INTRODUCTION

Obesity is now widely considered as a major public health problem. It is considered to be a global epidemic. In India the prevalence of obesity is 12.6% in women and 9.3% in men. In other words, more than a 100 million individuals are obese in India.11

Health care expenditures are significantly higher for overweight and obese individuals.1 Obesity carries a significant impact on psychological health, as well. It is indirectly labeled to anxiety, impaired social interaction and depression.4

Obesity has shown a well-established relation with conditions as coronary heart disease, type 2 diabetes mellitus, hypertension, hyperlipidemia, osteoarthritis, and obstructive sleep apnea and is indirectly associated to anxiety, impaired social interaction, and depression.

Overweight and obesity are defined by WHO as abnormal or excessive gain of fat accumulation that may impair health.1 Obesity has been accepted as a recognized public health problem internationally with rapidly rising prevalence in several industrialized countries, recorded to be 36% in American population in 2009- 2010.2 In most of the Asian countries, prevalence of obesity has increased many folds since last few decade. Body mass index (BMI) is the most commonly used method to define obesity. It is a ratio of weight in kilograms divided by height in meters squared (kg/m2).

The WHO uses body mass index (BMI) to classify underweight, overweight, and obesity. A BMI of 18.5–24.9 kg/m^2 is taken as normal, BMI 25–29.9 kg/m^2 overweight, and BMI >30 kg/m^2 taken as obese. Obesity can be further

characterized as by BMI as Class I (30–34.9 kg/m ^{2}), Class II (35–39.9 kg/m ^{2}), and Class III (>40 kg/m ^{2}).[3}

In accordance with the consensus by WHO, as per "Report of WHO Consultation", normal BMI for Asians is 18.5-22 kg/m2 , overweight (BMI >23kg/m2), at risk (BMI 23-24.9 kg/m2), obesity I (BMI 25-29.9 kg/m2), obesity II (BMI >30 kg/m2)6.

Obesity has a various effects and contributes to the pathogenesis of metabolic, cardiovascular, carcinogenic, musculoskeletal and cutaneous disorders.7 Altered physiological functions of skin secondary to obesity include the changes in the barrier function, excessive sweat and sebaceous glands secretion. Furthermore, there is an impaired lymphatic drainage, altered collagen structure and functioning, delayed wound healing, impaired micro and macrocirculation.8,9,10.

As a result of these changes multiple cutaneous manifestations are observed frequently in obese persons including acanthosisnigricans, acrochordons, manifestations of hyperandrogenism like hirsutism and striae. Other important manifestations are subcutaneous fat deposition, hidradenitissuppurativa, varicose veins and venous insufficiency and lymphedema. Infections including cellulitis, furunculosis, fungal and viral infections are also seen frequently. In addition to these, several preexisting dermatological conditions are also aggravated that includes psoriasis, gouty arthritis and insulin resistance syndrome.

OBJECTIVES

To determine the frequency of various dematological manifestation in patients with obesity .

REVIEW OF LIETERATURE

Obesity can be defined as a pathological state where by there is too much adipose tissue for the size of the body . The obese person is easily recognized by inspection , where as the overweight person,s weight is midway between the person with an ideal body weight. 10 The most widely used measurement of obesity is the BMI , which is determined by dividing weight (kg) by height (kg/m2). Under current definitions as adopted by the WHO and US CDS men and women are considered overweight with a BMI between 25 and 29.9 and obese with a BMI greater than 30 .There are many ways of determining obesity.

Obesity can be determined by directly measuring the percentage of body weight that is fat ,for which measurement BMI is a not true. This can be carried out by obtaining the weight under water by measuring skin fold thickness or by the use of radiographic imaging or dual –energy –x-ray absorptionmeteryscanning . Waist circumference and waist-to- hip ratio can also serve as important measurements of cental obesity .

Pathophysiology of obesity–Environmental and genectic factors both are involved in the causation of obesity .Based on many research , approximately 60% to 70% of the variance in BMI can be attributed to environment and 30% to 40% of the variance in BMI can be attributed to genetics.5 The contributions of environmental factors to the etiology of obesity are well known. Dietary choices, socioeconomic status, and behavioral factors, such as inactivity, are all important factors in obese patients.

Obesity results from a chronic imbalance between energy intake and energy expenditure. Specifically, 3 metabolic factors have been reported to be predictive of

weight gain: (1) low adjusted sedentary energy expenditure6 ; (2) high respiratory quotient (carbohydrate-to-fat oxidation ratio)7 ; and (3) low level of spontaneous physical activity. The interaction between genetics and environment is also important. Individuals may be genetically predisposed to become obese; however, the obesity genotype may only be expressed under certain environmental conditions.

Molecular genetics of obesity-

Although researchers have identified monogenic forms of obesity resulting from mutations in genes involved in central pathways of food intake regulation, the vast majority of obesity cases result from a varioscomplx genetic disease involving interactions between multiple genes and the environment. we will focus on the two gene products which are known to have direct effects on the skin—the leptin and proopiomelanocortin (POMC) genes .Leptin, the product of the Ob gene, is a hormone secreted by adipocytes that regulates energy homeostasis and food intake via specific receptors in the hypothalamus.10 Congenital leptin deficiency has been identified in humans and is associated with a r are, severe early-onset form of obesity.11 In such patients, treatment with leptin is successful.12

However, most obese patients actually have elevated circulating leptin levels in the setting of functional leptin resistance, and treatment with exogenous leptin is ineffective in ameliorating the obesity.13

The second possible genetic contributor towards obesity is the POMC gene. POMC is expressed in various tissues, including the pituitary, immune system, hypothalamus, and skin.20,21 In these tissues, POMC is cleaved into smaller peptides including beta-endorphin, adrenocorticotropin, and alpha-, beta- and gamma-melanocyte stimulating

hormones, which play various roles in control of analgesia, inflammation, adrenal steroidogenesis, and skin pigmentation.20,22

MC1 receptor is associated with human pigmentation and mutations in the gene for this receptor are known to cause red hair and fair skin.24 MC4r deficiency is the most common monogenic cause of obesity, and mutations in this gene affect at least 3% of extremely obese individuals.23,25 This receptor appears to play a key role in the control of eating behavior in humans.26 A syndromic variant of POMC deficiency has been described with complete lossof-function mutations of the POMC gene.23,27,28 The syndrome consists of severe early-onset obesity, adrenal insufficiency, reduced skin pigmentation, and red-orange hair. However, the contribution of the POMC gene to the pathogenesis of nonsyndromic obesity remains unclear. Secondary causes of obesity –

Secondary causes of obesity, that is, due to underlying pathology , including Cushing Syndrome ,eating disorder(bing eating disorder), genetic syndrome which includes – Albright, shereditary osteodystrophy, Alstrom syndrome, Angelman syndrome, prader-willi syndrome and Turner Syndrome .Growth hormone deficiency, hypogonadism, hypothalamic obesity, insulinoma , medication related which includes antidepressants, anticonvulsant, glucocorticoids, antipsychotic, insulin and oral contraceptive. Polycystic ovarian syndrome and peudohypoparathyroidism are other causes.

Obesity And Skin Physiology –

Obesity is related to a number of effects on skin physiology, including effects on skin barrier function, sebaceous glands and sebum production, sweat glands, lymphatics, collagen structure and function, wound healing, microcirculation and macrocirculation, and subcutaneous fat.

The role of Skin as a barrier - Obesity is associated with a number of significant changes in skin barrier function. Loffler, Aramaki, and Effendy29 used bioengineering methods to investigate the correlation between BMI and epidermal functions. Obese individuals demonstrated significantly increased transepidermal water loss and erythema compared with control subjects,29 suggesting a fundamentally altered epidermal barrier.

The role of Sebaceous glands and sebum production in obesity – There are no epidemiologic studies examining the relationship between obesity and sebum production. This relationship is potentially important because sebum production plays a major role in the pathogenesis of acne.30-33 In addition, diet may directly or indirectly influence causes of acne, including increased proliferation of basal keratinocytes within the pilosebaceous duct, incomplete separation of ductal corneocytes from one another and subsequent obstruction of the pilosebaceous duct, and androgen-mediated increases in sebum production.34The severity of Acne is increased by is by obesity-associated disorders, such as hyperandrogenism and hirsutism. Androgens, insulin, growth hormone, and insulinlike growth factors are frequently elevated in obese patients and have been demonstrated to activate sebaceous glands and influence acne severity.35,36

Role of Apocrine and eccrine sweat glands in obesity -Previous authors have suggested that obesity associated changes in skin physiology may be related to increased sweat gland activity.29 Obese patients have larger skin folds and sweat more profusely after becoming overheated because of thick layers of subcutaneous fat, thereby increasing both the frictional and moisture components.38 However, currently there are no specific published data on the structure and function of apocrine and eccrine sweat glands in obesity.

Lymphatics in obesity -Obesity produces resistance in lymphatic flow, which leads to collection of protein-rich lymphatic fluid in the subcutaneous tissue. This accumulation frequently results in lymphedema. Lymphedema is caused by dilatation of tissue channels and reduced tissue oxygenation.38 Further accumulation of fluid in the setting of decreased oxygen tension leads to fibrosis and a chronic inflammatory state.

Collagen structure in obesity and its function and wound healing-In animal studies, obesity is also associated with altered collagen structure and function and impaired wound healing. Enser and Avery40 demonstrated that the skin of obese mice was mechanically weaker and generated a lower hydrothermal isometric force compared with the skin of lean mice. The authors give the opinion that the decreased mechanical strength of skin in obese mice resulted from failure of collagen deposition to match the increase in skin surface area. One study on mouse suggest that that obese mice demonstrated slower wound healing and decreased wound collagen deposition.41 The authors proposed that structural changes in adipose tissue leads to decreased wound collagen deposition . Another study demonstrated that increased turnover of type III collagen correlated with obesity, particularly abdominal obesity.42

Microcirculation and macro circulationSeveral studies have demonstrated that obesity is associated with significant changes in cutaneous microcirculation and macrocirculation. Obesity appears to be a primary cause of microvascular dysfunction, which may contribute to the development of obesity-related microangiopathy and hypertension.43 Using bioengineering methods, Loffler, Aramaki, and Effendy29 demonstrated that higher BMI was significantly correlated with increased cutaneous blood flow. In obese children, there is increased cutaneous baseline blood flow and peak blood flow and greately lower peak capillary blood cell velocity compared with healthy control subjects.44 These changes in cutaneous microcirculation may be due to physiological compensation.

Subcutaneous fat-It is proposed that In adults individual , subcutaneous fat is made up mostly of white adipose tissue, which provides insulation and serves as an energy house . White adipose tissue plays an important role in endocrine functions as well as metabolism of lipids and glucose.47 Endocrine peptides secreted by adipocytes include leptin and tumor necrosis factor a, among others. The brown fat plays asignificant role in infant ,brown fat is most prominent in newborn infants and its exact role in obese adults is not very clear .

Table I. Skin disorders in obesity

Insulin resistance
Insulin resistance syndrome
Acanthosis nigricans
Acrochordons
Keratosis pilaris
Hyperandrogenism
Hirsutism
Mechanical
Plantar hyperkeratosis
Striae distensae
Cellulite
Adiposis dolorosa
Lymphedema
Chronic venous insufficiency
Infectious
Intertrigo
Candida
Dermatophytes
Folliculitis
Necrotizing cellulitis/fasciitis
Inflammatory
Hidradenitis suppurativa
Psoriasis
Metabolic
Tophaceous gout

Taken from JAAD –Cutaneos Manifestation of obesity -2004

SKIN MANIFESTATIONS OF OBESITY

Acanthosisnigricans Acanthosisnigricans is the most common cutaneous manifestation of obesity. Acanthosisnigricans appears as symmetric, velvety, hyperpigmented plaques that may occur in almost any location. It is most commonly observed in the sides of neck , axilla, groin, and posterior neck but it can also be seen on the elbows, knuckles, and face, particularly in ethnic skin . Acrochordons are frequently observed in the affected areas. The hyperpigmentation observed is secondary to acanthosis and papillomatosis of the epidermis rather than pigment-producing cells. The skin proliferation abnormalities in acanthosisnigricans are

frequently associated with hyperinsulinemia and insulin resistance.50 Hud et al51 found that 74% of an obese population exhibited acanthosisnigricans along with elevated plasma insulin levels. In obese women with hyperandrogenism and hirsutism, acanthosisnigricans most commonly affects the vulva.54 The proposed mechanism of how hyperinsulinemia leads to this epidermal change begins at the cellular level. Increased levels of circulating insulin leads to decreased numbers of functional insulin receptors.55,56 These ‘‘classic’’ insulin receptors regulate glucose uptake, cell growth, DNA synthesis, and protein and fat metabolism via tyrosine kinase activity. Keratinocytes and fibroblasts both express insulin-like growth factor (IGF) receptors that are also capable of binding insulin and have growthpromoting effects.57 Decreased numbers of functional insulin receptors cause a shift to increased binding to IGF receptors contributing to the development of acanthosis nigricans.55 Acanthosisnigricans plaques can be managed by improved control of hyperinsulinemia.

Acrochordons -Acrochordons are pedunculated soft brown papules most commonly seen on the neck and in the axillae and groin; they are frequently seen in association with acanthosisnigricans. The percentage of those with acrochordons increased with the severity of obesity.66 In general, acrochordons are more strongly associated with diabetes than with obesity. Kahana et al67 did not find an increased incidence with obesity but did report that those patients with acrochordons had greater impairment of carbohydrate metabolism.It is proposed that insulin sensitivity improves with weight loss68; therefore the increased incidence seen at higher BMI may be due to greater insulin resistance.

Hyperandrogenism and hirsutism - It is mentioned that hyperandrogenism can be the result of increased production of endogenous androgens due to increased volumes of adipose tissue (which synthesizes testosterone) and hyperinsulinemia (which increases the production of ovarian androgens). Cutaneous virilism can include hirsutism, acne vulgaris, hidradenitissuppurativa, and androgenic alopecia. There appears to be an association between cutaneous virilism, acanthosisnigricans, keratosis pilaris, and insulin resistance.53 Ruutiainenet al72 found that facial hirsutism is significantly correlated with BMI independently of age and testosterone level.

Striaedistensae - Striaedistensae, stretch marks, are linear atrophic plaques that are distributed perpendicular to the force of greatest tension and are commonly found on the breasts, buttocks, abdomen, and thighs . Striaedistensaebegin with an erythematous phase before turning violet, then finally becoming white depressed plaques. The exact pathogenesis of striae has yet to be elucidated, but mechanical, hormonal, and genetic factors may play a role. They are present in obese patients,66 and in other clinical settings such as pregnancy, Cushing's syndrome, and topical corticosteroid use.75 Hsu et al76 diagnosed striae in 40% of children with moderate to severe obesity, and incidence was higher in those with a longer duration of obesity.

The striaedistensaeclinically can be divided into an acute and a chronic state with both states frequently occurring simultaneously in the same patient. The acute state which is known as striaerubra is characterized by flattened, reddish to violaceous finely wrinkled lesions which run perpendicular to tension lines of the skin [3] . If these striaerubra remain untreated, these lesions evolve to a chronic state known as

striae alba. Striae alba present clinically as whitish, atrophic appearing, depressed, irregularly linear scar-like bands [3] .

Striae can be regarded as ‘‘scars’’ that result from injury of dermal connective tissue in which the newly generated collagen aligns in response to local stress forces.79 In the early stage of development, elastolysis, mast cell degranulation, and macrophage engulfment of elastic tissue have all been documented by light and electron microscopy.80 Typical histopathologic features also include densely packed eosinophilic thin collagen bundles parallel to epidermis, effacement of rete ridges, and lack of adnexal structures, thus reinforcing striae as forms of scars.81

Lymphedema -In obese patients, lymphedema results from impedance to lymphatic flow. In such patients, lymphedema presents clinically as initially soft, pitting edema most commonly beginning in the feet and spreading proximally). With time, further accumulation of fluid, decreased oxygen tension, and macrophage function lead to fibrosis and a chronic inflammatory state. In this setting of reduced tissue oxygenation, lymphedema provides a culture medium for bacterial growth.38 The patient is subject to repeated bacterial infections in the affected tissue that causes further perilymphatic scarring and impedance to lymphatic flow, thus entering the patient in a downward spiral. Chronic lymphedema can lead to elephantiasis nostrasverrucosa, defined by hyperkeratosis and papillomatosis of the epidermis overlying an indurated dermis and subcutaneous tissue.108

Chronic venous insufficiency Obesity is a recognized risk factor for the development of chronic venous insufficiency.113,114 Multiple studies have documented this association in both women115,116 and men.117,118 Padberg et al119 could not demonstrate definitive venous valvular disease in obese patients with chronic venous

insufficiency, which suggests that obesity alone may be inducing the morbidity associated with this disease. The increased intra-abdominal pressure found in obese patients causes an oppositional force to venous return from the lower extremities. Valvular incompetence and venous dilation leading to varicosities may result; however, the relationship between obesity and varicose veins is controversial. Because of increased hydrostatic pressure, components of intravascular fluid may leak into tissue. Red blood cells extravasated from veins deposit hemoglobin within the dermis and incite an inflammatory reaction with erythema and warmth. Pitting edema, brown macular hyperpigmentation, and scaling are also typically clinically apparent.38 Stasis dermatitis is the result of irritation of superficial nerve fibers by the increased pressure and metabolic break-down products increasing local pH.120 Lipodermatosclerosis and venous ulcerations may complicate chronic venous insufficiency. The fibrosingpanniculitis of lipodermatosclerosis presents as bound-down, brawny skin overlying an indurated dermis and subcutaneous tissue. Thelower limbs are the most common location for this process; however, the abdomen can also be affected in obese individuals.121 Venous ulcerations are found most commonly along the medial aspect of the lower extremity between mid-calf and the medial malleolus along the course of the greater saphenous vein. They account for approximately 70% of ulcerations found on the lower extremities.122 Overweight individuals have a greater risk for ulceration compared with those of normal body mass with comparable venous reflux severity.123

Plantar hyperkeratosis Plantar hyperkeratosis is a thickening of the skin over the soles of the feet or plantar area of the foot , secondary to the increased pressure and mechanical stress placed on the feet, in particularly the head of metatarsal , due to the weight gain associated with obesity (Figure 3). Studies have shown that nearly

50% of obese people suffer from plantar hyperkeratosis and that the relationship between the development of plantar hyperkeratosis and obesity increases with increasing BMI [13]. Hyperkeratosis is part of the skin's physiologic protective response or defencemechanism tohandle increased mechanical stress, it can create a variety of problems including impingement of the plantar nerves, arthritis of the small joint of the foot , painful ambulation, impaired balance, and difficulty wearing shoes [14]. The above mentioned abnormality as a result of hyperkeratosis not only disrupt the patient's activities of daily living but put the patient at an increased risk for falls.

Hyperkeratosis of the soles in obesity was first described by Garcia-Hidalgo et al66 in 1999. The horseshoe-shaped hyperkeratosis which is a common defect in obese person overlying the posterior portion of the sole was the most common skin finding in those weighing more than 176% of expected weight. Obese patients have higher plantar pressures during walking and standing127,128 and increased forefoot width.127 There is also an abnormal transference of weight during walking that alters the alignment of the foot, causing increased stress over bony prominences.129 The plantar hyperkeratosis that develops may be regarded as a physiologic response to mechanical trauma.130

The best approach for management of plantar hyperkeratosis includes weight loss counseling as this helps to alleviate the primary underlying cause of the lesion [15]. Fortunately, during the patient is losing weight, there are a number of effective symptomatic treatments. One of the quickest way of relief is surgical debridement of the lesion. Using a #15 scalpel blade to remove the excess keratin can provide almost immediate relief [15]. It is recommended to follow debridement with the placement of metatarsal padding in the shoe under the lesion to help in slowing the development

of further hyperkeratosis and provide symptomatic relief. Silicon toe sleeves are very effective because not only do they provide the necessary padding, but also they help soften the lesion. If the patient isfeeling irritation from the friction between two toes after the debridement, a foam toe spacer can be used [16]. The use of these devices may require a shoe with a more commodious toe box to avoid the risk of pressure ulcers.

Skin infections Obesity increases the incidence of cutaneous infections, including candidiasis, intertrigo, candida folliculitis, furunculosis, erythrasma, tineacruris, and folliculitis. Less common infections include erysipelas, cellulitis, necrotizing fasciitis, and gas gangrene.

Intertrigo-Intertrigo is not primarily an infectious disease, it is included in this category because of the frequent coexistence of yeast, bacteria, or fungi within the plaques. The macerated erythematous plaques developing within skin folds, such as inframammary, genitocrural, axillary, and abdominal folds, are as a result to both increased friction as well as moisture within these areas. Obese patients have increased skin folds and sweat more profusely after becoming overheated due to thick layers of subcutaneous fat, thus increasing both frictional and moisture components.38 There is a direct relationsheep between the severity of obesity and intertrigo.66

In the study conducted in past it was believed that Malassezia species could cause secondary infections in patients with intertrigo; however, there is currently a debate among dermatologists as to whether these are true secondary infections or whether they are the result of concomitant seborrheic dermatitis. Examinatin with Wood's

lamp will reveal a characteristic green fluorescence with pseudomonas infection and a coral-red fluorescence with infection with corynebacteriumminitissumum [20].

There are many recent evidence which points to Group A Streptococcus as an underdiagnosed culprit of secondary infection in obese patients suffering from intertrigo. Clinical signs that leads to suspicion of infection with Group A Streptococcus include a foul smell, lack of candida's characteristic papular or pustular lesions, and the presence of a much more delineated, fiery red lesion [19]. Bacterial culture can be helpful for determining the specific cause of secondary infection and antibiotic sensitivity test should also be obtained to help guide subsequent therapy.

Bacterial infections - Obesity may be associated with a multitude of bacterial infections, ranging from less complicated infections such as folliculitis and furunculosis142 to more serious infections including erysipelas143,144 and necrotizing fasciitis145-148 that require hospitalization. Erysipelas is commonly caused by streptococcal species and has been known to complicate lymphedematous limbs.149 Obesity without coexistent lymphedema has also proven to be an independent risk factor for erysipelas.144 Necrotizing infections of the skin include necrotizing cellulitis and necrotizing fasciitis. Necrotizing fasciitis is a deep gangrenous infection of subcutaneous tissue that leads to progressive destruction of fascia and fat, but occasionally spares the skin.152 Characteristics of necrotizing cellulitis and necrotizing fasciitis include extensive tissue destruction, systemic toxicity, and high mortality.

Hidradenitissuppurativa - Hidradenitissuppurativa is a chronic recurrent disease manifested by abscesses, fistulas, and scarring tracts along predominantly apocrine gland bearing skin . It is a common skin disease affecting an estimated 2% of the

population.154 The etiology of hidradenitissuppurativa is still poorly understood; however, it appears to be caused primarily by follicular occlusion with secondary involvement of the apocrine glands.155 Obesity has not been consistently found to be associated with this suppurative disease,156 but likely exacerbates underlying disease by increasing shearing forces and androgen effects.155 Studies attempting to demonstrate primary hyperandrogenism as a cause of the disease have been complicated by the fact that the majority of these patients are obese,157 which further supports the role of obesity as an exacerbating factor.

Psoriasis Recent data demonstrate a significantly higher prevalence of obesity among psoriasis patients than in the general population.170 Inverse psoriasis appears to be particularly associated with obesity and sometimes can be indistinguishable from intertrigo in obese patients.170 Obesity appears to be associated with morbidity of psoriasis.172-174 Sakai et al175 analyzed a cohort of 169 psoriasis patients over more than 10 years and found that elevated BMI ([25) was significantly associated with long-term prognosis of psoriasis. In a large case-control study with 560 psoriasis patients, Naldiet al176 found that BMI > 30 was associated with an odds ratio of 1.9. Other studies have also found a significant association between obesity and increased morbidity of psoriasis.177,178

Keratosis pilaris- Keratosis pilaris is usually seen as small, perifollicular, horny papules on the outer aspects of the arms and legs . Keratosis pilaris is one of the minor clinical signs of atopic dermatitis, but this benign dermatosis is more common in patients with higher BMIs.12 It may be linked to insulin resistance, but this association has not been confirmed.4

Adiposa Dolorosa- Adiposadolorosa, other name of adipose dolorosa is Dercum's disease, is a rare multisystem disease which is most commonly seen in obese, perimenopausal females. The disease has four characteristic l symptoms: 1) generalized obesity, 2) multiple, painful lipoma-like masses 3) psychiatric illness including dementia, depression, confusion, and emotional lability and 4) chronic fatigue [33]. Patients suffering from adiposa dolorosa will mainly complain of very severe pain which is often out of proportion to what would be expected by the given physical findings. Most cases appear spontaneously, the occurrence of the disease in multiple family members has been reported [34]. These patients often are hyperalgesic to palpation of the lipoma-like masses.

The patients often cannot identify individual masses as painful prior to physical examination, palpation of masses tends to elicit pain in some of them. Common site of masses include the abdomen, thighs, and upper arms; however, lesions have been documented everywhere except for the head. The skin over these masses are characterized by dilated superficial veins.

Diagnosis of this disease presents a difficulty in diagnosis particular challenging as the multiple associated psychiatric symptoms tend to obscure the clinical picture. In addition to that , the lack of inflammatory, autoimmune, or general laboratory markers that might aid in diagnosis contribute to the difficulty in accurate diagnosis. It is worth noting that biopsy is likely not of great use in these patients as multiple studies have found that the masses of adiposa dolorosa are indistinguishable from the histologic findings of the common lipoma.The diagnosis is generally made by magnetic resonance imaging (MRI) which will usually reveal the following pathognomonic findings: (1) multiple ill-defined, oval shaped masses that appear to

bluish on unenhanced MRI; (2) shows no enhancement following the administration of gadolinium contrast; (3) decreased T1-weighted signal; and (4) increased water-sensitive sequences [35]. , Rest of the cases are diagnosed via ultrasound by identifying superficial oblong shaped subcutaneous fatty nodules that are hyperechoic and do not produce an increased color Doppler echo.

MATERIAL AND METHODS

All Obese patients visiting the OPD, of KLE Dr.Prabhakar Kore Charitable Hospital were included in the study. The study population consist of 100 obese patients attending OPD clinic in KLE Dr.Prabhaka rkore charitable Hospital. The duration of case collection study was from January 2017 to December 2017.Data were recorded on a sheet designed for such a purpose. The cases were taken on OPD basis and includes both male and female. The cases taken were more than 11 years of age .The age range of cases were 11 years to 69 years .After taking consent a detail dermatological examination was done .Apart from dermatological examination , General examination and systemic examination of the patient was also done. Demographic details, height, weight and waist circumference of each and every patient was measured. Weight and Height were determined to calculate body mass index [BMI].At the institute we use a BMI >30kg/m2 as a crieteria for obese patient .Patient were included in three grades of obesity .Grade 1 obesity BMI 30-35 kg/m2,Grade 2 obesity BMI 35-40kg/m2 , Grade 3 obesity BMI >40 kg/m2.The study was started after the ethical clearance. Cutaneous manifestation secondary to Diabetes Mellitus and pregnancy was not included in the study as well as cutaneous change secondary to

any drugs was also not included .Routine investigation of the patient like – FBS ,if fasting blood blood sugar is raised than HBA1C.The Lipid profile of the patient was also done .

RESULTS

Table 1: Gender wise distribution of patients

Sex	Number of patients	% of patients
Male	58	58.00
Female	42	42.00
Total	100	100.00

Figure 1: Gender wise distribution of patients

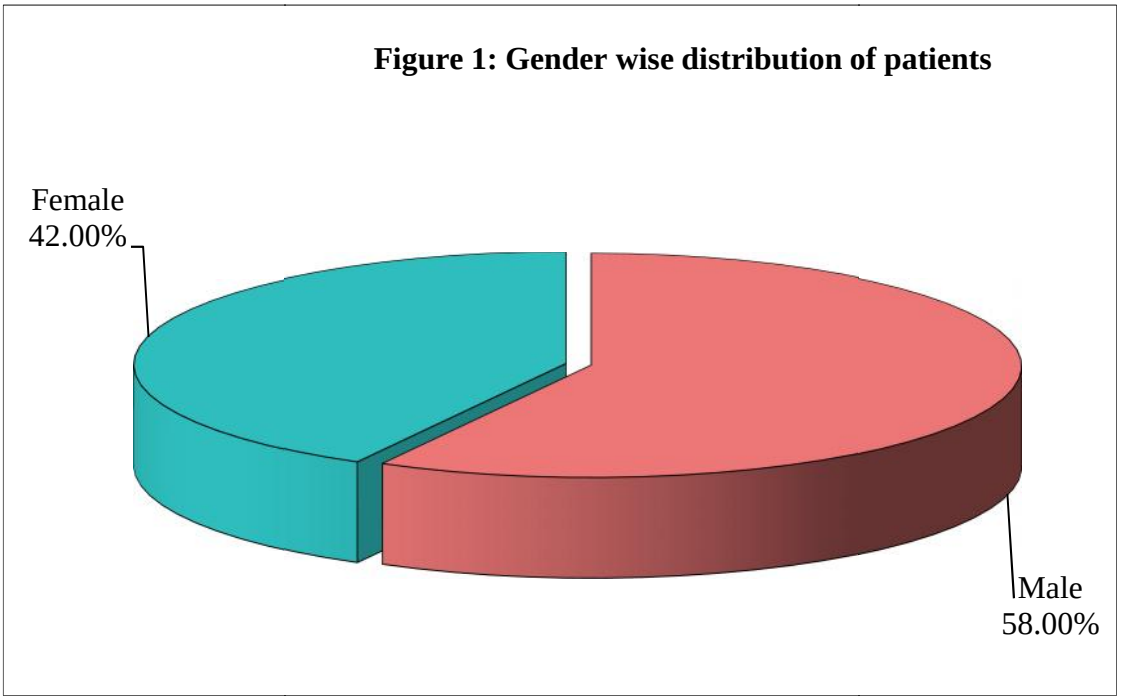


Table 2: Age groups wise distribution of patients

Age groups	Number of patients	% of patients
<=20yrs	11	11.00
21-40yrs	52	52.00
41-60yrs	34	34.00
>=61yrs	3	3.00
Total	100	100.00
Mean age	37.11	
SD age	12.53	

Figure 2: Age groups wise distribution of patients

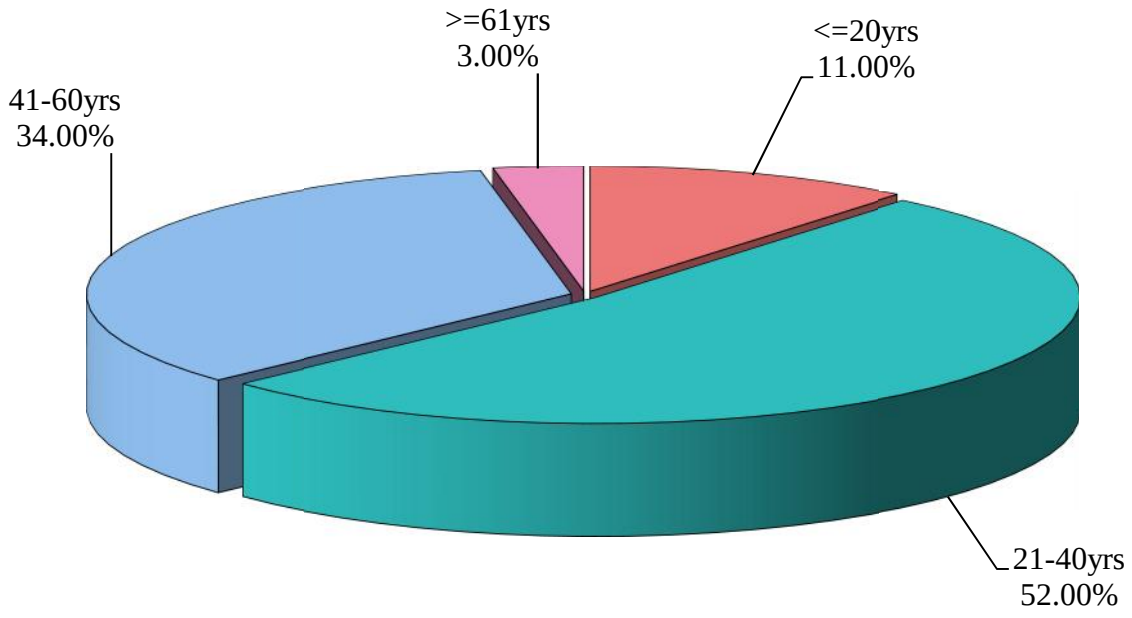


Table 3: BMI groups wise distribution of patients

BMI groups	Number of patients	% of patients
30-33 kg/m^2	61	61.00
34-37 kg/m^2	16	16.00
38-45 kg/m^2	23	23.00
Total	100	100.00
Mean BMI	33.83	
SD BMI	3.86	

Figure 3: BMI groups wise distribution of patients

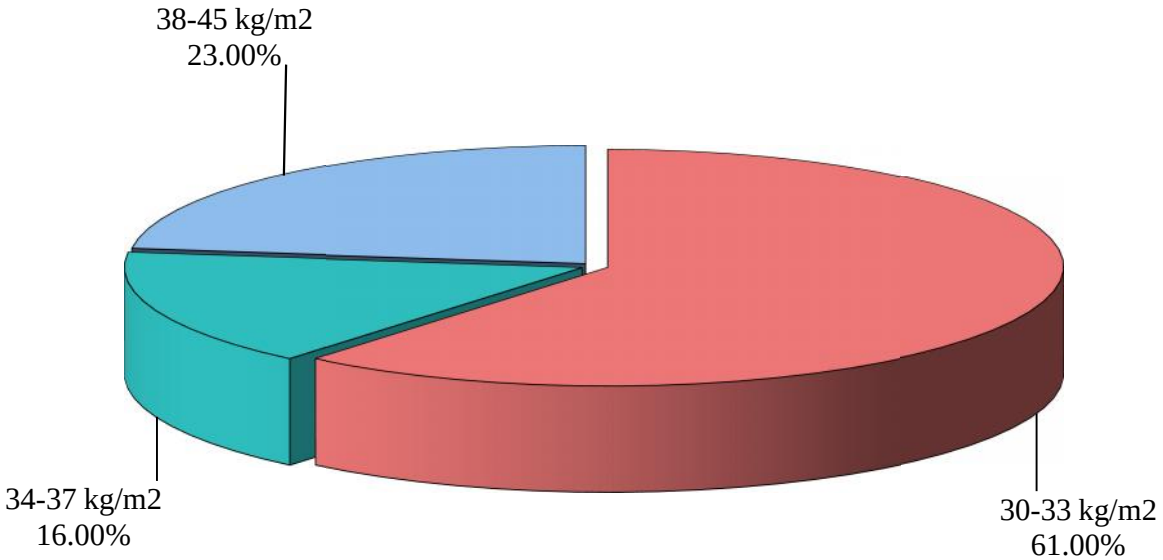


Table 4: Waist circumference wise distribution of patients

Waist circumference	Number of patients	% of patients
38-42	66	66.00
43-45	31	31.00
>=46	3	3.00
Total	100	100.00
Mean BMI	39.85	
SD BMI	2.97	

Figure 4:Waist circumference wise distribution of patients

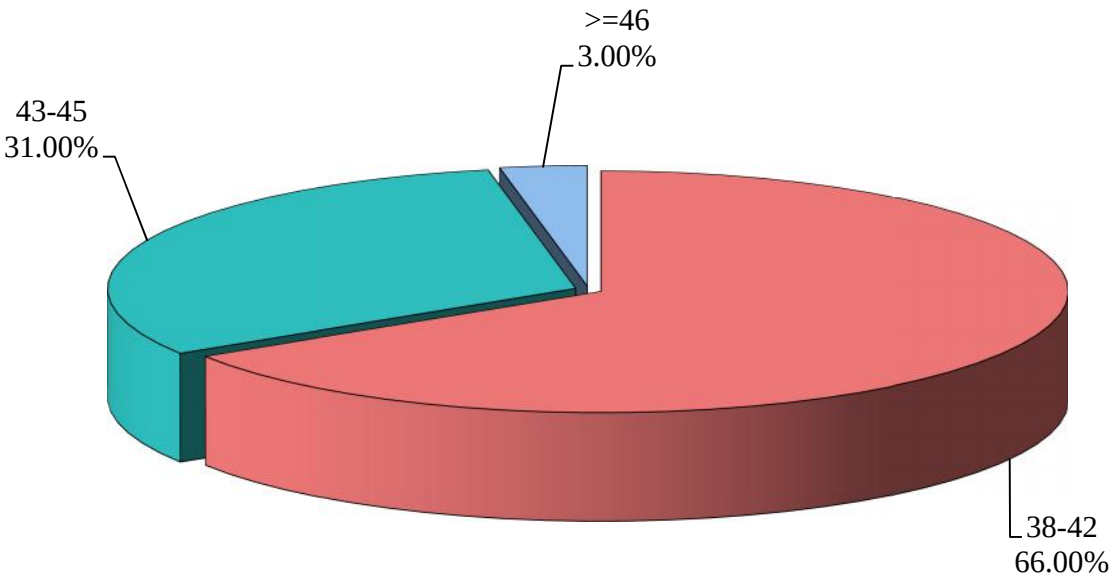


Table 5: FBS, HBA1C wise distribution of patients

FBS, HBA1C	Number of patients	% of patients
Increased	1	1.00
Normal	99	99.00
Total	100	100.00

Figure 5:FBS, HBA1C wise distribution of patients

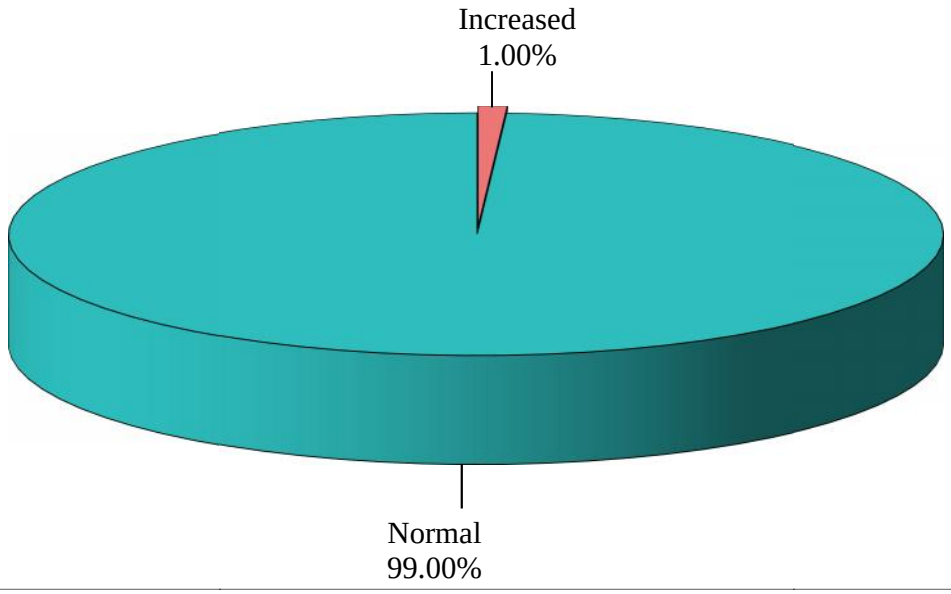


Table 6: Lipid profile wise distribution of patients

Lipid profile	Number of patients	% of patients
Increased	2	2.00
Normal	98	98.00
Total	100	100.00

Figure 6:Lipid profile wise distribution of patients

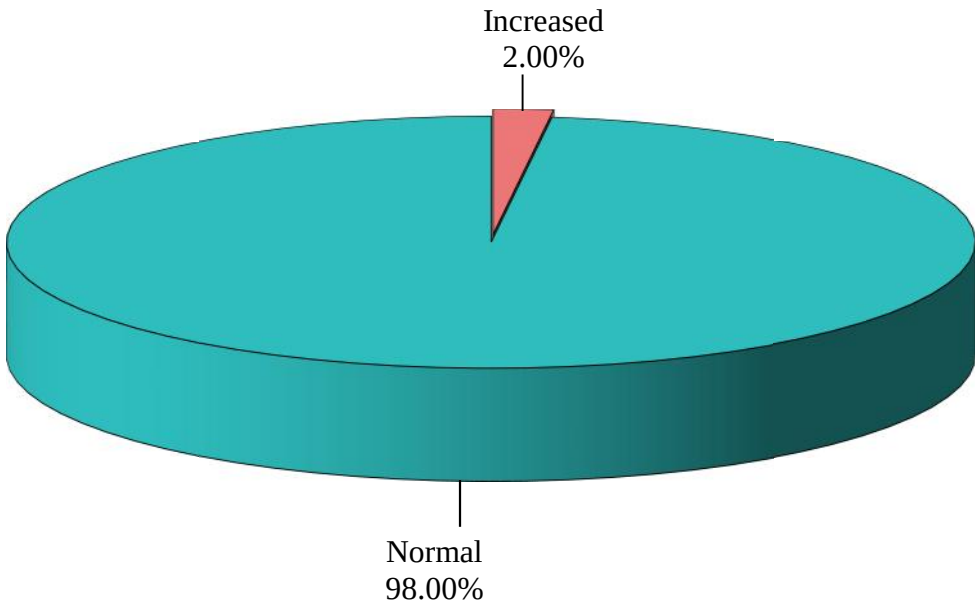


Table 7: Cutaneous manifestation wise distributions of patients

Cutaneous manifestation	Number of patients	% of patients
AcanthosisNigricans	59	59.00
Striae	25	25.00
Acrochordon	33	33.00
Bacterial infection	2	2.00
Hirsuitism	1	1.00
Tineacorporis	21	21.00
Fungal infection	2	2.00
Keratosis Pilaris	1	1.00

Figure 7:Cutaneous manifestation wise distributions of patients

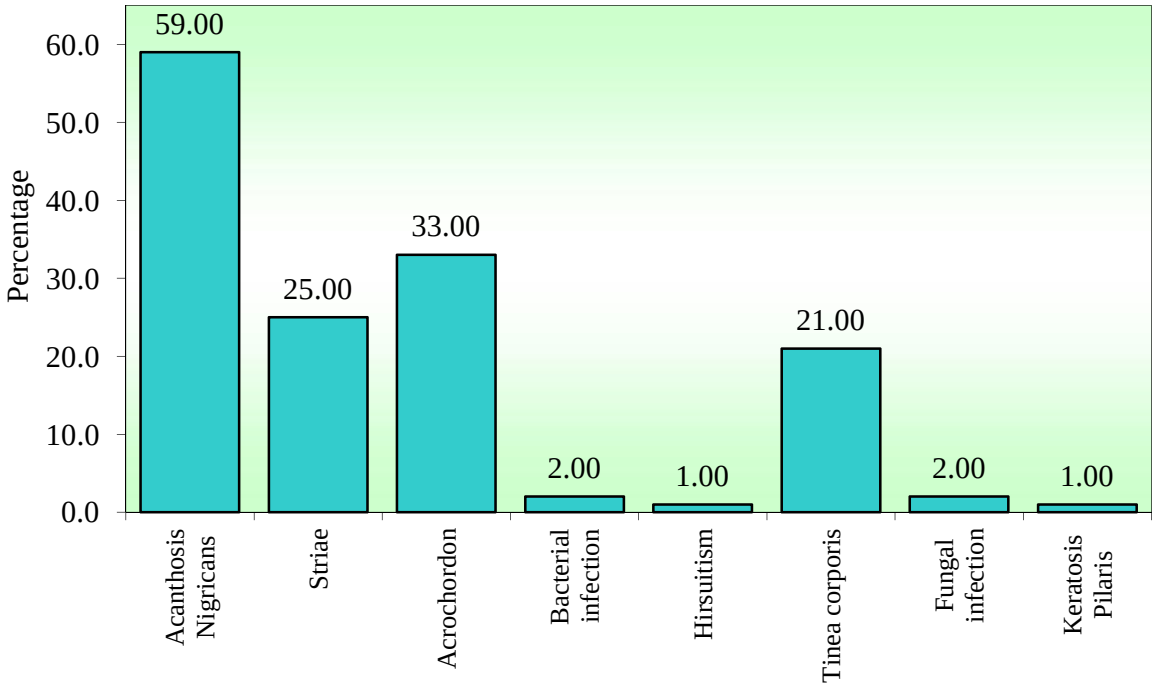


Table 8: Association between gender and cutaneous manifestation of patients

Cutaneous manifestation	Male	Female	Total	Chi-square
AchanthosisNigricans	30	29	59	χ^2=2.018 P = 0.155
Striae	11	14	25	χ^2=6.314 P = 0.012*
Acrochordan	18	15	33	χ^2=0.241 P = 0.623
Bacterial infection	2	0	2	χ^2=0.242 P = 0.623@
Hirsuitism	0	1	1	χ^2=0.027 P = 0.871@
Tineacrusis	16	5	21	χ^2=3.611 P = 0.050*
Fungal infection	2	0	2	χ^2=0.242 P = 0.623@
Keratosis Pilaris	1	0	1	χ^2=0.027 P = 0.871@

*p<0.05, @applied Yates corrected chi-square

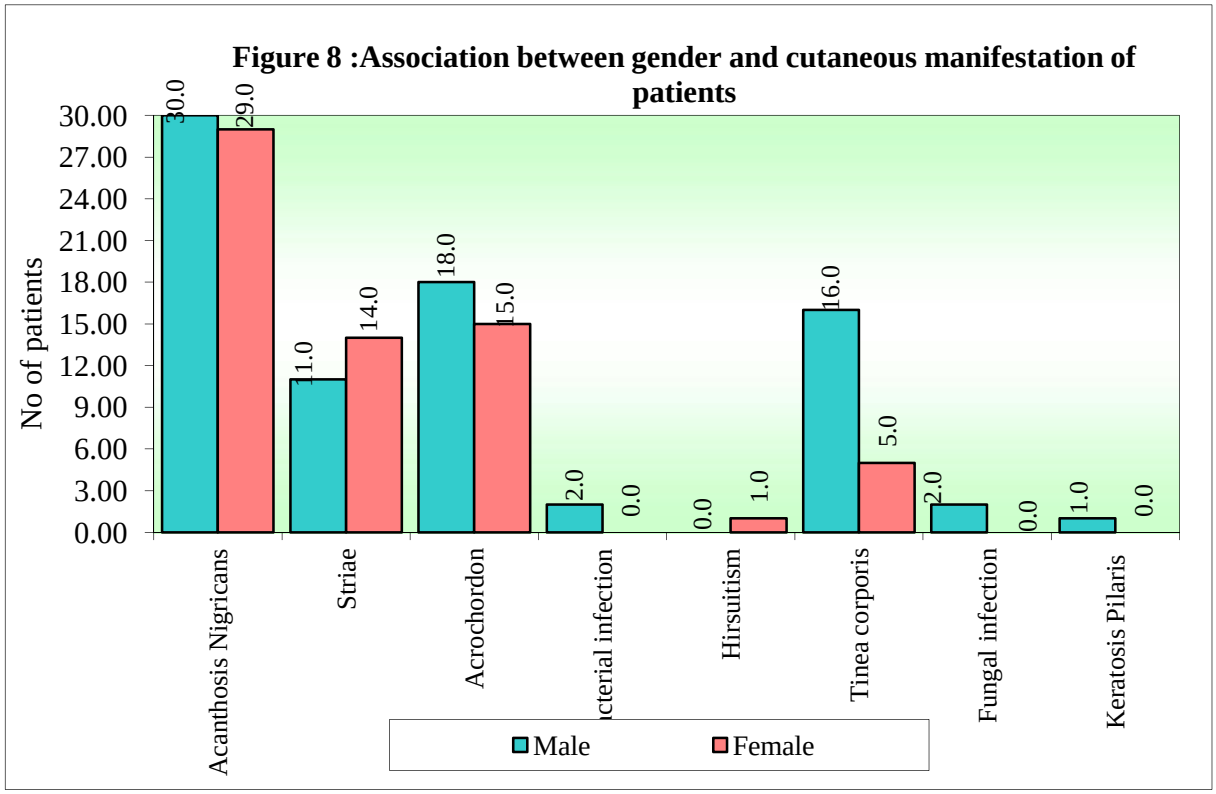


Table 9: Association between age groups and cutaneous manifestation of patients

Cutaneous manifestation	<=20 yrs	21-40 yrs	41-60 yrs	>=61 yrs	Total	Chi-square
Achanthosis Nigricans	11	25	23	0	59	χ^2=12.350 P = 0.006
Striae	4	12	9	0	25	χ^2=1.899 P = 0.594
Acrochordan	1	22	8	2	33	χ^2=7.799 P = 0.050
Bacterial infection	0	2	0	0	2	χ^2=1.884 P = 0.597
Hirsuitism	0	0	1	0	1	-
Tineacrusis	2	13	6	0	21	χ^2=1.582 P = 0.663
Fungal infection	0	0	2	0	2	χ^2=3.962 P = 0.266
Keratosis Pilaris	0	0	0	1	1	-

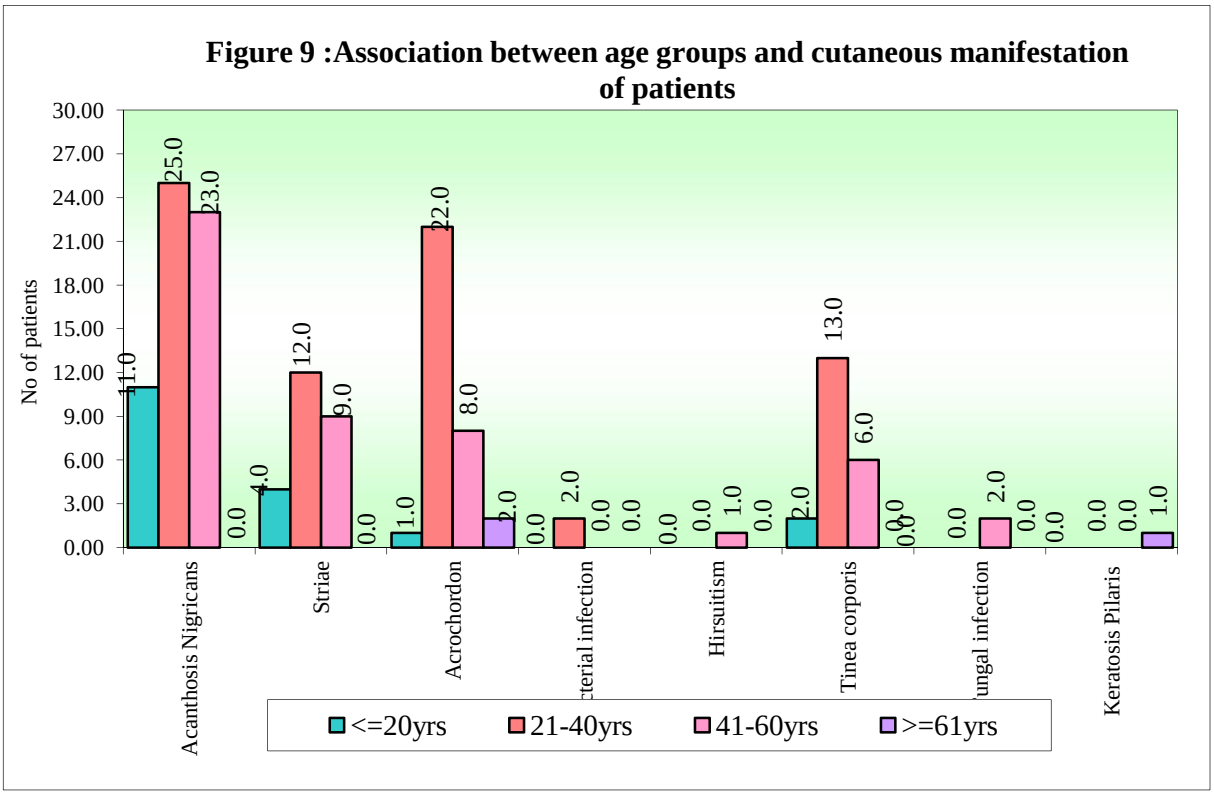


Table 10: Association between BMI groups and cutaneous manifestation of patients

Cutaneous manifestation	30-33 kg/m^2	34-37 kg/m^2	38-45 kg/m^2	Total	Chi-square
AchanthosisNigricans	37	9	13	59	χ^2=0.283 P = 0.868
Striae	17	4	4	25	χ^2=0.978 P = 0.613
Acrochordan	15	8	10	33	χ^2=5.185 P = 0.075
Bacterial infection	1	1	0	2	-
Hirsuitism	0	0	1	1	-
Tineacrusis	13	2	6	21	χ^2=1.059 P = 0.589
Fungal infection	2	0	0	2	-
Keratosis Pilaris	0	0	1	1	-

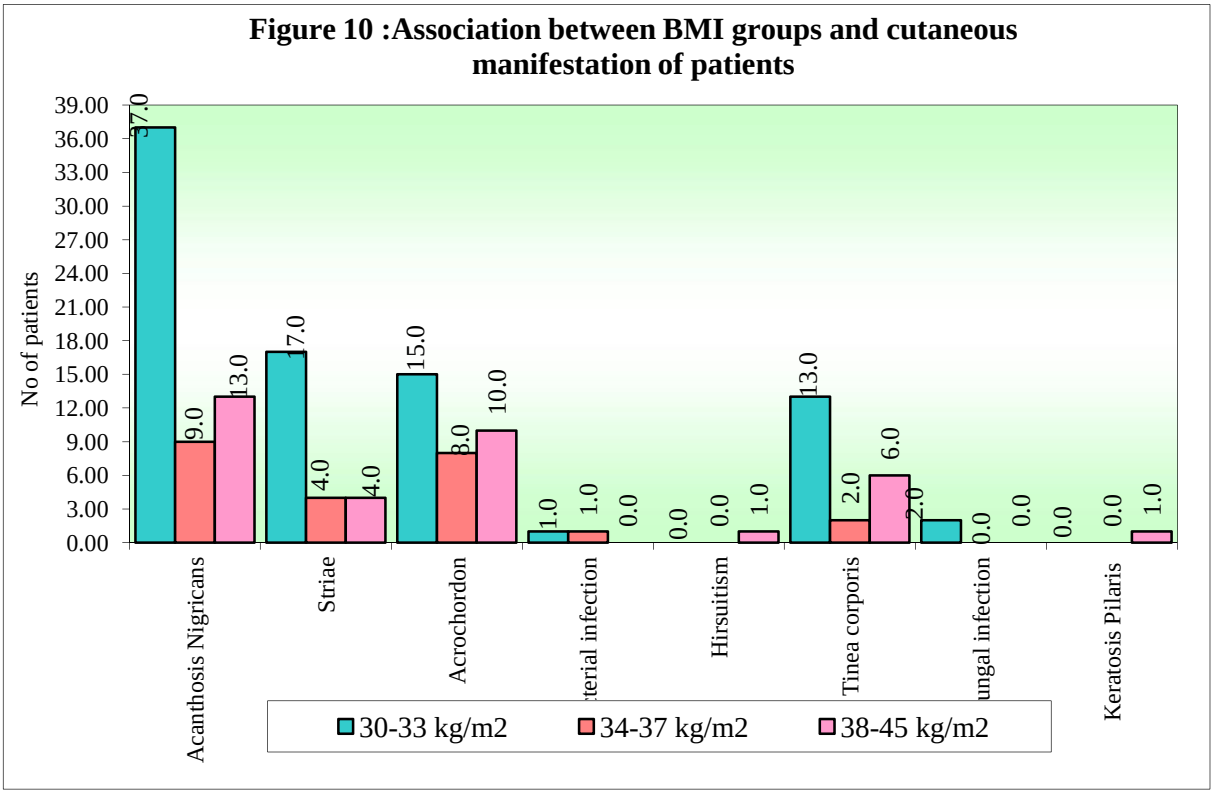


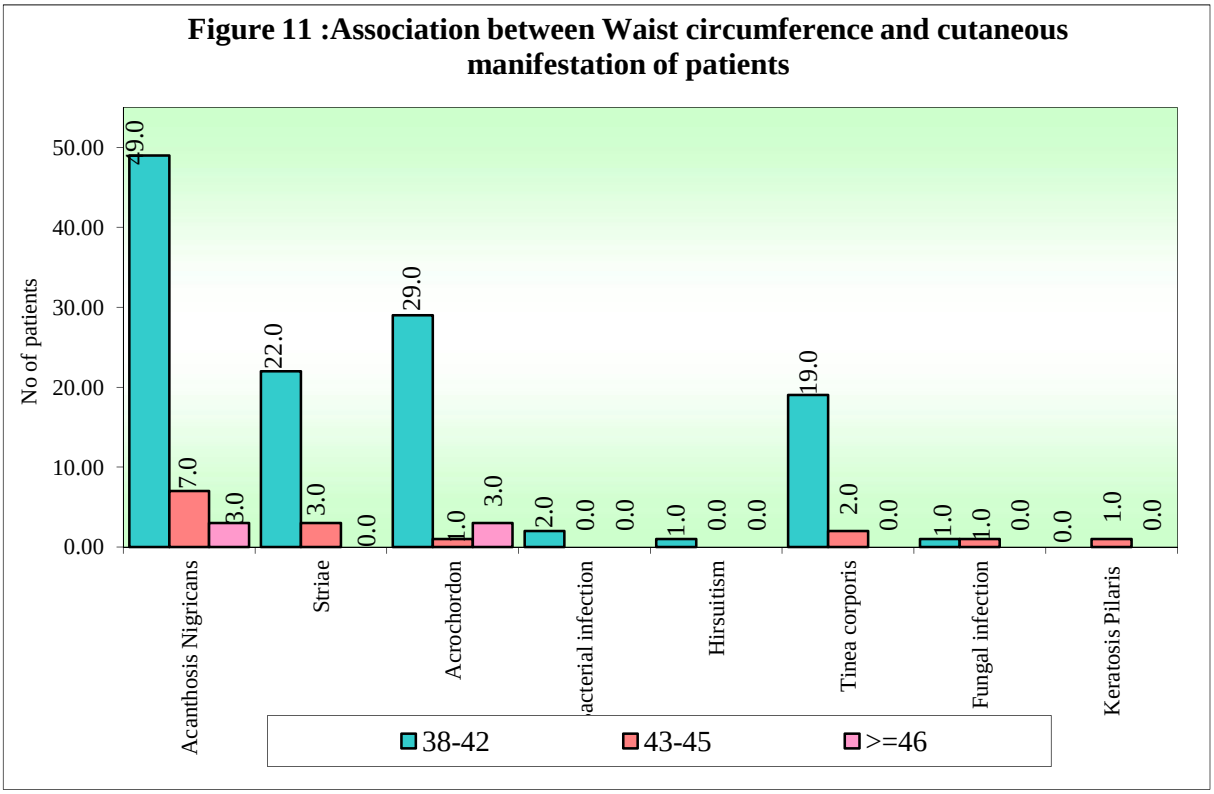
Figure 10 :Association between BMI groups and cutaneous manifestation of patients

Table 11: Association between Waist circumference and cutaneous manifestation of patients

Cutaneous manifestation	38-42	43-45	>=46	Total	Chi-square
AchanthosisNigricans	49	7	3	59	29.852 P = 0.0001*
Striae	22	3	0	25	7.326 P = 0.026*
Acrochordan	29	1	3	33	22.093 P = 0.0001*
Bacterial infection	2	0	0	2	1.051 P = 0.591
Hirsuitism	1	0	0	1	-
Tineacrusis	19	2	0	21	7.165 P = 0.028
Fungal infection	1	1	0	2	-
Keratosis Pilaris	0	1	0	1	-

*p<0.05

Figure 11 :Association between Waist circumference and cutaneous manifestation of patients



DISCUSSION

The present study is a hospital based cross sectional study done during a period of one year from November 2016 to October 2017 in KLE Dr.Prabhakar Kore Charitable Hospital .In this study 100 obese patient were taken for study on OPD basis .In our study out of 100 obese patient .There were 58 males and 42 female patient. Acanthosis Nigrican is the most common finding (59%) followed by Acrochordon (33%), Striae (25%), Fungal infection (21%). Acanthosi s Nigricans are mostly associated with Acrochordon.

Obesity, previously considered as a health problem of industrialized and developed countries, has been accepted and recognized as an emerging public health problem of developing countries like India now. The factors which leads to increase in incidence of obesity include rising urbanization, dietary habits and changing life style. Cutaneous manifestations of obesity show direct relationship with increasing BMI. In this study, we used the criteria by WHO BMI >30kg/m2. The mean BMI recorded in our patients was 33.83kg/m2. Hermanns et al.11 in their study of 126 patients, reported mean BMI to be 37.4 kg/m2. Therefore, it can be appreciated that the finding in our study is comparable to the study aforementioned.11

The most common finding in our study was acanthosisnigricans (59%), that was significantly more in males as compared to female.

Ahsan et al.13 have reported a higher frequency of acanthosisnigricans. The frequency of various cutaneous manifestation can vary from one study to another depending upon the setting and design of the study. Hermannset al.11 documented that acanthosisnigricans is associated with obesity, endocrine disorders including diabetes mellitus, hyperinsulinaemia, insulin resistance and metabolic syndrome.

Boza et al. 12 also reportedthat there is association between obesity and acanthosisnigricans. Boza et al. 12 did not mention any association of acanthosisnigricans with diabetes or insulin resistance and claimed that obesity is an independent factor for acanthosisnigricans. This may because of racial and genetic differences.12

Striae had a frequency of 25% in our study and were significantly associated with grade II obesity. The frequency of striae was not statistically significant gender wise. Ahsanet al.13 mentioned a higher frequency of striae. The frequency for striae can vary from one study to another depending upon the setting and design.

Acrochordons were a feature in 33% of the enrolled subjects. Ahsanet al.13 mentioned a significantly higher frequency of acrochordons (52%). The difference in frequency canbe influenced by the sampling technique. Jowkar et al.14 has documented an association between acrochordons and hyperinsulinaemia in non-diabetics. So, the association of acrochordons and obesity is in agreement with the reports in literature.14

Several factors including greaterbodysurface area, increased friction and moisture predispose obese people to infections. Among the skin infections (25%), fungal infections were the most frequent (23%), followed by bacterial infections (2%). On the contrary, Ahsan et al. 13 were not able to correlate the frequency of infections with the grades . The frequency of bacterial and fungal infections was not affected by the grades of obesity. Bozaet al.12 have also found a significant association between obesity and various infections. so, it can be appreciated that the findings in the current study are consistent with the past studies.12,13

In our study, hirsutism (1%). Ahsan et al.13 have reported a frequency of 16% in a similar set of patients. Hirsutismhas been directly associated with hyperinsulinaemia and hyperandrogenism in the past.12,15

Skin care in obese patients demands particular attention because of morbidity, associated systemic diseases and susceptibility to infections.19,20 Limited work has been done on this subject in our part of the world. This study adds to currently available literature from India and allows us comparison with international studies.

CONCLUSION

Obesity is commonly associated with a wide range of cutaneous manifestation such as acanthosis nigricans, striae, hirsutism, skin infections. Other less common associations includes- corns, plantar hyperkeratosis and acne.

SUMMARY

The dissertation –A one year cross sectional study of Dermatological manifestation of obesity in Dr.Prabhakar Kore charitable Hospital is done . A total of 100 obese patient were studied. Cutaneous manifestation secondary to pregnancy, Diabetes Mellitus is not included in the study . Acanthosis Nigricans is the most common manifestation in both male and female. In most of the cases Acanthosis Nigrican and Acrochordon were present in the same patient. Acrochordon was the second most common finding followed by Fugal infection, Striae ,bacterial infection, Hirsuitism and Keratosis Pilaris. The cutaneous manifestation of obesity was in direct association of BMI. The incidence of obesity in India is increasing and so cutaneous manifestation with obesity is also increasing .More study require in the futue for understanding pathophysiology of cutaneous manifestation of obesity .

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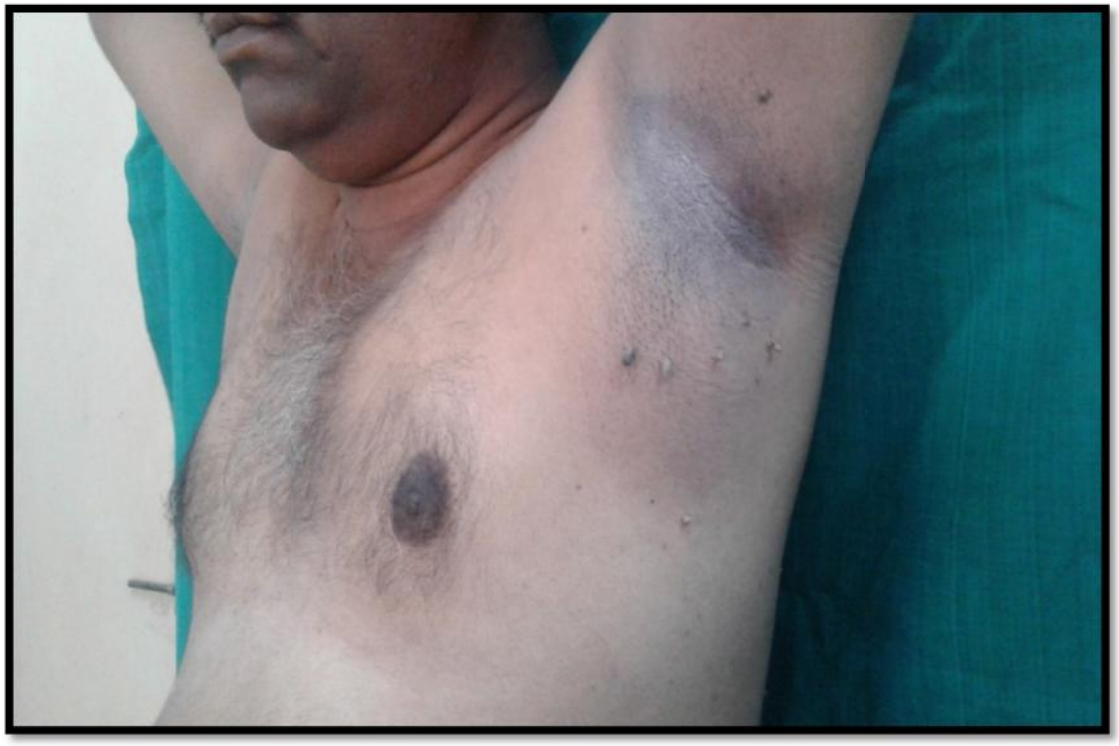
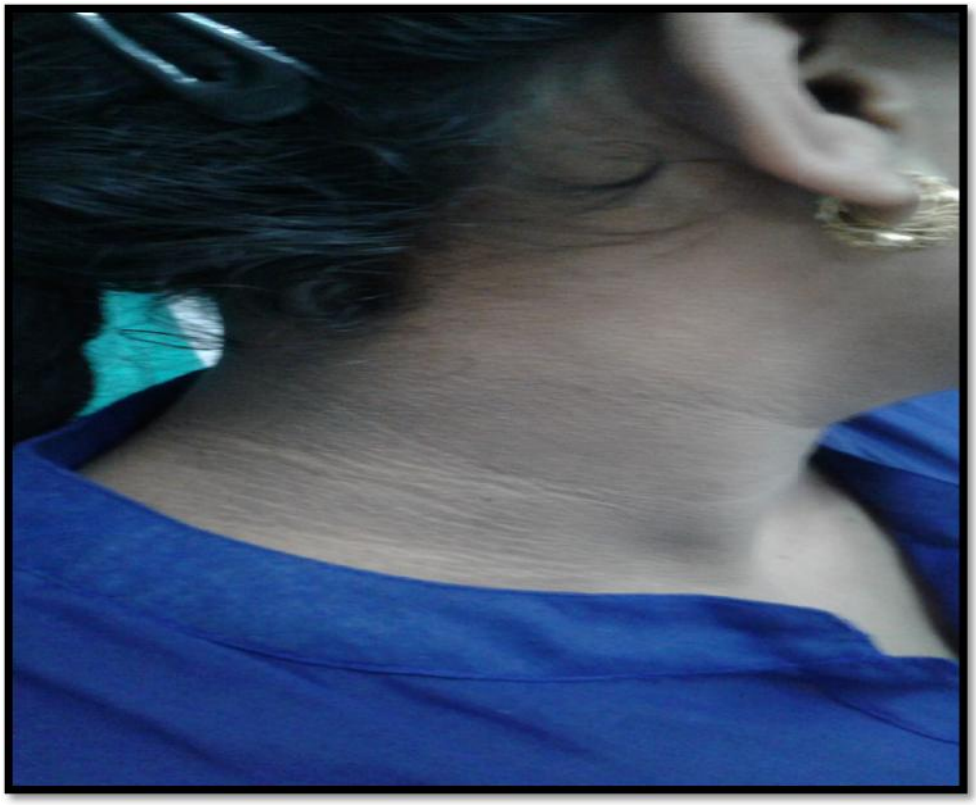
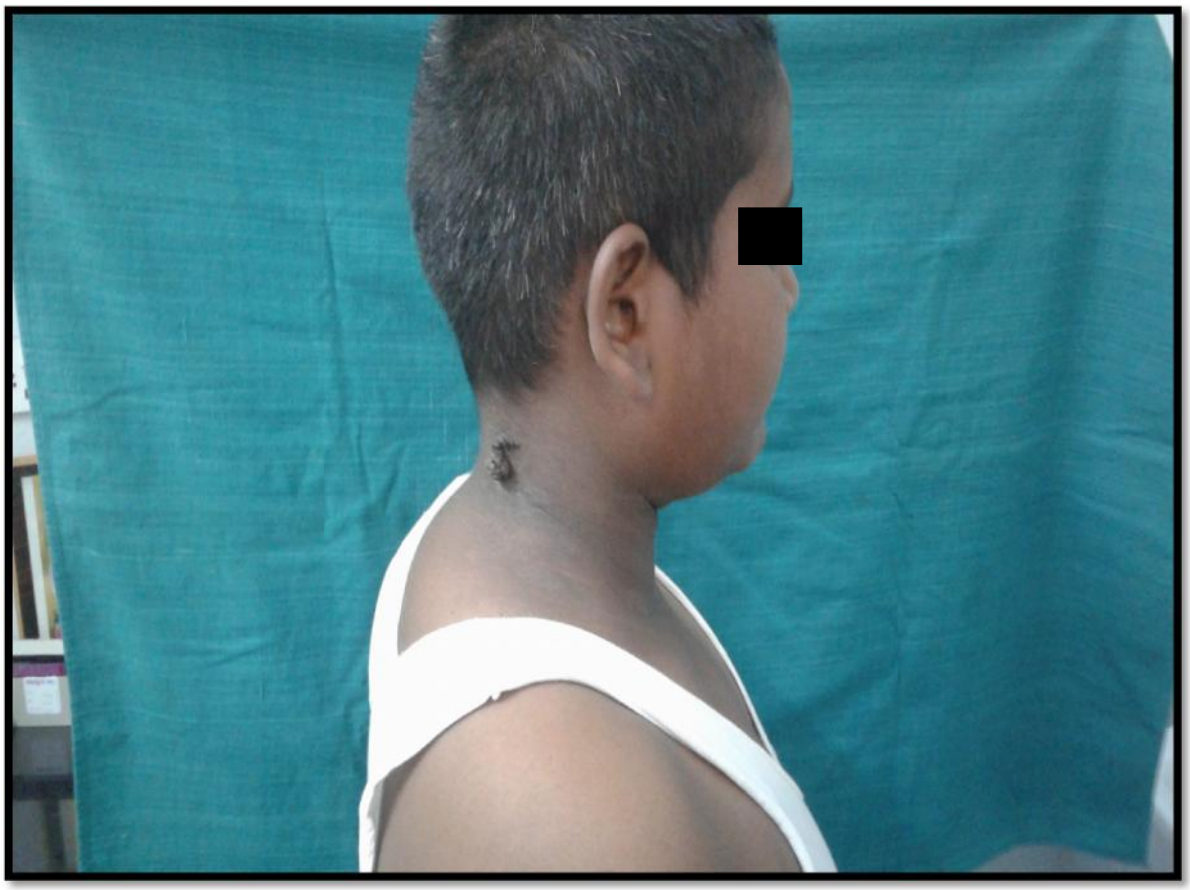
ANNEXURE I - PHOTOGRAPHS



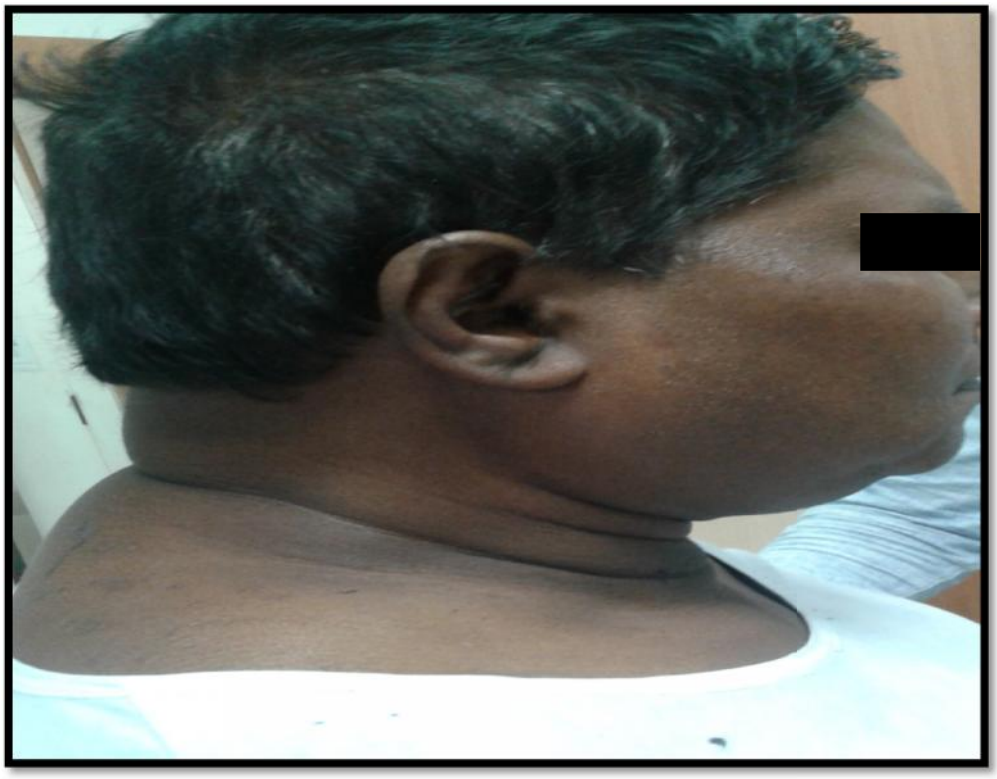
Photographs 1: Acrochordon



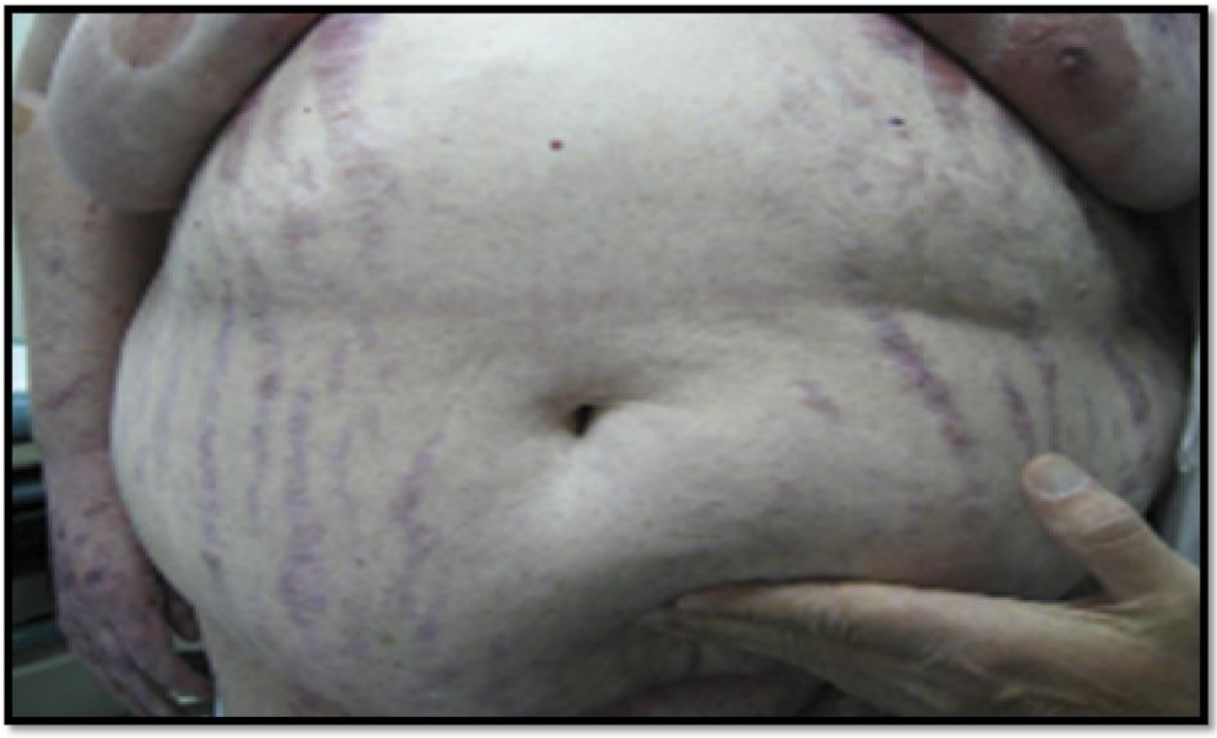
Photographs 2: Acanthosis nigricans



Photographs 3: Acrochordon



Photographs 4: Acanthosis nigricans



Photographs 5: Striae

ANNEXURE II - INFORMED CONSENT FORM

I.D.NO. | | | |

Title of the study:A one year cross sectional study of dermatological manifestation of obesity in KLE,S prabhakarkore hospital Belagavi Karnataka.

The study is conducted by Dr. DHANANJAY PostGraduate (M.D)student in Dermatology under the guidance of DR. B. SIDDARAMAPPA MDProfessor Department of Dermatology,Venereology and Leprosy,JNMC, BELAGAVI.

Respected Sir/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:

This study will help us in finding out the pattern of frequency of dermatological manifestation of obesity.This study will help in finding the association of grades of obesity and dermatological manifestation of obesity .

Procedure and treatment:

Should you choose to participate, you will be asked to give a detailed history of your disease, undergo a physical examination, recording waist circumference, weight and blood pressure and consent for investigations like estimation of fasting blood sugar levels and fasting lipid profile levels, which requires drawing of 2 ml blood.

Risks and benefits:

While drawing of 2 ml blood for investigations like estimation of fasting blood sugar and fasting lipid levels, you may experience slight pain due to needle prick.However all necessary steps and precautions will be taken to ensure your safety. The result of you taking part in this research would help health care providers towards a better understanding of this disease, and thus we will be able to provide improved patient care.

Alternatives:

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

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 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.

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 of your current disease, nor your future relations with the doctor or the hospital. In the event if you suffer any physical injury as the result of your participation in this study, you may contact Dr. dhananjayTelephone No. 7996683565 orDR. B. SIDDARAMAPPA MD Telephone No: 9341100796. In the event of an emergency, you should contact KLE'S Dr. PrabhakarKore Hospital and MRC on Telephone No. 08312473777.

In case you need further information regarding your rights as a study participant, you may please contact Dr. Ganga.S.Pilli,Chairman of the ethical committee, J N Medical College, Belagavi.

STATEMENT OF CONSENT

I.D.NO: | | | |

I Mr/Ms/Mrs ---------- volunteer and consent to participate in this study. I have read the consent document orit 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 name:

Signature or left thumb print of participant:

Witness name:

Signature of witness:

Signature of the investigator:

Date:

STATEMENT OF ASSENT

I.D.NO: | | | |

I Mr/Ms/Mrs ----------

parent/guardian/ward/ of---------- consent to enrol my daughter to participate in this study. I have read the consent document orit has been read to me in my vernacular language. I give my acceptance on behalf of my daughter for her participation 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 name:

Participant's parent/guardian name:

Signature or left thumb print of parent/guardian of participant:

Witness name:

Signature of witness:

Signature of the investigator:

Date:

ANNEXURE III - PROFORMA

DERMATOLOGICAL MANIFESTATION OF OBESITY

CASE NO

OP NO

IP NO

DATE

NAME

AGE

GENDER

DIAGNOSIS

OCCUPATION

ADDRESS WITH PHONE NUMBER

PRESENTING COMPLAINTS

PAST HISTORY

K/C/O MEDICAL DISORDER HTN a)present b)absent

FAMILY HISTORY

PERSONAL HISTORY 1. DIET A) VEGETARIAN B)MIXED

2.APPETITE A)NORMAL B)REDUCED

3.SLEEP A)ADEQUATE B)DISTURBED

4. BOWEL/BLADDER A)NORMAL B)ALTERED

ALCOHOL A)PRESENT B)ABESENT

STRESS A)PRESENT B)ABESENT

GENERAL PHYSICAL EXAMINATION

BUILT 1)POOR 2)MODERATE 3)GOOD

VITALS 1)PULSE 2)BP 3)TEMPERATURE

HEIGHT cm weight kg BMI kg/m^2 wc

Pallor icterus clubbing cyanosis lymphadenopathy oedema

SYSTEMIC EXAMINATION

RESPIRATORY SYSTEM 1) NORMAL 2) ABNORMAL;

SPECIFY If SO

CARDIOVASCULAR SYSTEM 1) NORMAL 2) ABNORMAL;

SPECIFY If SOPER

ABDOMEN 1) NORMAL 2) ABNORMAL;

SPECIFY If SO

CENTRAL NERVOUS SYSTEM 1)NORMAL 2)ABNORMAL;

SPECIFY If SO

INVESTIGATION

FBS- mg/dl

PPBS-mg/dl

HbA1c

GTT if needed

LIPID PROFILE -TC- mg/dl

TG - mg/dl

HDL- mg/dl

LDL- mg/dl

VLDL - mg/dl

Dermatological manifestations in obese patient.

1.Acanthosisnigricans	yes [ ]	no [ ]
2.Striae	yes [ ]	no[ ]
3.Acrochordons	yes [ ]	no[ ]
4.Fungal infections	yes [ ]	no[ ]
5.Bacterial infections	yes [ ]	no[ ]
6.Viral infections	yes [ ]	no[ ]
7.Hirsutism	yes [ ]	no[ ]

8.Xanthoma	yes [ ]	no[ ]
9.Corn	yes [ ]	no[ ]
10.Acne	yes [ ]	no[ ]
11.Varicosities	yes [ ]	no[ ]
12.Plantar hyperkeratosis	yes [ ]	no[ ]
13.Miscellaneous	yes [ ]	no[ ]

SL NO	0P/IPNO	AGE	SEX	CUTANEOUS MANIFESTATION	BMI KG/M2	FBS, HBA1C	LIPID PROFILE	WC
1	4318542	40	F	AN,ACR.	32.9	N	N	40
2	4242808	40	F	TINEA CORPORIS	30.05	N	N	38
3	4100638	45	M	T. CRURIS	30.57	N	N	40
4	117673	42	F	AN, HIRSUITISM	37.9	INC.	N	40
5	4100536	40	M	T. CRURIS	31.2	N	N	40
6	4348033	15	M	AN,ACRO	35	N	N	42
7	4100236	40	F	AN,STRIAE DISTEN	35	N	N	40
8	4323168	40	M	AN,STRIAE,ACR	36.09	N	N	41
9	799684	45	M	ACRO	32.87	N	N	46
10	4416967	50	F	AN,ACRO	44	N	INC.	50
11	813409	63	M	AN, ACR	34.375	N	N	42
12	4385790	48	M	ACR	30.3	N	N	40
13	806851	69	M	KP	41.04	N	N	44
14	4416967	50	F	AN,ACRO	44.02	N	INC.	48
15	4146301	18	M	AN,STRIAE	30.31	N	N	44
16	2155748	35	M	AN, FURUNCULOSIS	31.3	N	N	38
17	813459	58	M	AN,ACR	35.05	N	N	42
18	4106156	11	M	AN	31	N	N	38
19	2491673	26	F	AN	30	N	N	38
20	4407590	40	F	AN,ACRO	30	N	N	37
21	813021	32	M	AN,ACRO	31	N	N	42
22	4363002	25	M	T. CORPORIS	32	N	N	44
23	2767266	24	M	AN, STRIAE	30.42	N	N	38
24	2767256	40	M	AN, STRIAE	30.21	N	N	38
25	4363000	25	M	ACRO	32.25	N	N	39
26	813024	32	F	AN	30.6	N	N	40
27	4407591	40	F	T.CRURIS	30.5	N	N	38
28	2767264	24	M	AN,ACRO	30.1	N	N	36
29	4106155	28	M	AN,ACRO	32.8	N	N	38
30	4419803	32	M	AN, ACRO	32.01	N	N	40
SL.NO	**OP/IP NO**	**AGE**	**SEX**	**CUTANEOUS MANIFESTATION**	**BMI**	**WC**	**FBS, HBA1C**	**LIPID PROFILE**
31	4616340	20	M	AC,AN	34	44	N	N
32	3599246	50	M	T.INCOGNITO	32	42	N	N
33	843812	45	F	AC	31	42	N	N
34	839368	35	F	AC	33	44	N	N
35	3210094	42	M	CAND. INTER	32	35	N	N
36	3210092	44	M	AN	31	40	N	N
37	4616340	32	M	T. CRUSIS	3O	38	N	N
38	4617370	28	F	ACR	31	40	N	N
39	4617343	24	M	AN, ACRO	32	40	N	N
40	4617323	20	F	AN	34	44	N	N
41	3210084	26	F	T. CRUIS	34	43	N	N
42	3310084	28	M	ACR,AN	30	40	N	N
43	3310076	20	M	AN	31	38	N	N
44	3312286	24	M	T.CRUIS	40	30	N	N
45	3212276	38	F	ACR	42	32	N	N
46	4105156	34	F	STRIAE,AN	42	32	N	N
47	4104126	30	F	AN,ACR	40	31	N	N
48	788682	22	M	AN, ACR	39	30	N	N
49	788684	20	M	AN, T.CRUIS	38	36	N	N

SL NO	0P/IPNO	AGE	SEX	CUTANEOUS MANIFESTATION	BMI KG/M2	FBS, HBA1C	LIPID PROFILE	WC
50	788624	28	F	ACR,AN	40	31	N	N
51	788523	28	M	AN	40	32	N	N
52	754522	52	M	AN,ACR	40	31	N	N
53	4385791	54	F	STRAI, ACR	40	31	N	N
54	4375792	48	F	AN, STRIAE	42	32	N	N
55	4475892	52	M	T. CRUIS	38	29	N	N
56	4455792	36	M	T.CRUIS	40	32	N	N
57	4125782	20	F	AN, STRIAE	40	31	N	N
58	4025682	34	M	AN, STRIAE	30	39	N	N
59	4135472	40	F	ACR,AN	31	40	N	N
60	4535672	35	F	AN	32	42	N	N
61	4516342	50	M	AN	32	42	N	N
62	4317352	60	F	AN,ACR	32	44	N	N
63	4416350	65	M	ACR	36	41	N	N
64	3210087	29	F	AN, ACR	36	41	N	N
65	3212286	34	M	T. CRUIS	30	41	N	N
66	3120067	48	M	STRIAE	35	44	N	N
67	3121257	35	F	ACR	36	40	N	N
68	788750	38	M	T. CRUIS	31	42	N	N
69	788659	30	F	AN, STRIAE	32	44	N	N
70	767858	40	F	AN,T. CRUIS	38	42	N	N
71	767758	46	M	AN	30	41	N	N
72	767658	28	F	AN,STR	31	38	N	N
73	767457	27	M	T.CRU	32	40	N	N
74	757447	48	M	T.CRU	31	41	N	N
75	737537	26	F	AN,STR	30	38	N	N
76	3122257	58	M	ACN	32	40	N	N
77	3111257	48	F	AN,STR	31	38	N	N
78	3212247	58	M	ACN	34	40	N	N
79	3412257	28	F	AN,STR	33	40	N	N
80	4125671	50	M	ACN	30	40	N	N
81	4125561	58	M	AN, STR	32	39	N	N
82	4114571	50	F	T.CRU	32	38	N	N
83	41254681	28	F	AN	31	39	N	N
84	41253679	19	M	AN,STR	30	40	N	N
85	41253871	26	M	AN,STR	32	40	N	N
86	4125467	42	F	AN,STR	32	38	N	N
87	4125567	45/F	31	AN, ST	39	N	N	N
88	4135578	48/M	32	T.CRURIS	40	N	N	N
89	4135677	25/M	30	STR	38	N	N	N
90	4137651	58/M	31	ACN	39	N	N	N
91	3413357	45/F	30	AN,STR	38	N	N	N
92	3512247	46/M	32	AN, STR	40	N	N	N
93	3411157	38/M	32	T.CRURIS	41	N	N	N
94	33122356	18	F	AN,STR	35	40	N	N
95	3212267	18/M	34	T.CORPORIS	42	N	N	N
96	3412267	24/F	38	ACRO,AN	40	N	N	N
97	3421157	30/F	35	AN, ACR	42	N	N	N
98	4136651	41/M	40	T.CRUSIS	40	N	N	N
99	4315561	42/F	40	AN	42	N	N	N
100	4115671	38/M	38	AN,ACR	40	N	N	N