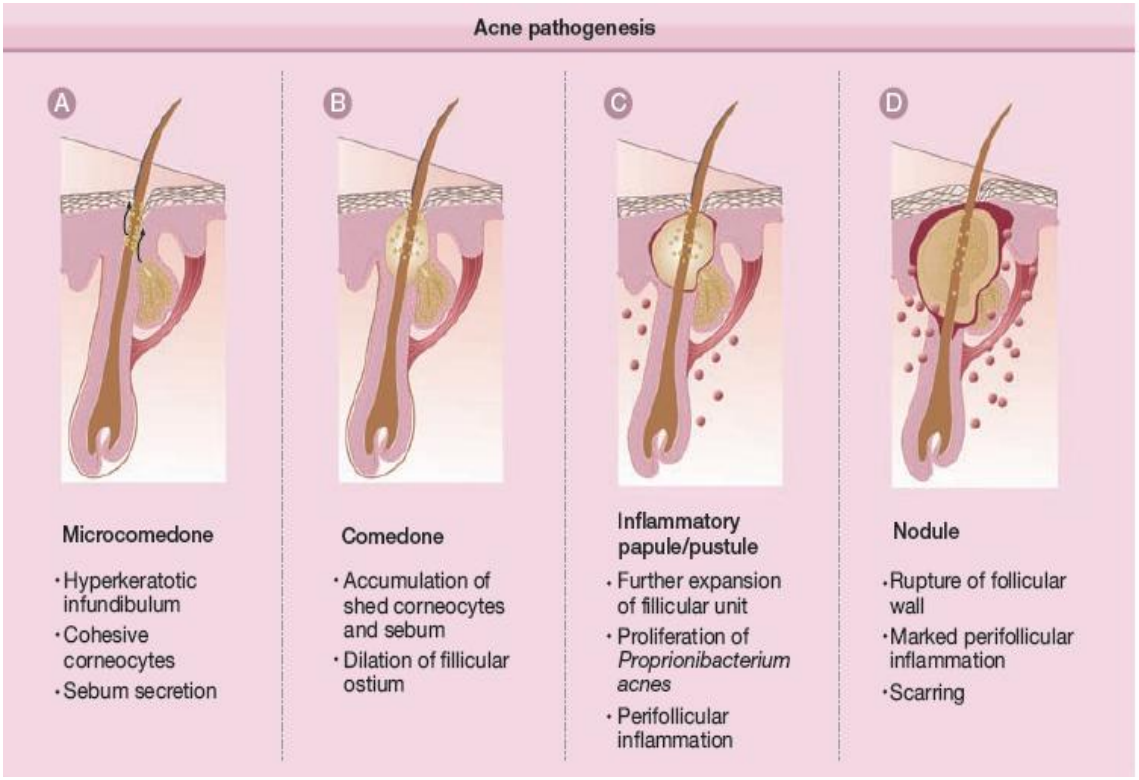


Figure 1: Pathogenesis of acne[4]



The factors affecting severity of acne are:

1.Inflammation: It is hypothesized that there is increase in interleukin-1 (IL-1) which precedes hyperkeratinization of the follicular duct.[2] The release of IL-1 and keratinocyte markers K6 and 16 cause activation of keratinocytes.[6] The ILs, tumor necrosis factor(TNF)-α and complement fractions increase synthesis of leucotriene B4 (LTB4) which further induce and activate polymorphonuclear leucocytes (PMNL) and monocytes.[6] The antibody titre of *Propionobacterium acne* (*P. acne)* is increased which promotes the release of lysosomal hydrolases by PMNL.[6] It is found that there is increased immunoglobulin(Ig) G1 and IgG3 in moderate acne and IgG2 in severe acne in response to *P. acne*.[6] It is also demonstrated that *P. acne* activates toll like receptors 2 (TLR 2) which triggers the release of the inflammatory cytokines.[6]

2. Sebaceous hyperplasia and seborrhea: There is increased sebum secretion, sebaceous gland hypertrophy and hyperplasia in patients with acne vulgaris.[6] These patients have an increased level of squalene, wax esters and fatty acid in their sebum. There is also a decrease in the level of linoleic acid. All of these factors promote accumulation of cornified cells.[6]

3. Altered cornification and differentiation:

Increase in number of keratinocytes in follicular infuldibulum

Retention hyperkeratosis

Microcomedone formation

Comedones becomes permeable to inflammatory substances due to hyperkeratinization of follicular epithelium. This decreases the level of ceramides which further decreases the barrier function.[6]

4. Colonization of duct by microbial flora: *P. acne* is an inhabitant of normal skin flora. In acne vulgaris, there is increase in the density of *P. acne*[2]

Increased *P. acne* flora.

⇩

Produce lipase

⇩

Sebaceous gland triglyceride ⟶ free fatty acid

hydrolysis

⇩

Follicular hyperkeratinization and rupture of follicles

Other factors:

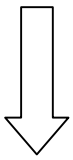
i. **Genetics :** Acne is more commonly heritable in a first degree relative and is more severe in patients with positive family history.[18] It is mainly the common environmental factor that affects the severity of acne.[9] Prevalence was high when mother had acne followed by father and siblings.[13] There are several reports of monozygotic twins developing acne more commonly and of severe form compared to dizygotic twins.[18]

Goulden *et al*[18] states that genetic factors may determine abnormal follicular keratinization or sebaceous gland hyperplasia due to androgen response in individuals

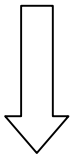
with persistent acne. Autosomal inheritance pattern was seen in a family with multiple comedones providing a evidence that abnormal follicular keratinization may be inherited.[18]

ii. **Diet:** It is found that people who consume foods with high sugar and fat content are more prone for developing acne.[7] It is also proved that increased intake of high glycemic diet like milk is associated with increased risk of acne.[6]

Increase in glycemic load



Hyperinsulinemia



Endocrine response stimulates unregulated tissue growth and

enhanced androgen synthesis.[19]

Therefore, diet inducing hyperinsulinemia may represent an environment that triggers development of acne due to their androgen influence on follicular epithelial growth and keratinisation as well as increased sebum secretion.[19] Diet with low glycemic load foods reduced serum androgen levels, fasting glucose levels, and improved insulin metabolism and increased concentrations of Steroid hormone binding globulin(SHBG).[19,20]

iii. **Smoking:** Severity of acne is higher in smokers as compared to non-smokers.[9] Factors which can play an important part in development of acne in smoker are:[21]

- Impaired vaso-reactivity
- Relative ascorbic acid deficiency and
- Impaired collagen synthesis and wound healing.

Acne is also directly proportional to stress and as people may tend to smoke when they are stressed, it can also be one of the reasons for increased acne in smokers.[21] Thus, stress might be a confounding variable in the association of acne and smoking.[21]

iv. **Stress and sleep deprivation:** Stress and sleep deprivation are also risk factor for acne.[2] A study observed reduction in severity of acne on adopting relaxation techniques on stress reduction.[22] Even recurrence of open and closed comedones, when relaxation technique were in use reduced.[22]

v. **Pre-menstrual flare:** Acne lesions flare up 2-7 days prior to the onset of menstruation.[2]

vi. **Climatic changes:** Severity of acne increase in summer and humid climate. UV radiation also enhances the sebum comedogencity.[6]

vii. **Cosmetics:** Use of oils, pomades and other comedogenic chemicals increase the acneiform eruption.[2]

Clinical features:

Acne mainly occurs around the period of puberty. It predominantly involves the face, back, chest, shoulders and upper arm.[9] It includes inflammatory and non-inflammatory lesions.

Non-inflammatory lesions: Comedones are the non-inflammatory lesions of acne resulting from plugging of keratin and sebum in dilated pilosebaceous unit which is the earliest presentation in young patients.[2] Comedones are mainly of two types:

1. *Closed comedones or whiteheads*: They have no visible follicular opening and are generally 1mm in diameter with skin colored lesions.[2]

2. *Open comedones or blackheads*: Represent visible follicular opening with a wide dilated orifice.[6]

The other sub-types of comedones are:

- Sandpaper comedones: are type of closed comedones characterized as multiple small lesions which give a roughened, gritty feel to the skin, seen especially on the forehead.[2]

- Macrocomedones : are larger than 1mm in diameter. They respond poorly to the treatment.[2]

- Submarine comedones : are larger than 0.5cm in diameter and situated deeply in the skin. They are also associated with the recurrent inflammatory lesions.[2]

- Secondary comedones : Exposure to topical steroids, pomade (pomade acne), chemical (chloracne), thick oils and other drugs causes secondary comedones.[2]

The inflammatory lesions are papules, superficial pustules (<5mm in diameter) or deep seated pustules and nodules (>5mm in diameter).[2] Papules and pustules are largely distributed on face and back.[2] These lesions arise from the non-inflammatory lesions which may remain superficial or become deep seated in nature.[2]

The various clinical variants of acne vulgaris are presented in Table 1.

Table 1: Clinical variants of acne [2]

S.No	Types	Salient features
1.	**Drug induced acne**	Corticosteroids, ACTH, anticonvulsants, antidepressants, antitubercular, antiviral, calcium antagonists, vitamins and anti psychotic drugs induce acne.[2]
2.	**Cosmetic acne**	Cosmetics containing comedogenic substances like lanolin, petrolatum, vegetable oils, butylstearate, lauryl alcohol, oleic acid. Also caused with the use of pomade and alkaline detergent.[2]
4.	**Occupational acne**	Due to exposure to polyhalogenated oraganic compounds containing naphthalene, biphenyls and phenols .[2]
5.	**Severe forms** – a)Acne fulminans	Tender inflammatory ulcerative nodules with hemmoragic crusts. Commonly distributed on back, upper chest and shoulders. More common in males aged between 13-22yrs.[2]
	b) Acne conglobata	Multiple comedones and inflammatory papules, pustules, tender nodules, abscess and draining sinus tracts. Common in males of age 20-30yrs.[2]
6.	**Acne associated with pyschosis problems** – a)Acne excoriee	Commonly seen in adolescent girls who picks real or imaginary acne mostly on face.[2]
	b)Body dysmorphic disorder	Patients are often depressed, or have obsessive compulsive disorder.[2]
	c)Eating disorder	Anorexia nervosa
7.	**Granulomatous acne**	Deep well demarcated lesions commonly on cheeks
8.	**Acne mechanica**	Due to repeated mechanical trauma and friction examples are fiddler's neck in violin players, headband and collars

Acne scoring systems – Acne scoring systems are used to assess the severity of acne. They include:[8]

- Lesion counting and
- Grading of acne.

Lesion counting – This is done by counting each type of acne lesions and recording them and determining the overall severity of acne.[5]

Grading of acne – It is a subjective method based on observing the dominant lesion, evaluating the presence or absence of inflammation and estimating the extent of involvement to determine the severity of acne.[7]

The different grading systems of acne vulgaris are listed in Table 2.

Table 2: Grading systems of acne vulgaris [23]

Scales	Grading methods
1. Pillsbury, 1956	1-4 grades with text description.
2. Plewig and Kligman, 1975	Separate grade for comedonal and inflammatory acne.
3. Blaney and Cook, 1976	0-8 grades based on text description and standard photographs.
4. Michaelsson *et al*, 1977	By couonting the number of lesions on face.
5. Cook *et al*, 1979	0-9 grades for face, 0-8 grades for overall severity.
6. Liden *et al*, 1980	Summation of number of lesions of each type multiplied by weight score.
7. Allen and Smith, 1982	0-8 grades for overall severity, 0-8 grades for comedones with text description.
8. Burke and Cunliffe, 1984	0-10 grades with photographic examples of face, chest and back.
9. Gibson *et al*, 1984	0-8 grades for overall ac with text description, 0-8 grades for comedonal acne.
10. Samuelson, 1985	0-9 grades severity with text description
11. Pochi *et al*, 1991	Inflammatory acne is graded mild, moderate, severe and very severe depending on global evaluation of lesions and their complication.
12. Doshi *et al*, 1997	Total score determines the severity of grade
13. O'brien *et al*, 1998	12 grades for face, 8 chest, 8 back; 3 facial grades for non-inflammed acne based photographic images.
14. FDA Guidance, 2005	0-4 grades based on text description
15. Del rosso *et al*, 2007	Severity of chest and back based on numeric range of lesions for each primary lesions type.
16. Tan *et al*, 2007	0-5 grades based on text description, apllied to face,back and chest
17. Hayashi *et al* ,2008	0-4grades based on numeric range of papules/pustules per half face and reference photographs.
18. Dreno *et al*, 2011	0-5 grades based on text description similar to tan et al.

Adityan *et al*[11] has devised a simple grading system for acne vulgaris based on the predominant lesions present and is described in Table 3:

Table 3: Acne grading system of by Adityan *et al*.[24]

Grades	Lesions
Grade 1	Comedones, occasional papules
Grade 2	Papules, comedones, few pustules
Grade 3	Predominantly pustules, nodules and abscess
Grade 4	Mainly cysts or abscesses, widespread scarring

Investigation:

In general investigations are not required in acne patients. They are required to evaluate and/ or monitor underlying endocrine cause. Signs and symptoms that may indicate hyperandrogenism are:[2]

- Seborrhea
- Hirsutism
- Androgenic alopecia
- Cushingoid features

- Increased libido
- Deepening of voice
- Irregular menstrual cycle
- Acanthosis nigricans

In suspected hyperandrogenism investiagtions to be done are–

- Serum testosterone
- Serum prolactin
- DHEAS
- 17 hydrxyprogesterone acetate
- Fasting and postprandial blood insulin level
- Serum LH and FSH
- USG of pelvis

Treatment:

Treatment of acne vulgaris depends upon the severity. Various treatment strategies used to treat acne have been listed in Table 4.

Table 4: Treatment options for acne vulgaris

	Mild	Moderate	Severe
First line treatment	• Benzoyl peroxide (BP) 2.5% • Topical retinoid (tretinoin 0.025%) Or • Topical combination therapy • BP+ antibiotic or • Retinoid + BP or • Retinoid + BP + Antibiotic	• Topical combination therapy • BP + antibiotic or retinoid + BP + antibiotic Or • Oral antibiotic + topical retinoid + BP Or • Oral antibiotic + topical retinoid+ BP +topical antibiotic	• Oral antibiotic + topical combination therapy • BP + antibiotic Or • Retinoid + BP Or • Retinoid +BP+antibiotic Or • Oral isotretinoin
Alternative therapy	• Add topical retinoid or BP (if not on already) Or • Consider alternative retinoid Or • Consider topical dapsone	• Consider alternative combination therapy Or • Consider change in oral antibiotic Or • Add combined oral contraceptive or oral spironolactone (females) • Consider oral isotretinoin.	• Consider change in oral antibiotic Or • Add combined oral contraceptive or oral spironolactone (females) • Consider oral isotretinoin

METHODOLOGY

7.1. SOURCE OF DATA :

Patients with acne vulgaris attending the outpatient department of Dermatology, Venereology and Leprosy of B.L.D.E (deemed to be university), Shri. B.M. Patil Medical College Hospital and Research Centre, Bijapur, were enrolled for the study.

Period of study:

The study was conducted during the period of September 2016 to August 2018

Study design:

A hospital based cross-sectional study.

7.2 METHOD OF COLLECTION OF DATA:

A total of 506 (n-200) patients of acne vulgaris were enrolled for the study. Informed written consent was taken from all study subjects.

Inclusion criteria:

1. All the patients with acne vulgaris aged >10 years of both gender.

Exclusion criteria:

1. Patients with acne vulgaris who were not-willing to participate in the study.

Methods:

Detailed history was taken from the patients including age, profession, age and site of onset, duration of lesions, distribution of lesion, family history and aggravating factors like: any drugs used, sun exposure, seasonal variation, diet, use of cosmetics, premenstrual flare, stress and habits.

Clinical examination of the lesions including type of lesions, distribution of the lesions and grade of the acne were recorded. The lesions were graded by the grading system devised by Adityan *et al*.[11]

Signs of hyperandrogenism were also noted.

7.3: INVESTIGATIONS:

Only in patients with signs and symptoms suggestive of hyperandrogenism like acanthosis nigricans, hirsutism and irregular menstrual cycles, following investigations were carried out

- FSH
- LH
- FSH:LH ratio
- USG of pelvis to rule out PCOS

7.4: STATISTICAL ANALYSIS:

All characteristics were summarized descriptively. For continuous variables, the summary statistics of mean, standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries. Chi-square (χ^2)/ Freeman-Halton Fisher exact test was employed to determine the significance of differences between groups for categorical data. The difference of the means of analysis variables between two independent groups was tested by unpaired t test. The t test (also called Student's T Test) compares two averages (means) and tells if they are different from each other. If the p-value was < 0.05, then the results were considered to be statistically significant otherwise it was considered as not statistically significant. Data were analyzed using SPSS software v.23.0. and Microsoft office.

7.5: HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION: YES

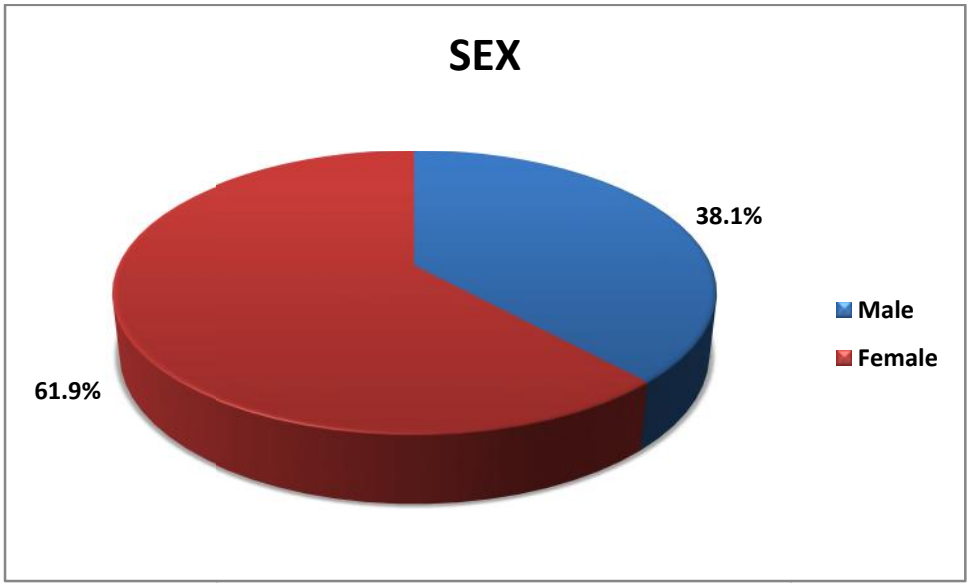
RESULTS

A hospital based cross sectional study was conducted from September 2016 to August 2018. A total of 506 acne patients with acne vulgaris were included in the study.

Sex distribution:

Of the 506 patients enrolled in the study, 313(61.9%) were females and 193(38.1%) were males. Female: male ratio was 1.62:1. Figure 2 represent the distribution of patients with acne vulgaris according to sex.

Figure 2: Distribution of cases according to sex



Age distribution

Among the 506 acne patients enrolled in the study, acne vulgaris was most commonly seen in the age group between 16-20years in 291 patients(57.5%) followed by 20-25years in 150 patients(29.6%), 11-15years in 39 patients(7.7%), 26-30years in 25 patients(4.9%) and >30years in 1 patient. Figure 3 represents distribution of acne vulgaris cases according to age. Mean age (±SD) among males and females was 20.2 (3.1) years and 19.6 (3.5) years respectively (*P*=0.035). Table 5 and Figure 4 represent mean age among males and females.

Figure 3: Distribution of cases according to age

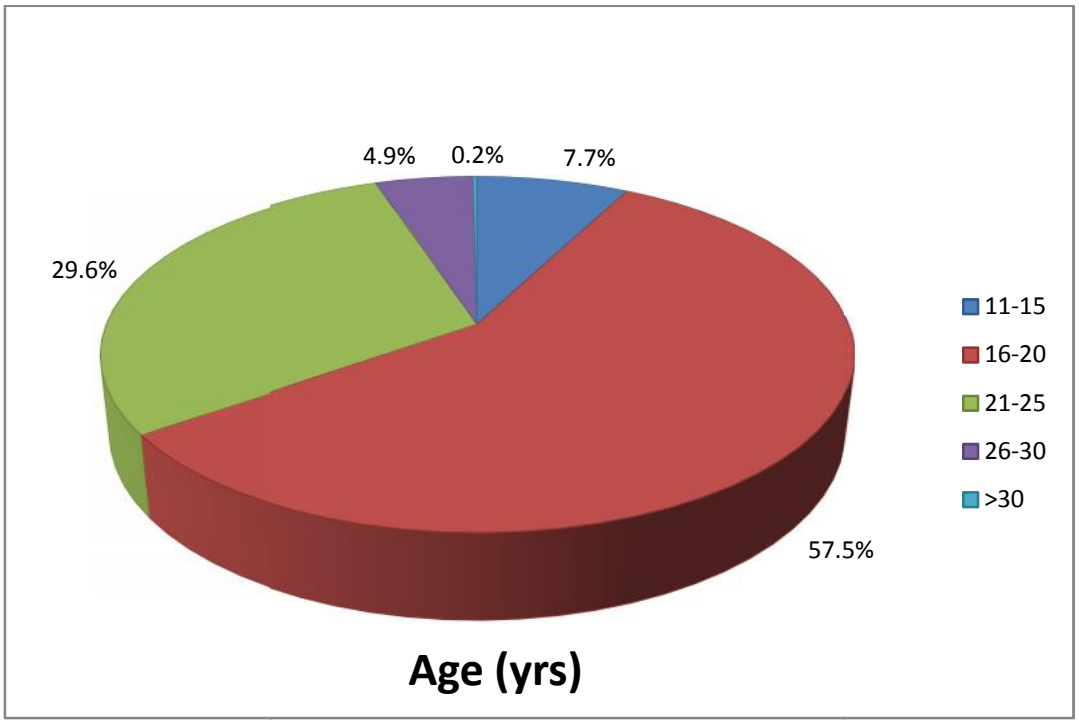
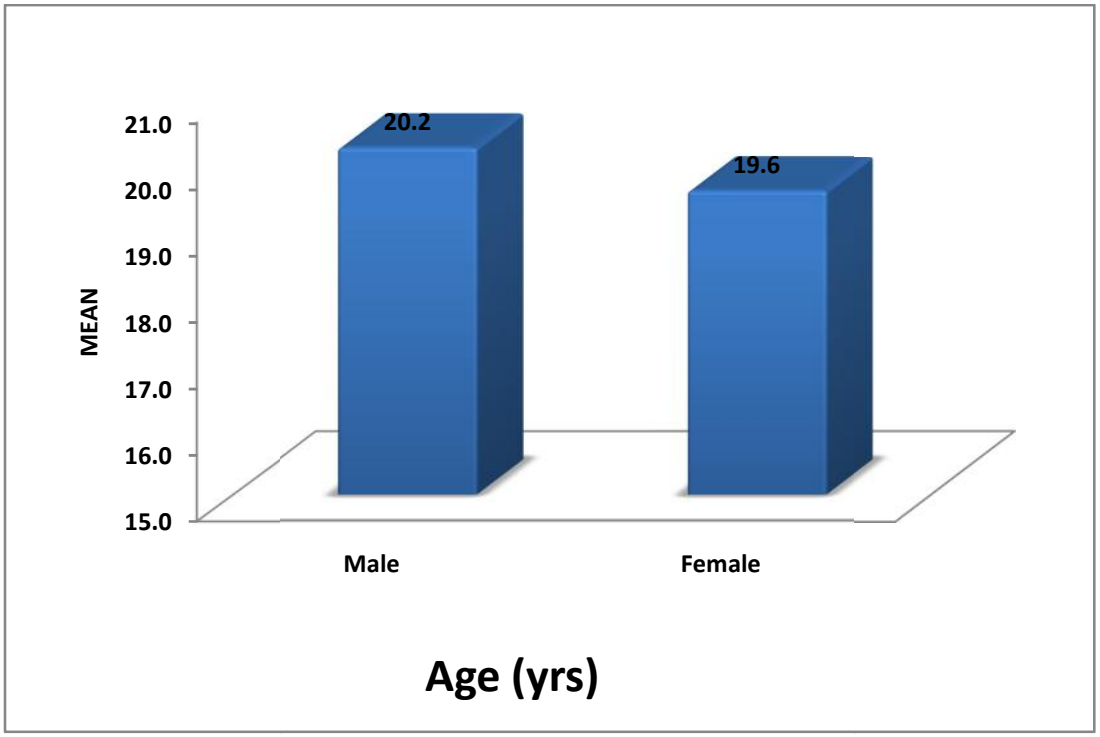


Table 5: Mean age of patients according to gender

	Male		Female		p value
Age (yrs)	**Mean**	**SD**	**Mean**	**SD**	
	20.2	3.1	19.6	3.5	0.035*

Note: * significant at 5% level of significance (p<0.05)

Figure 4: Mean age of patients according to gender



Age of onset:

Age of onset was most commonly between 16-20 years in 314 patients (62.1%) followed by 21-25 years in 103 patients (20.4%), 11-15years, 26-30 years in 77 patients (15.2%) and 11 patients (2.2%) respectively. Figure 5 represent distribution of cases according to age of onset.

Among 193 males enrolled in study, majority i.e., 112 patients (58%) initial presentation of acne was during age group of 16-20years followed by 54 patients(28%) in 21-25years, 20 patients(10.4%) in 11-15years and 7 patients(3.6%) in 26-30years. Among 313 female patients, 202 patients(64.5%) initial age of presentation was during age group16-20years followed by 57 patients(18.2%) in 11-15years, 49 patients(15.7%) in 21-25years, 4 patients(1.3%) in 26-30years and 1 patient(0.3%) in >30 years group. Figure 6 represents association of age with sex.

Mean age of onset (±SD) among males and females was 19.1 (3) years and 18 (3.2) years respectively (p=0.035). Table 6 and Figure 7 represent mean age of onset among males and females.

Figure 5: Distribution of cases according to age of onset

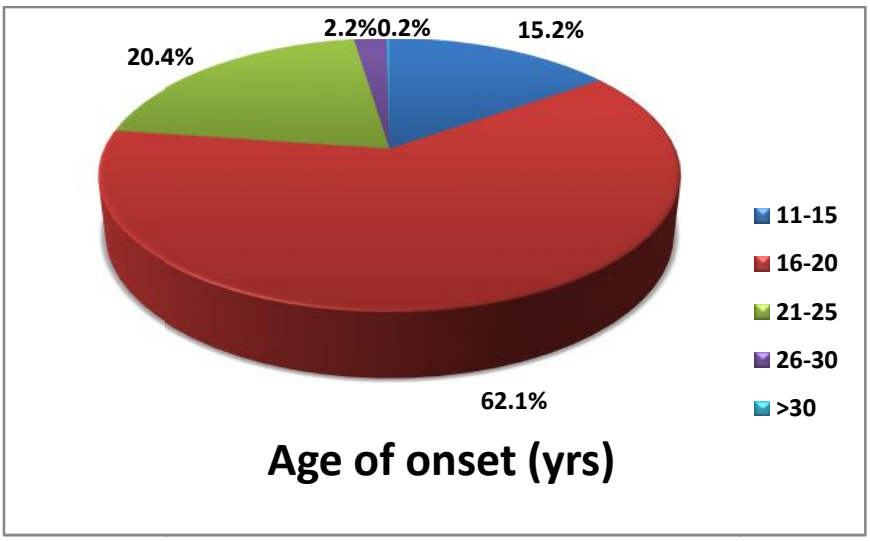


Figure 6: Association of age of onset and sex

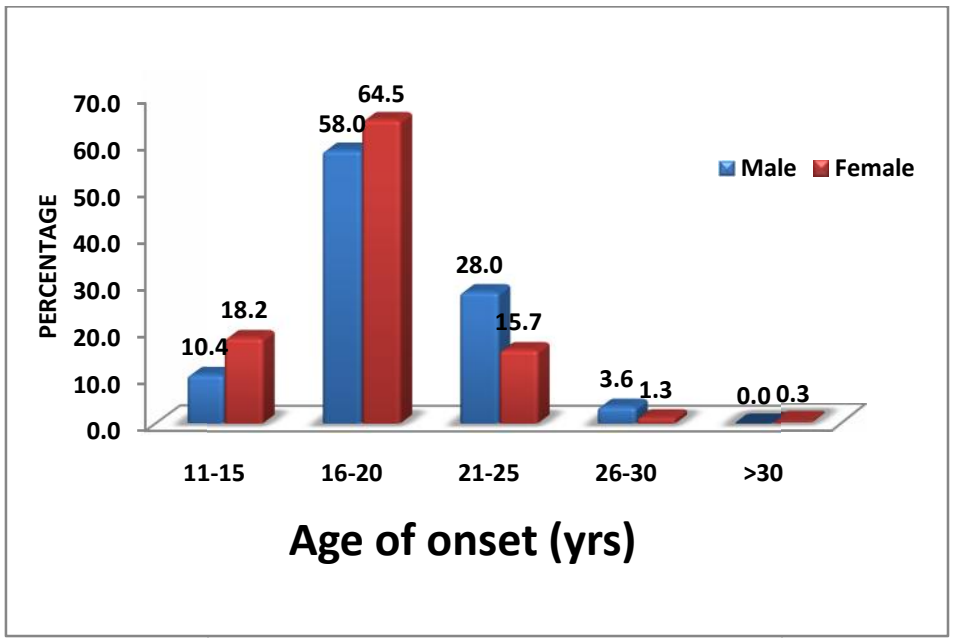
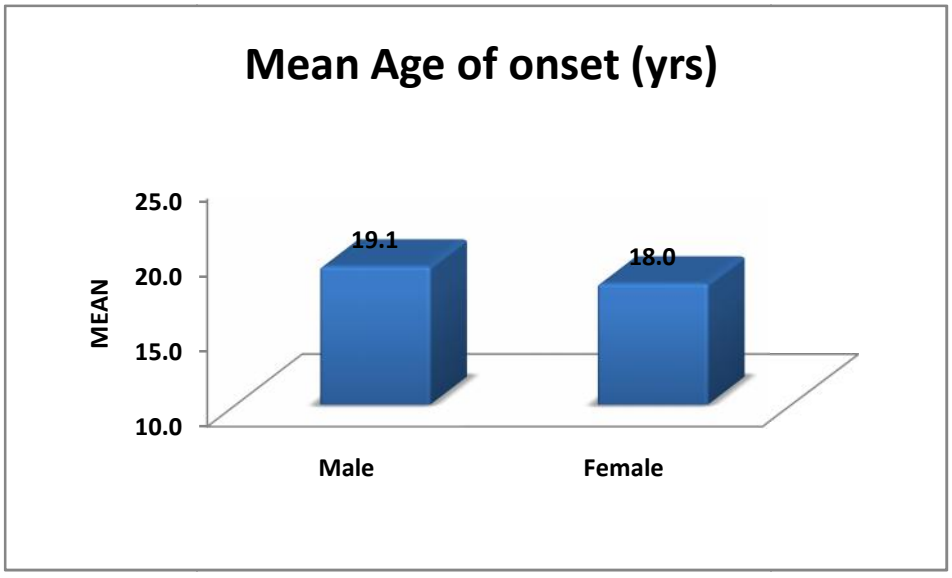


Table 6: Mean age of onset between males and females

	Male		Female		p value
	Mean	SD	Mean	SD	
Age of onset (yrs)	19.1	3.0	18.0	3.2	<0.001*

Note: * significant at 5% level of significance (p<0.05)

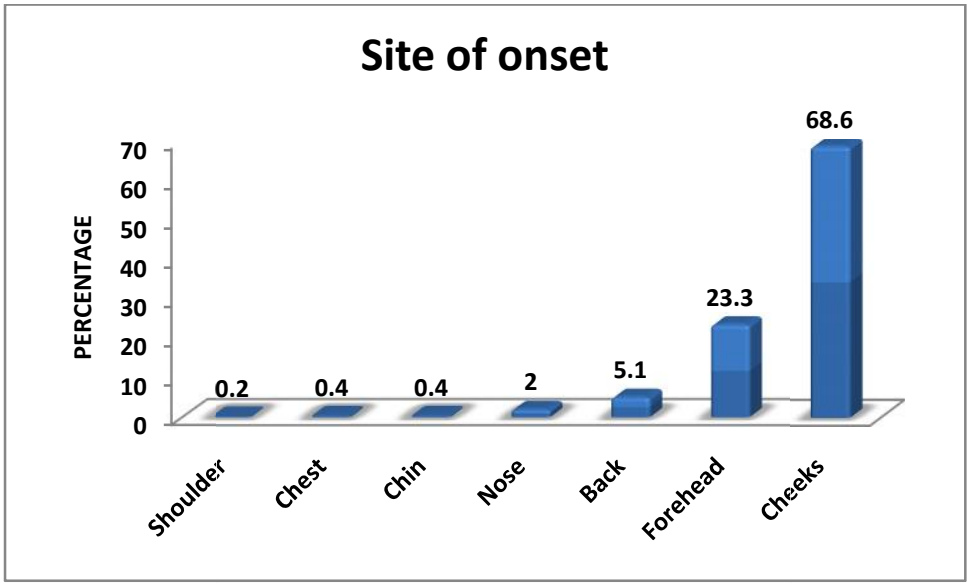
Figure 7: Mean age of onset in males and females



Site of onset:

Most common site of onset was cheeks in 347 patients (68.6%) followed by forehead in 118 patients(23.3%), back in 26 patients(5.1%), nose in 10 patients(2%), chin and chest in 2 patients each(4.4%) and shoulder in 1 patients(0.2%). Figure 8 represent most common site of onset.

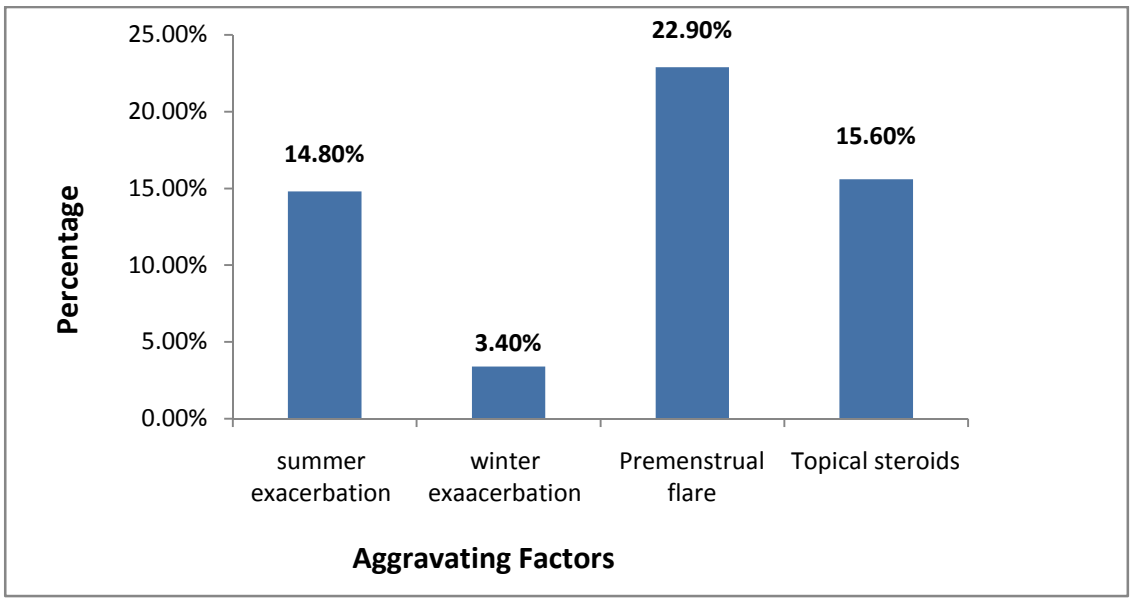
Figure 8: Distribution of cases according to site of onset



Aggravating factors:

Aggravating factors like seasonal variation, premenstrual flare and exacerbation after use of topical steroid application were observed. Summer exacerbation was seen in 75 patients (14.8%), winter exacerbation in 17 patients(3.4%), premenstrual flare in 116 patients(22.9%) and steroid application in 79 patients(15.6%). Figure 9 represents the aggravating factors among cases.

Figure 9: Aggravating factors among cases



Family history

Among the 506 patients, there was a positive family history in 63 patients, most commonly associated with mother in 40 patients (7.9%), followed by sister and father with 20 patients (4%) and 3 patients (0.6%) respectively. Figure 10 represents distribution of cases according to family history.

Patients with positive family history had higher grade of acne as compared to patients without positive family history. Patients who gave history of acne in mother presented with grade III in 18 patients(26.1%) followed by grade IV in 16 patients(59.3%) and grade II in 6 patients(2%).

Similarly patients with family history in sister had grade III acne in 14 patients(20.3%) followed by grade IV in 4 patients(14.8%) and grade II in 2 patients(0.7%). Cases who gave family history of acne in father had grade III in 2 patients(2.9%) and grade IV in 1 patient(3.7%). Figure11 represents distribution of family history according to grade of acne.

Figure 10: Distribution of cases according to family history of acne

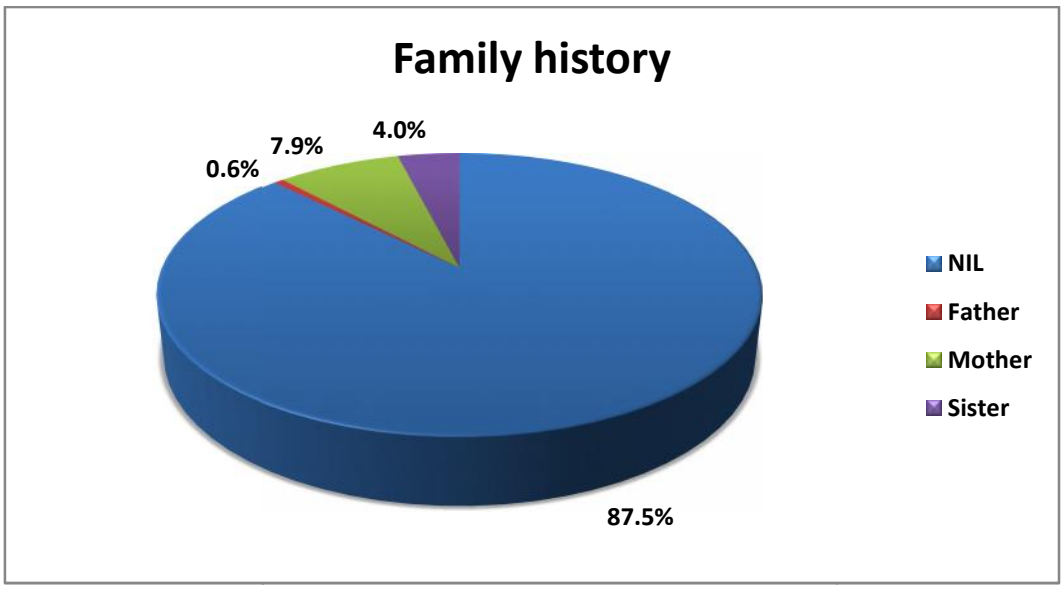
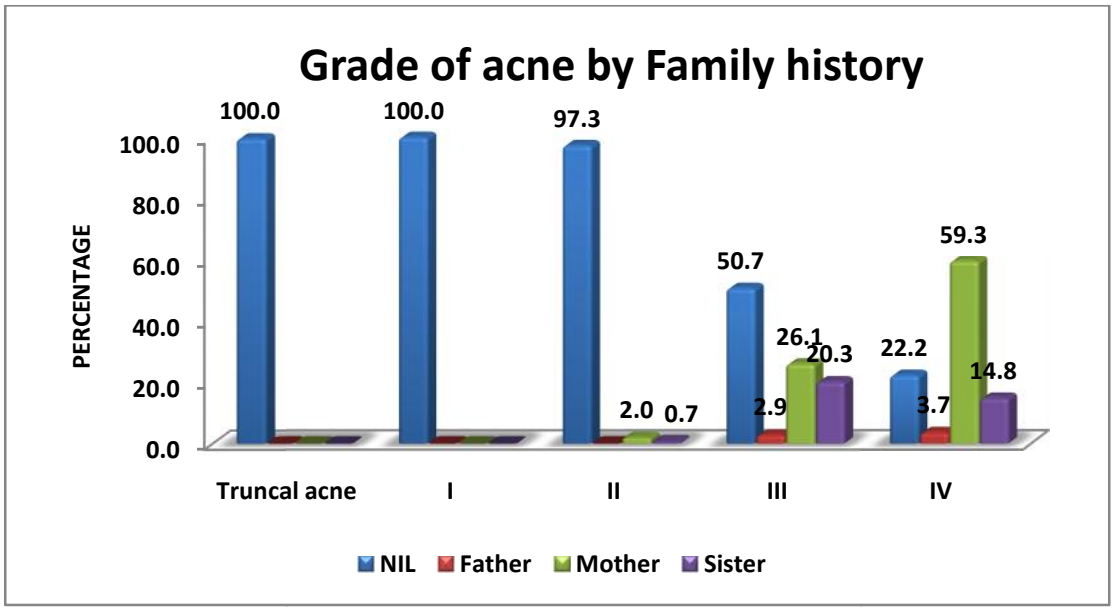


Figure11: Distribution of family history according to grade of acne



Site(s) involved

In the present study of 506 patients, 443(87.5%) had lesions only over the face, 26 patients(5.1%) had lesions over face and back, 22 patients (4.3%) had lesions over face, back and chest, and 15 patients(3%) had lesions over back and chest. Figure 12 represents distribution of cases according to site of involvement. Over the face, cheek was the predominant site of involvement in 212 patients (47.9%) followed by cheeks and forehead (38.1%), forehead(12.6%), and cheeks and chin(1.4%). Figure 13 represents predominant site of involvement over the face.

Figure 12: Distribution of cases according to site involved

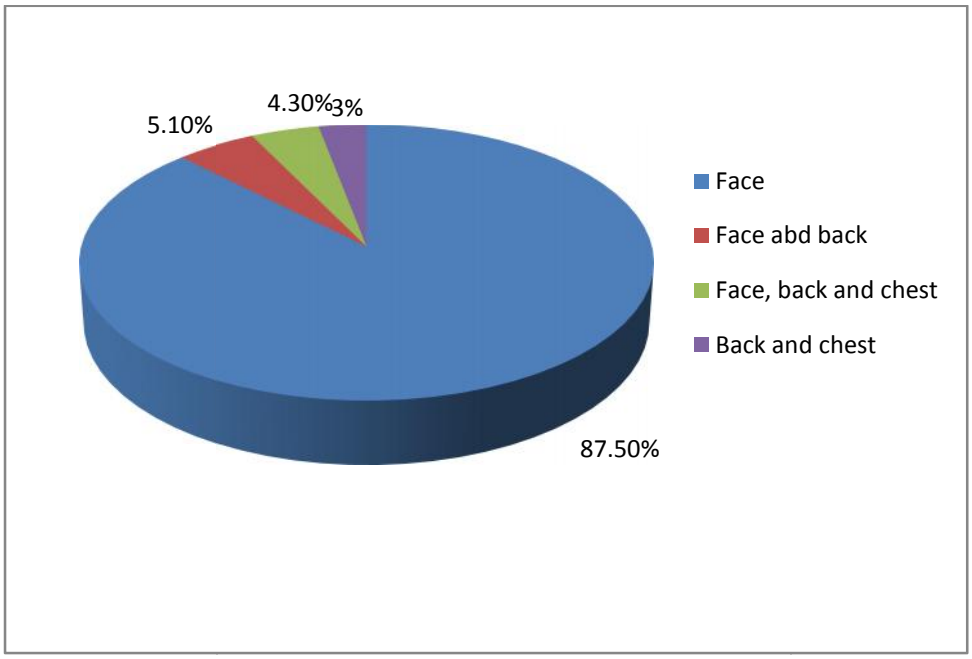
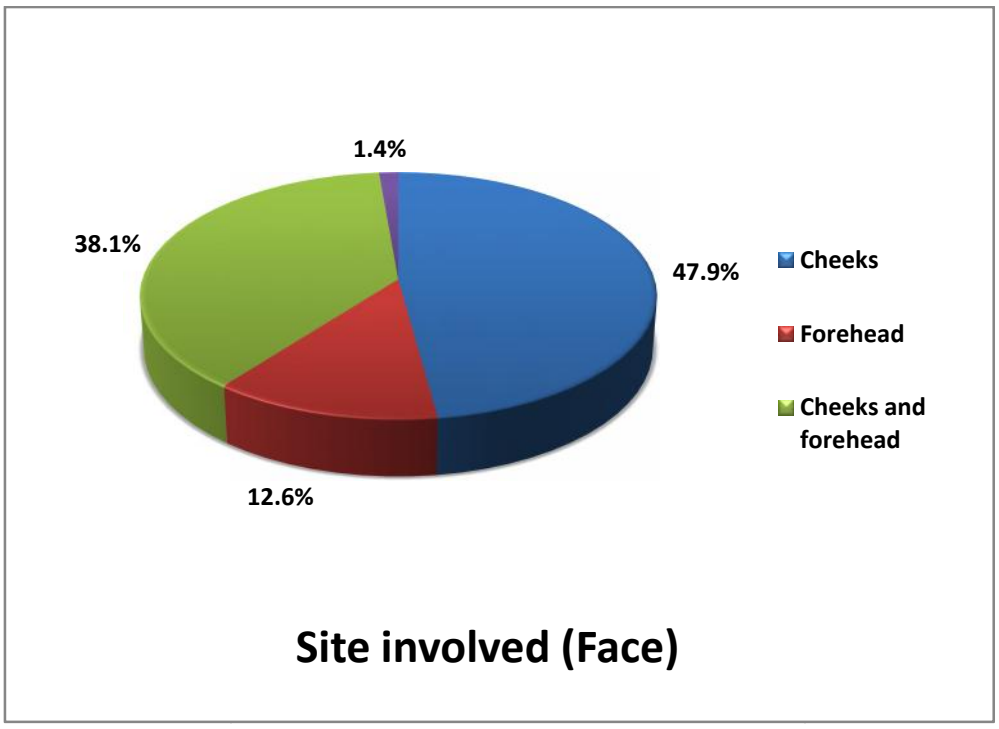


Figure 13: Site of involvement over the face



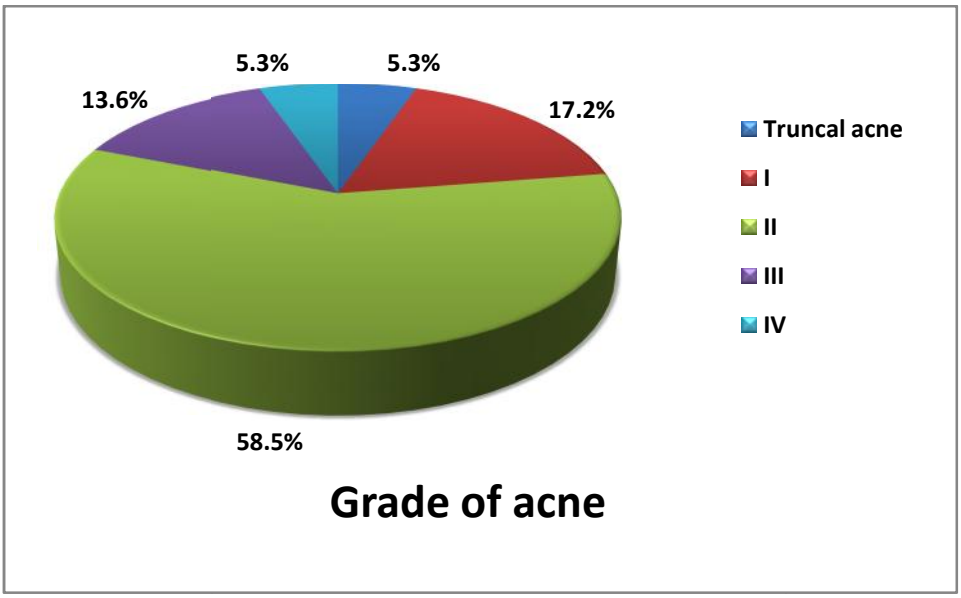
Grade of acne :

Most prevalent grade of acne was grade 2 affecting 296 patients (58.5%) followed by grade I affecting 87 patients (17.2%), grade III affecting 69 patients (13.6%) and grade IV affecting 27 patients (5.3%). Truncal acne was present in 27 patients (5.3%). Table 7 and Figure 14 represent distribution of cases according to grade of acne.

Table 7: Distribution of cases according to grade of acne

Grade of acne	Number of cases	Percentage(%)
Truncal acne	27	5.3
I	87	17.2
II	296	58.5
III	69	13.6
IV	27	5.3
Total	506	100

Figure 14: Distribution of cases according to grade of acne



The clinical photographs of patients with various grades of acne have been presented in figure 15 to 19

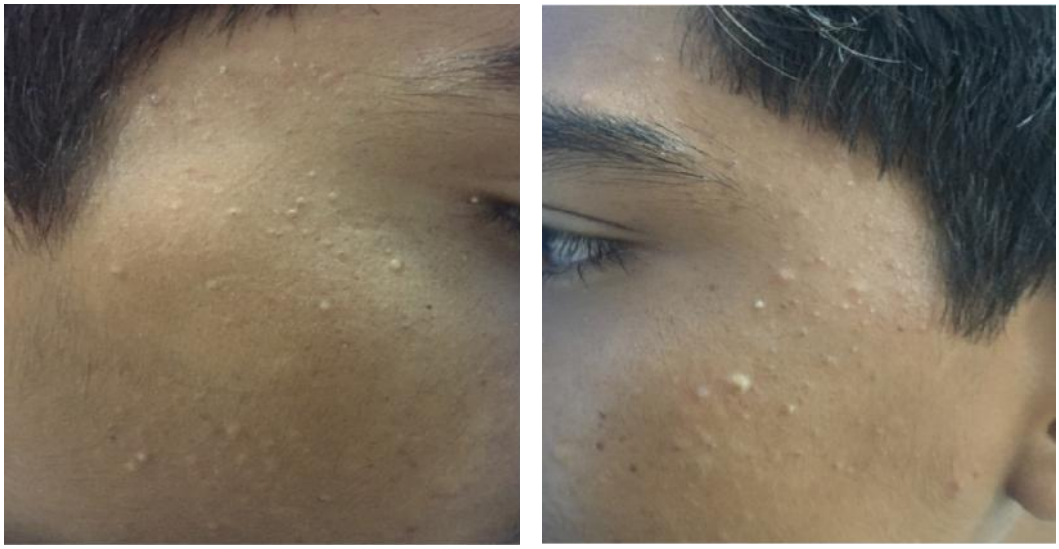


Figure 15 and 16 Grade 1 Acne vulgaris

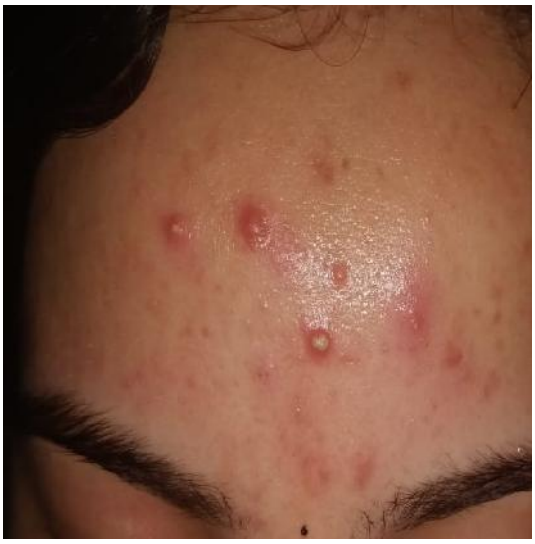


Figure 17: Grade II Acne vulgaris

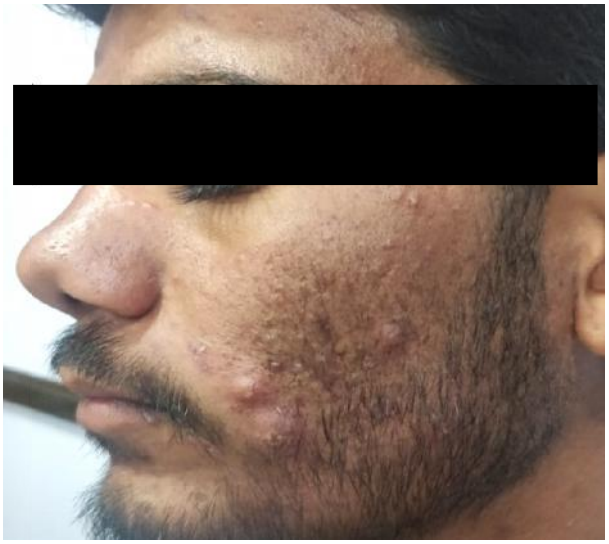


Figure 18: Grade III Acne vulgaris

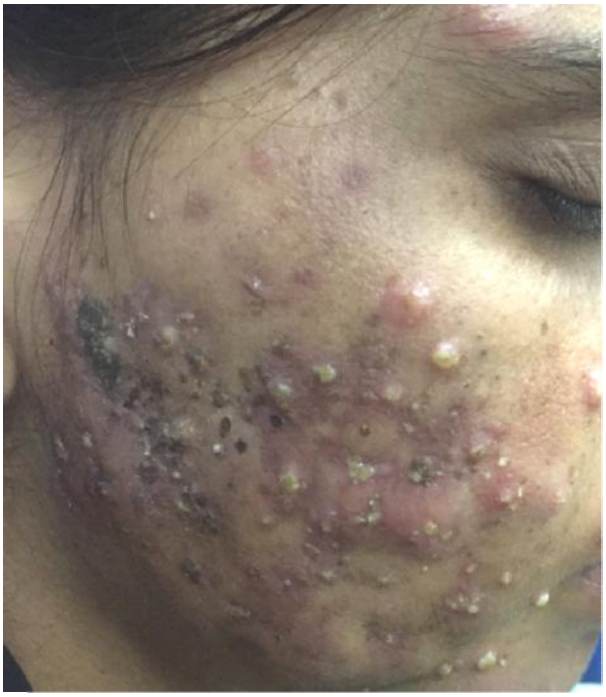
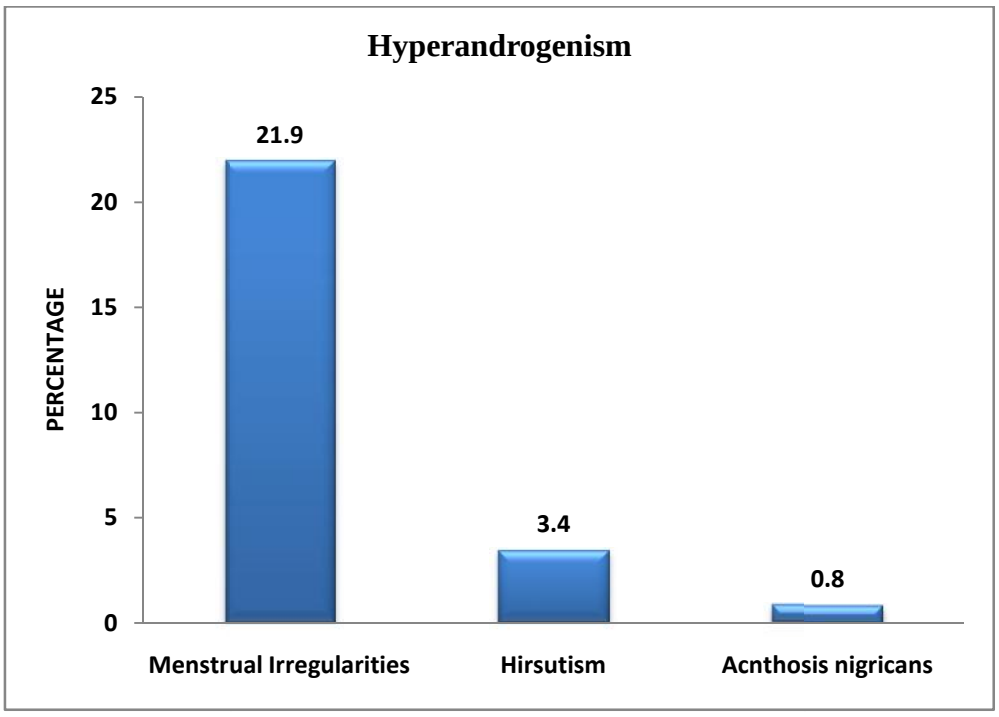


Figure 19: Grade IV Acne vulgaris

Signs of hyperandrogenism

Menstrual irregularities were seen in 111 females (21.9%) out of 313 female patients in our study. Other signs of hyperandrogenism like hirsutism and acanthosis nigricans were observed in 17(3.4%) and 4(0.8%) patients respectively. In 111 patients, ultrasonography report of 5 patients(4.5%) showed polycystic ovarian disease and LH:FSH was >2. Figure 15 represents distribution of cases according to signs of hyperandrogenism.

Figure 20: Distribution of cases according to signs of hyperandrogenism



DISCUSSION

Acne vulgaris is a common chronic inflammatory disorder which is considered to be disease of adolescent age. It is characterized by non inflammatory lesions like comedones and inflammatory lesions like papules, pustules, nodules, cyst and sinus formation.[2] Face is the commonest site of involvement.[2] Although acne is considered a cosmetic problem, it causes psychological impairment in patients mainly due to its complications like scarring and post inflammatory hyperpigmentation.[8]

In our study of 506 patients, majority of patients belonged to age group of 16-20 years (291 patients, 57.5%) followed by 21-25years(150 patients, 29.6%). Adityan *et al.* also observed similar findings with majority of patients being in age group of 16-20years (59.8%) followed by 21-25 years (19.4%). Patil *et al.* reported most common age group of 16-20 years (51%) followed by 11-15 years (21%). Sharma *et al.* observed majority of patients in age group of 14-16years (69.2%) followed by 11-13 years with (16%). Table 8 represents comparison of age distribution between present study with other studies

Table 8: Comparison of age distribution between present study with other studies

Age distribution	Present study	Adityan *et al*	Patil *et al*
11-15years	7.7%	35%	21%
16-20years	57.5%	59.8%	51%
21-25years	29.6%	19.4%	16%
25-30years	4.9%	4.8%	14%

Al-Ameer *et al.*[25] observed most common age of presentation of acne at 19.2±3 years for males and 18.4± 4.2 in their study of 225 patients. In our study, mean age among males was 20.2±3.1years and females 19.6±3.5 years.

Among 506 patients, 313 patients were females (61.9%), which can be due to more consciousness on the part of females as compared to males. Patil *et al.* also reported majority of females patients (69, 57.5%) and males being 51 patients (42.5%).

However, Adityan *et al.* reported predominance of male patients (172 patients, 55.7%) followed by females (137 patients, 44.3%). Sharma *et al.* also observed prevalence among males was slightly more at 73.2% than females at 71.1%.

In our study, 79 patients (15.6%) reported topical steroid application prior to aggravation of acne. Steroid application leads to hypercornification of upper portion of pilosebaceous unit which can exacerbate acne.[1]

Khunger *et al.* also observed aggravation of acne on application of topical steroids in 11.8% patients. Goulden *et al.* noted similar results where steroids exacerabated acne lesions. They also observed exacerabation of acne with drugs like antitubercular drugs, phenytoin, lithium and anabolic steroids. In our study no patients reported taking any oral medications. No significant association between acne and any topical or systemic medication was observed by Sharma *et al*.

Seasonal variation was observed in 92 patients, among which summer exacerbation was seen in 75 patients (14.8%) and winter exacerbation in 17 patients (3.4%). Adityan *et al.* reported summer exacerbation in 71 patients(23%) and winter exacerbation in 9 patients(2.9%). Patil *et al.* also reported similar findings with

summer exacerbation in 22 patients(18.3%) and winter exacerbation in 3 patients(2.5%). Sardana *et al*[26] in their study noted summer exacerbation in 28.5% patients. Our study correlates well with the above studies.

Sharma *et al.* reported no significant relation between acne and weather changes. Al-Ameer *et al.* showed majority of patients having exacerbation of acne in winter season which improved in summer season.

In our study, 313 patients were females of which 116 patients (22.9%) had premenstrual flare. Patil *et al.* observed premenstrual flare in 32 patients(46.4%) out of 69 female patients in their study. Adityan *et al.* noticed 57.7% patients having premenstrual flare of the 137 female patients in their study. Sharma *et al.* noticed premenstrual flare in 21.1% female patients. But in a study by Khanna *et al*[27] noticed reduction in non inflammatory and inflammatory lesions during premenstrual period.

Pilosebaceous ducts get smaller and, hence, get blocked during 15-20 days of menstrual cycle leading to premenstrual flare.[15]

In our study, family history of acne among 63 patients (12.4%) was observed . Positive family history was seen in mother among 40 patients (7.9%), in sister among 20 patients(4%) and in father among 3 patients(0.3%). Ghodsi *et al.* and Dreno *et al.*, also noticed increased incidence of acne in children in patients with positive family history as compared to those with no family history. Family history of acne was more common in patients with moderate to severe grade of acne as compared to those with mild grade of acne. However, Sharma *et al.* reported no significant association between acne and family history.

In the present study, 443(87.5%) patients had lesions only over face, 26 (5.1%) had lesions over face and back, 22 (4.3%) had lesions over whereas 15 patients (3%) had lesions over back and chest. Patil *et al.* found similar findings with face being the predominant site of involvement in 80 patients (66.7%) followed by lesions over face, back and chest in 18 patients(15%), face and back in 15 patients(12.5%), face and chest in 5 patients(4.2%) and back, chest and arm in 2 patients(1.6%). Biswa *et al.*[28] also observed similar findings with face being the most predominant area of involvement among 65% patients. Adityan *et al.* also observed face as the most common site involved in 202 patients(65.4%) in their study of 309 patients.

On face, cheeks were the predominantly involved sites in 212 patients (47.9%), with similar findings observed by Swati *et al.*[29] and Khunger *et al.* in their studies. After cheeks, chin and madibular area were the next most commonly involved sites.

In our study, 296 patients had grade II acne (58.5%) followed by grade I in 87 patients(17.2%), grade III in 69 patients(13.6%) and grade IV in 27 patients(5.3%). Swati *et al.* also reported that majority of the patients presented with grade II acne (80%) followed by grade I acne (10%), which was similar to our study but Adityan *et al.* observed grade I acne being predominant in 186 patients (60.2%), grade II in 85 patients(27.5%) , grade IV in 30 patients(9.7%) and grade III in 8 patients(2.6%).

In our study features of hyperandrogenism such as menstrual irregularities was observed in 111 patients(21.9%), hirsutism in 17 patients(3.4%) and acanthosis nigricans in 4 patients(0.8%). Although clinical features suggestive of

hyperandrogenism like menstrual irregulaties, hirsutism and acanthosis nigricans were present, only 5 patients(4.5%) had raised laboratory markers of hyperandrogenism. Khunger *et al*. also observed signs of hyperandrogenism like hirsutism in 6.95% and alopecia in 2.17% but laboratory markers were raised in only 2 patients.

CONCLUSION

Acne vulgaris is a common adolescent disorder which is characterized by non-inflammatory open and closed comedones in mild cases and inflammatory papules, pustules, nodules and sinus formation in severe cases.

In present study of 506 patients, higher incidence of patients was seen between 16-20 years of age with peak age of onset being 16-20years. Females were more commonly affected, which can be due to more cosmetic consciousness among females as compared to males.

Aggravating factors like summer exacerbation and premenstrual flare contribute to exacerbation of acne. Topical steroid application is also observed to exacerbate acne vulgaris in the present study. Positive family history was found in some patients which was associated with severe grade of acne at presentation (grade III and IV).

Face was the most commonly involved site in our study, cheeks being predominant site on the face. In the present study, grade II acne was most commonly seen, followed by grade I. Therefore most common lesions seen were papules and comedones.

Patients also presented with features of hyperandrogenism like menstrual irregularities, hirsutism and acanthosis nigricans, among whom, 5 patients showed ultrasonography features suggestive of polycystic ovarian syndrome and LH:FSH=>2.

From all the above observations, we find that acne has a multifactorial etiology. It poses a dermatological and cosmetic problem in patients which can have a negative psychosocial impact on their life. Educating the patients about the aggravating factors and counseling about the role of positive family history may lead to reassurance of the patients. As acne presents with different clinical lesions and grades, early diagnosis and appropriate treatment according to the grade not only improves the quality of life of patients but also prevents further dermatological and psychosocial complications.

SUMMARY

A hospital based cross sectional study to determine the epidemiological and clinical aspects of acne vulgaris in a tertiary care hospital was conducted during the period of September 2016 to August 2018.

- A total of 506 patients were included in the study.
- Females constituted the majority of patients i.e., about 61.9%
- Majority of patients were in age group of 16-20 years i.e., about 57.5%.
- Peak age of onset in males and females was 16-20 years of age.
- Early onset of acne was seen in females compared to males.
- Family history was present in 12.5%.
- Patients with positive family history had severe grade of acne.
- Topical steroid application aggravated acne in 15.6% .
- Summer exacerbation was seen in14.8% and winter exacerbation in 3.4%%.
- Premenstrual flare was seen in 22.9%.
- Most commonly affected site was face (87.5%). On face, cheeks were predominantly involved in 47.9%.
- Grade II was predominantly found grade of acne in 58.5% followed by grade I in 17.2%
- Truncal acne was seen in 5.3% of patients

- Features of hyperandrogenism such as menstrual irregularities was observed in 111 patients(21.9%), hirsutism in 17 patients(3.4%) and acanthosis nigricans in 4 patients(0.8%). 5 patients(4.5%) had raised laboratory markers of hyperandrogenism

BIBLIOGRAPHY:

1. Khunger N, Kumar C. A clinico - epidemiological study of adult acne: Is it different from adolescent acne? Indian J Dermatol Venerol Leprol 2012;78:335-41.

2. Layton AM, Eady EA, Zouboulis CC. Acne. In: Griffiths C, Barker J, Bleiker T, Chalmers R and Creamer D, editors. Rook's textbook of dermatology, 9th edn. Blackwell Publishing; 2016.p.90.1-65.

3. James WD, Berger TG, Elston DM. Acne. Andrews' diseases of skin, 12th edn. Elsevier Publishers;2016.p.225-44.

4. Zaenglein AL, Graber EM, Thiboutot DM. Acne vulgaris and acneiform eruptions. In: Wolff K, Goldsmith LA, Katz SI, Gilchrest BA, Paller AS and Leffell DJ, editors. Fitzpatrick Dermatology in General Medicine, 8th edn. New York: Mcgraw-Hill;2012. p:897-925.

5. Patil M, Bendigeri J. Clinico-epidemiological study of acne vulgaris in Southern India. International Journal of Biomedical Research 2015;6:509-11.

6. Vaishampayan S, Baveja S, Garg S. Acne, rosacea and perioral dermatitis. In: Sacchidanand S, Oberoi C, Inamadar AC, editors. IADVL Textbook of dermatology, 4th edn. Mumbai: Bhalani Publishing House;2015.p.1365-1412.

7. Lynn DD, Umari T, Dunnick CA, Dellavalle RP. The epidemiology of acne vulgaris in late adolescence. Adolesc Health Med Ther 2016;7:13-25.

8. Durai PT, Nair DG. Acne vulgaris and quality of life among young adults in South India. Indian J Dermatol 2015;60:33-40.

9. Evans DM, Kirk KM, Nyholt DR, Novac C, Martin NG. Teenage acne is influenced by genetic factors. Br J Dermatol 2005;152:565–595

10. Drenoa B, Polib F. Epidemiology of Acne. Dermatol 2003;206:7–10

11. Duquia RP, Santos ID, Almeida HD, Souza PR, Breunig JA, Zouboulis CC. Epidemiology of Acne Vulgaris in18-Year-Old Male Army Conscripts in a South Brazilian City. Dermatol 2017;233:145–154

12. Bagatin E, Timpano DL, Guadanhim LR, Nogueira VM , Terzian LR, Steiner D *et al*. Acne vulgaris: prevalence and clinical forms in adolescents from São Paulo, Brazil. An Bras Dermatol. 2014;89:428-35.

13. Ghodsi SZ, Orawa H, Zouboulis CC. Prevalence, Severity, and Severity Risk Factors of Acne in High School Pupils: A Community-Based Study. J Investigative Dermatol 2009; 129: 2136–2141

14. Yeung CK, Teo LH, Xiang LH, Chan HH. A Community-based Epidemiological Study of Acne Vulgaris in Hong Kong Adolescents. Acta Dermtol Venereol 2002; 82: 104–107

15. Adityan B, Thappa DM. Profile of acne vulgaris- A hospital based study from South India. Indian J Dermatol Venerol Leprol 2009;75:272-78.

16. Sharma RK, Dogra S, Singh A, Kanwar AJ. Epidemiological patterns of acne vulgaris among adolescents in North India: A cross - sectional study and brief review of literature. Indian J Paed Dermatol 2017;18: 196-201

17. Gollnick H, Cunliffe W. Management of Acne. J Am Acad Dermatol 2003; 49:1-36

18. Goulden V, Mcgeown CH, Cunliffe WJ. The familial risk of adult acne: a comparison between first-degree relatives of affected and unaffected individuals. Br J Dermatol 1999;141: 297-300

19. Cordain L, Lindeberg S, Hurtado M. Acne Vulgaris- A Disease of Western Civilization. Arch Dermatol 2002;138:1584-1590

20. Cordain L. Implication for the role of diet in acne. Semin Cutan Med Surg 2005; 24:84-91

21. Chuh AT, Zawar V, Wong WC, Lee A. The association of smoking and acne in men in Hong Kong and in India: a retrospective case–control study in primary care settings. Clin Exp Dermatol 2004;29: 597–599

22. Bhate K, Williams HC. Epidemiology of acne vulgaris. Br J Dermatol 2012;168:474-85.

23. Tan JK, Jones E, Allen E, Prinpotnev S, Raza A, Wolfe B. Evaluation of essential clinical components and features of current acne global grading scales. J Am Acad Dermatol 2013;69:754-61.

24. Adityan B, Kumari R, Thappa DM. Scoring system in acne vulgaris. Indian J Dermatol Venerol Leprol 2009;75:323-26.

25. Al-Ameer AM, Al-Akloby OM. Demographic features and seasonal variations in patients with acne vulgaris in Saudi Arabia: a hospital-based study. Int J Dermatol 2002; 41: 870-1.

26. Sardana K, Sharma RC, Sarkar R. Seasonal variation in acne vulgaris - myth or reality. J Dermatol 2002; 29:484-488.

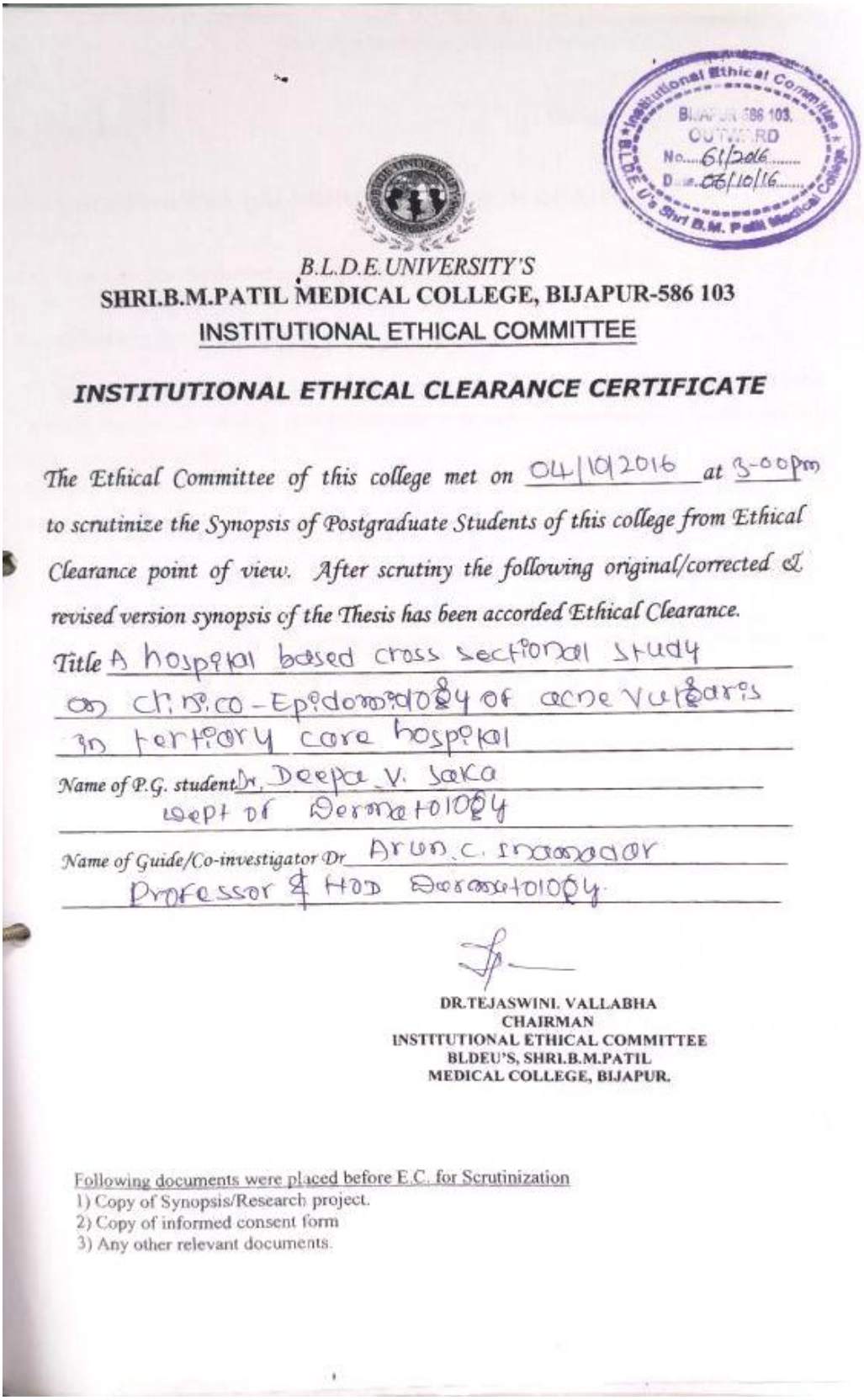
27. Kanna VN, Pandhi KR. Relationship of acne with menstrual period. Indian J Dermatol Venerol Leprol 1991;57:138-40.

28. Biswas S, Mondal KK, Saha I, Dutta RN, Lahiri SK. Clinico-epidemiological features of Acne vulgaris. A tertiary hospital- based study. Iran J Dermatol 2010;13: 37-41.

29. Swati G, Kusagur MS. A Clinico-Epidemiological Study of Acne in Adults. Int J of Science and Research 2015;4: 822-25.

ANNEXURES

ETHICAL CLERANCE CERTIFICATE



Institutional Ethical Committee
BLDE U's Shri B.M. Patil Medical College
BIJAPUR 586 103.
OUTWARD
No. 61/2016
Date 06/10/16

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 04/10/2016 *at* 3-00pm *to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.*

Title A hospital based cross sectional study on clinico-Epidemiology of acne vulgaris in tertiary care hospital

Name of P.G. student Dr. Deepa V. Saka
Dept of Dermatology

Name of Guide/Co-investigator Dr Arun. C. Inamadar
Professor & HOD Dermatology

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

B.L.D.E.(DEEMED TO BE UNIVERSITY)

SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR.

Department of Dermatology, Venereology and Leprosy.

Name: SL NO:

Age: Date:

Sex: IP NO/ OP NO:

Occupation:

Address:

1. Chief complaints:
2. Age of onset:
3. Site of onset:
4. Duration of lesion:
5. Aggravating factors:
   - Any drug used:
     - Oral:
     - Topical:

       Steroid cream or combination of steroid used

       Name of drug-
   - Seasonal variation:
   - Sun exposure:
   - Diet:
   - Application of cosmetics:
   - Premenstrual flare:

- Others :

6. Family history:

7. Personal history:

8. General Physical Examination:

Weight:	BP:	Pulse rate:
Pallor:	Cyanosis:	Icterus:
Clubbing:	Lymphadenopathy:	Edema:

9. Cutaneous examination

- Examination of acne:-
  - Site involved:
  - Morphology of lesions:
  - Grade of acne:

Grade 1
Grade 2
Grade 3
Grade 4

- Signs suggestive of hyperandrogenism : Acanthosis nigricans/ Hirsutism

10. Systemic Examination:

11. Diagnosis:

12. Investigations :

INFORMED WRITTEN CONSENT FORM

B.L.D.E. .(DEEMED TO BE UNIVERSITY) SHRI B M PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR-586103

RESEARCH INFORMED CONSENT FORM

TITLE OF THE PROJECT : A HOSPITAL-BASED CROSS SECTIONAL STUDY ON CLINICO-EPIDEMIOLOGICAL STUDY ON ACNE VULGARIS IN TERTIARY CARE HOSPITAL

PG GUIDE : - DR ARUN C.INAMADAR

PG STUDENT : - DR. DEEPA V SAKA

PURPOSE OF RESEARCH:-

I have been informed that this project will be studied to know clinico-epidemiological study of acne vulgaris

BENEFITS:-

I understand that my participation in this study will help the investigator to study the clinico-epidemiology of acne vulgaris

PROCEDURE:-

I understand that relevant history will be taken and I will undergo detailed clinical examination after which necessary investigations will be done whenever required.

RISK AND DISCOMFORTS:-

I understand there is no risk involved during the procedures performed.

CONFIDENTIALITY:-

I understand that medical information produced by this study will become a part of my hospital records and will be subjected to the confidentiality and privacy regulation of the said hospital. Information of a sensitive personal nature will not be a part of the medical records, but will be stored in the investigator's research file.

If the data are used for publication in the medical literature or for teaching purposes no names will be used and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand I may see the photographs, videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:-

I understand that I may ask more questions about the study at any time concerned. Dr. Deepa V Saka is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which may influence my continued participation.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:-

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in this study at any time without prejudice. I also understand that Dr. Deepa V Saka may terminate my participation in this study at any time after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician, if this is appropriate.

INJURY STATEMENT:-

I understand that in the unlikely event of injury to me resulting directly from my participation in this study and if such injury were reported promptly, then medical treatment will be available to me, but no further compensation will be provided. I

understand that by my agreement for my participation in this study, I am not waiving any of my legal rights.

I have explained to (patient's / relevant guardian's name) the purpose of the research, the procedures required, and the possible risks and benefits to the best of my ability in patient's own language.

_____ _____

Investigator / P. G. Guide Date

I confirm that …….(Name of the PG guide / chief researcher) has explained to me the research, the study procedures that I undergo and the possible risks and discomforts as well as benefits that I may experience. I have read and I understand this consent form. Therefore, I agree to give my consent for my participation as a subject in this research project.

_____ _____

Participant / guardian Date

_____ _____

Witness to signature Date

KEY TO MASTER CHART

A : USG: suggestive of polycystic ovarian syndrome, FSH: 4.2mIU/ml, LH:30mIU/ml, LH:FSH:>2

B:USG: suggestive of polycystic ovarian syndrome, FSH: 5mIU/ml, LH:40mIU/ml, LH:FSH:>2

C:USG: suggestive of polycystic ovarian syndrome, FSH: 3mIU/ml, LH:38mIU/ml, LH:FSH:>2

D : USG: suggestive of polycystic ovarian syndrome, FSH: 3.2mIU/ml, LH:40mIU/ml, LH:FSH:>2

E:USG: suggestive of polycystic ovarian syndrome, FSH: 5.6mIU/ml, LH:42mIU/ml, LH:FSH:>2