A STUDY OF CUTANEOUS MANIFESTATIONS IN OVERWEIGHT AND OBESE PAEDIATRIC POPULATION

By, Dr POOJA KOTIAN

DISSERTATION SUBMITTED TO BLDE (Deemed to be University), VIJAYAPURA

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE OF

MD IN DERMATOLOGY VENEREOLOGY AND LEPROSY

UNDER THE GUIDANCE OF

DR. ARUN C INAMADAR

PROFESSOR

DEPARTMENT OF DERMATOLOGY, VENEREOLOGY & LEPROSY

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

SHRI B M PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA 586103

2024

DOI 10.5281/zenodo.15493925
https://zenodo.org/records/15493926

BLDE (DEEMED TO BE UNIVERSITY)

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

VIJAYAPURA, KARNATAKA

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled " **A STUDY OF CUTANEOUS MANIFESTATIONS IN OVERWEIGHT AND OBESE PAEDIATRIC POPULATION**" is a bonafide and genuine research work carried out by me under the guidance of Dr.Arun C Inamadar, Professor, Department of Dermatology Venereology and Leprosy, at BLDE (Deemed to be University) Shri B.M. Patil Medical College and Research Centre, Vijayapura.

DATE: Dr. POOJA KOTIAN

PLACE: VIJAYAPURA

BLDE (DEEMED TO BE UNIVERSITY)

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

VIJAYAPURA, KARNATAKA

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled " **A STUDY OF CUTANEOUS MANIFESTATIONS IN OVERWEIGHT AND OBESE PAEDIATRIC POPULATION** " is a bonafide and genuine research work carried out by Dr POOJA KOTIAN in partial fulfilment of the requirement for the degree of MD in Dermatology, Venereology and Leprosy.

Dr Arun C Inamdar

DATE:

PLACE: VIJAYAPURA

DR. ARUN C. INAMADAR
PROFESSOR,
DEPARTMENT OF DERMATOLOGY,
VENEREOLOGY AND LEPROSY,
B.L.D.E. (Deemed to be University)
SHRI B. M. PATIL MEDICAL COLLEGE,
HOSPITAL AND RESEARCH CENTRE,
VIJAYAPURA.

BLDE (DEEMED TO BE UNIVERSITY)

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

ENDORSEMENT BY THE HOD, PRINCIPAL/HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled " **A STUDY OF CUTANEOUS MANIFESTATIONS IN OVERWEIGHT AND OBESE PAEDIATRIC POPULATION** " is a bonafide research work done by Dr POOJA KOTIAN under the guidance of Dr ARUN C. INAMADAR, Professor, Department of Dermatology, Venereology and Leprosy, Shri B. M. Patil Medical College and Research Centre, Vijayapura.

Seal & Signature:

DR. KESHAVAMURTHY ADHYA

M.D, D.V.L

H.O.D & Professor

Department of Dermatology,

Venereology and Leprosy

B.L.D.E (Deemed to be University)

SBMPMC&H, Vijayapura

DATE:

Seal & Signature:

Dr. ARAVIND PATIL

M.S General Surgery

PRINCIPAL

B.L.D.E (Deemed to be university)

Shri. B. M. Patil Medical College,

Hospital & Research Centre, Vijayapura.

BLDE (DEEMED TO BE UNIVERSITY)

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

COPYRIGHT DECLARATION BY THE CANDIDATE

I hereby declare that the BLDE University, Karnataka shall have the right to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic/research purposes.

DATE:

PLACE: VIJAYAPURA

Dr. POOJA KOTIAN

ACKNOWLEDGEMENT

I wish to express my profound gratitude to my guide, **Dr. Arun C. Inamadar, MD, DVD, FRCP**, Professor, Department of Dermatology, Venereology, and Leprosy, for his invaluable guidance, insightful suggestions, and unwavering support throughout my postgraduate studies and the preparation of this dissertation. His mentorship has been instrumental in my academic and professional development.

I extend my sincere thanks to our Principal and Professor, Dr. Aravind Patil, MS, for his generous support and for facilitating the utilization of hospital resources essential for the completion of my research.

I am deeply indebted to my esteemed teachers: **Dr. Keshavmurthy A. Adya,** Professor and HOD**; Dr. Ajit B. Janagond,** Associate Professor**; Dr. N. S. Deshmukh**, Senior Resident**; Dr. Sanmitra Aiholli**, Assistant Professor**; Dr. Shruti Kulkarni,** Associate Professor**; and Dr. Uma,** Senior Resident. Their expert guidance and encouragement have been invaluable during my study.

I also wish to express my heartfelt gratitude to my family**: My Grandparents**, **Late Shri Damodar R. Suvarna** and **Late Varija Suvarna, Late Dasu Kotian**

and **Late Varija Kotian**; my parents**, Dr. Prem Kotian** and **Roopa Kotian**; my siblings**, Dr. Abhinandan Kotian, Greeshma Bangera**, and **Ashvith Bangera**; my uncles, **Uday Suvarna, Jagdeep Suvarna,** and **Dinu Kotian**; and my aunts, **Swarna Kumari, Kalpana Sairam, and Shilpa Suvarna.** Their unwavering support and encouragement have been a constant source of my strength.

I am grateful to my fellow postgraduate colleagues**: Dr. Mayuri Motgi, Dr. Namratha Shivaraj, Dr. Salman Hyder, and Dr. Trupthi A. L**. Their camaraderie and support have been invaluable.

I also wish to acknowledge my juniors: **Dr. Tvisha, Dr. Vinay, Dr. Devrat, Dr. Anaswara, Dr. Vaishnavi, Dr. Parvati, Dr. Anuhya, Dr. Monisha, Dr. Karthik, and Dr. Sanjana,** for their cooperation and assistance throughout my course.

My thanks extend to **Mrs. Shamshad G, Mr. Hiremath, Mrs. Yelawwa, the library staff,** and all the staff in the departments of pediatrics for their cooperation during my study.

I am particularly grateful to **Dr. Himani Kotian**, statistician, Department of Community Medicine, for her patient assistance with the statistical analysis of my data.

I would like to acknowledge the patients who participated in this study; their cooperation was vital for the successful completion of this research.

Finally, I express my gratitude to the Almighty for His blessings and guidance.

DATE:

PLACE: VIJAYAPURA Dr. POOJA KOTIAN

LIST OF ABBREVIATIONS

HOMA-IR : Homeostatic Model Assessment for Insulin Resistance

AN : Acanthosis Nigricans

MS : Metabolic syndrome

T2DM : Type 2 Diabetes mellitus

CVD : Cardiovascular disease

ABSTRACT

- **Introduction :**

Overweight and obesity are defined as excessive fat accumulation in the body. There has been a significant rise in recent years. The cutaneous manifestations of obesity emerge early in childhood and impose a risk of developing metabolic diseases in the future.

- **Aims and objectives :**

To assess the prevalence of different types of skin manifestations and its correlation with HOMA-IR in overweight and obese paediatric population.

- **Materials and Methods :**

This is a prospective study of seventy-eight pediatric individuals aged 5 to 18 years. Demographic details, clinical examination, photographs and BMI were documented. HOMA-IR was estimated using serum fasting Insulin and glucose levels and it was correlated with the cutaneous findings.

- **Results :**

Among 78 children, there were 49 males and 29 females with a mean age of 13.55 ± 2.87 years. The most common cutaneous finding was acanthosis nigricans (87.2%). The second most common finding was cutaneous infections, affecting 78.3% of the cases. Within these infections, 37.2% had dermatophyte infections, 7% had bacterial infections, 3.8% had viral infections, and 2.6% had parasitic infestations. Other observed cutaneous findings included striae (73.1%), acrochordons (39.7%), acne (32.1%), xerosis (21.8%), keratosis pilaris (17.9%), gynaecomastia (6.4%), and hirsutism (1.3%). These cutaneous manifestations were more prevalent in individuals aged 10-15 years with a BMI >95th percentile and HOMA-IR >2.6 suggesting a correlation between cutaneous findings and obesity.

- **Conclusion :**

Overweight and Obesity is a major health problem and continues to rise in both adult and pediatric population. It is implicated in a wide range of Cutaneous lesions. Early identification along with lifestyle modifications can help to avoid future risk and complications of metabolic diseases.

Keywords: Obesity, HOMA-IR, BMI

LIST OF TABLES

LIST OF FIGURES

TABLE OF CONTENTS

INTRODUCTION

INTRODUCTION

Overweight and obesity are a serious nutritional problem in today's modern world. It is characterized by excessive/abnormal fat accumulation within the body. The terms overweight and obesity were described as early in the medieval period, which symbolized richness, power, and fertility. However, Hippocrates described obesity as an illness in the Antique[1]. Obesity has become a global concern in both developed and developing nations, particularly among the paediatric population[2]. Obesity rates have risen rapidly since the Industrial Revolution, especially over the last three decades, as a result of rapid socioeconomic development in many countries. Obesity has emerged as a worldwide epidemic and is exponentially increasing in developing countries. In India, a significant proportion of the paediatric population are overweight or obese, and the explanation for this current situation may be attributed to changing lifestyle preferences and cultural environments, as well as a dominating hereditary propensity. Obesity not only hampers physiologically, but it also has a psychological impact owing to social bullying, rejection, and teasing, resulting in low self-esteem.

Early detection of obesity is crucial for a variety of reasons. Firstly, if obesity is not controlled at a young age, it can persist during adulthood. Second, being overweight or obese at a young age increases the risk of developing chronic

diseases such as hypertension and type 2 diabetes. Third, obesity is linked to low academic achievement because of the societal judgment associated with it.

Hence, early identification and education regarding paediatric obesity play an important role in preventing the progression to adult obesity and the long-term systemic consequences associated with it.

OBJECTIVE OF THE STUDY

❖ OBJECTIVE OF THE STUDY :

- To assess the prevalence of different types of skin manifestations and its correlation with HOMA-IR in overweight and obese paediatric population.

ANNEXURE – II

6.2 REVIEW OF LITERATURE

Obesity is a multifactorial condition characterized by the excessive accumulation of fat in the body, which can profoundly affect an individual's health and well-being.Obesity in the paediatric age group has become a significant global health issue, with prevalence rates increasing dramatically over the past few decades. As reported in various studies, the prevalence of childhood obesity, is as high as 40%, indicating a significant increase of up to 24 times in the past 2-3 decades . With a global prevalence of 18%, it is estimated that about 200 million schoolchildren worldwide are overweight or obese [3,4].

The prevalence of childhood obesity in India has significantly risen, with approximately 12.5 million children aged 5 to 19 being overweight in 2022, compared to only 0.4 million in 1990[5]. This trend is concerning, as obesity in childhood is known to progress into adulthood.

Obesity is measured using body mass index (BMI), a mathematical formula of weight-for-height index. BMI is calculated by dividing a person's weight in

kilograms by the square of their height in meters (kg/m^2). BMI is valued for its simplicity, low cost, and ease of use, making it suitable for routine screenings and assessments. One of the key advantages of BMI is its ability to track changes in weight status over time. This is particularly important for children and adolescents, as it allows healthcare providers to monitor their growth patterns and identify potential issues early on.

Traditionally, the BMI cut-offs used by Centre for Disease control (CDC) and WHO for overweight and obesity in children 5-18 years are ≥85th percentile (≥ +1SD) and ≥95th percentile (≥+2 SD), respectively[6,7,8].. These cut-offs coincide with the adult cut-offs for overweight and obesity of BMI 25 kg/m2 and 30 kg/m2, respectively.

The IAP charts for BMI devised in 2015 follow the WHO and IOTF suggested cutoffs, where the 23 adult equivalent is used to define overweight and 27 adult equivalent is used to define obesity[9].

Category	BMI percentile range
Underweight	Less than 5th percentile
Healthy weight	5th to 84th percentile
Overweight	85th to 95th percentile
Obese	Above 95th percentile

Table1: BMI Percentile ranges.

❖ Factors influencing obesity:

There are various factors that influence obesity. These include an interplay of genetic, environmental, behavioural, and socioeconomic factors

1.Genetic Factors play an important role in determining an individual's susceptibility to obesity. Their influences can affect metabolism, fat storage, appetite regulation, and body fat distribution[10]. Understanding the genetic basis of obesity can provide insights into personalized approaches to prevention and treatment.

2. Environmental factors, including the physical and social environments in which people live and work play a crucial role in the development of obesity. Access to healthy food options and opportunities for physical activity can significantly impact weight status. Conversely, environments that promote sedentary behaviour and the consumption of high-calorie, low-nutrient foods can contribute to obesity. Addressing environmental factors through policy and environmental changes can help create healthier communities[11].

3.Behavioral factors, such as eating habits and physical activity levels, are key determinants of weight status. Poor dietary choices, including the consumption of high-calorie, low-nutrient foods and sugary drinks, can contribute to weight gain. Sedentary behaviours, such as excessive screen time and lack of physical activity, can also contribute to obesity. Interventions targeting behavioural factors, such as nutrition education and promotion of physical activity, are essential for obesity prevention and management.

❖ Pathology:

The effects of obesity are primarily attributed to two key factors: the expansion of adipose tissue mass and the altered secretion of bioactive peptides from enlarged fat cells. [912] These factors are pivotal in understanding the physiological and metabolic changes that accompany obesity.

Obesity is characterized by adipose tissue expansion, which induces significant changes in adipocyte morphology and function, leading to increased infiltration of immune cells, particularly pro-inflammatory macrophages. This adipose tissue remodeling is accompanied by the secretion of various pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-alpha), interleukin-6 (IL-

6), and interleukin-1beta (IL-1beta), resulting in a state of chronic, low-grade inflammation within the adipose tissue microenvironment[13].

Furthermore, the inflammatory mediators produced by the adipose tissue can enter the systemic circulation, leading to systemic inflammation. This systemic inflammatory state is associated with insulin resistance, dyslipidemia, and endothelial dysfunction, all of which are key components of the metabolic syndrome and significantly increase the risk of cardiovascular disease in obese individuals.

The relationship between obesity and inflammation forms a feed-forward loop, whereby obesity-induced inflammation exacerbates metabolic dysfunction, further promoting inflammation[13]. Breaking this cycle is essential for effectively managing the metabolic complications associated with obesity and reducing the risk of obesity-related cardiovascular disease and type 2 diabetes mellitus.

❖ Effects of obesity and insulin resistance :

Chronic inflammation in obesity disrupts insulin signalling pathways in insulin-sensitive tissues, such as muscle, liver, and adipose tissue. This disruption leads to impaired glucose uptake by cells and increased hepatic glucose production, contributing to the development of insulin resistance[14]. Insulin resistance is a central feature of type 2 diabetes mellitus and is influenced by the inflammatory milieu associated with obesity.

Insulin resistance (IR), commonly associated with obesity, has significant effects on the skin. One key consequence is hyperinsulinemia, where elevated insulin levels directly influence skin physiology and result in various dermatological conditions. For instance, insulin promotes the proliferation of keratinocytes, the predominant cells in the epidermis, contributing to hyperkeratosis and forming microcomedones, which are the initial lesions in acne development. Additionally, insulin can stimulate sebocytes and sebum production, which is a major factor in the pathogenesis of acne.

Furthermore, IR can lead to elevated levels of insulin-like growth factor-1 (IGF-1). IGF-1 is known to play a role in the pathogenesis of acne and hirsutism [15].

It can increase the proliferation of keratinocytes, stimulate sebum production, and promote the production of androgens, which contribute to the development of acne and hirsutism. In pediatric populations, IR can impair wound healing by reducing collagen synthesis, leading to prolonged healing times and increased susceptibility to infections.

Insulin resistance (IR) and its associated cutaneous manifestations are significant indicators of underlying metabolic disturbances, serving as crucial markers for assessing the risk of metabolic syndrome (MS), type 2 diabetes mellitus (T2DM), and cardiovascular disease (CVD). Traditionally linked to adults and the elderly, these conditions are increasingly recognized in children and adolescents due to rising rates of childhood obesity worldwide.

Dermatologists play a pivotal role in identifying and interpreting early signs of IR through cutaneous manifestations and correlating them with assessments such as the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) in obese children and adolescents. Detecting these signs of obesity in childhood is essential for preventing or delaying the onset of serious metabolic complications later in life.

Insulin resistance (IR) is a pathological state where tissues exhibit a diminished response to insulin, affecting insulin-induced cellular activities[16]. This condition is a critical factor in developing various metabolic disorders, including obesity, diabetes mellitus, arterial hypertension, and nonalcoholic fatty liver disease (NAFLD)[17].

There are multiple methods to assess insulin resistance, with the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) being commonly used due to its minimally invasive nature. HOMA-IR is determined using a mathematical formula involving fasting glucose and insulin serum levels. In adults, a HOMA-IR value above 2.5 is typically considered elevated. However, no universally accepted cut-off value exists for pediatric populations. Some studies use fixed values ranging from 1.8 to 3.16, while others rely on age- and sex-specific percentiles to identify elevated HOMA-IR levels[18,19,20].

❖ Normal physiology of the skin:

Normal skin physiology plays a crucial role in maintaining skin health and overall well-being. Serving as a protective barrier, the skin shields the body from external threats such as pathogens, UV radiation, and chemicals. It also regulates body temperature, prevents dehydration by minimizing water loss, and houses sensory receptors that allow for the perception of touch, pressure, temperature, and pain[21]. The skin's ability to perform these functions effectively is essential for overall health and vitality.

Obesity significantly impacts normal skin physiology by altering the cutaneous functions. These changes can lead to various cutaneous manifestations. Understanding how obesity affects skin physiology is crucial for developing effective strategies to manage skin health in obese individuals.

EFFECTS OF OBESITY ON SKIN

1. Skin Barrier Function:

- Obesity is associated with several changes in skin barrier integrity, such as xerosis (dry skin) and altered transepidermal water loss (TEWL)[22].
- Xerosis is particularly common in morbidly obese individuals and is related to changes in the hydration levels of the stratum corneum[23].
- Additionally, obesity can compromise the skin barrier, increasing susceptibility to infections.

2. Sebum Production:

- Excess weight can influence sebum production, potentially contributing to acne.
- Androgens, insulin, growth hormone, and insulin-like growth factors are frequently elevated in obese patients, affecting acne severity by further increasing sebum production[24].

3. Sweat Gland Function:

- Obesity can Cramer MN, Jay O. Explained variance in the thermoregulatory responses to exercise: the independent roles of biophysical and fitness/fatness-related factors. J Appl Physiol (1985). 2015;119(9):982-989lead to increased sweating due to larger skin folds and thicker subcutaneous fat layers, trapping heat and moisture.
- This creates a humid environment that promotes local inflammation, increasing the risk of infections[25].
- Obesity can also contribute to bromhidrosis, causing unpleasant body odour due to sweat gland dysfunction.

4. Lymphatics:

- Obesity can impact the lymphatic system, leading to conditions such as lymphedema.
- Excess adipose tissue can compress lymphatic vessels, impairing lymphatic flow and leading to fluid buildup in the tissues[26].

5. Collagen Structure:

- Changes in collagen structure due to obesity can affect skin elasticity. Excess adipose tissue can lead to the breakdown of collagen fibers, contributing to the development of stretch marks, especially in areas prone to stretching like the abdomen, thighs, and buttocks.

6. Wound Healing:

- Obesity is known to impair wound healing. Leptin has been identified as a growth factor that can stimulate the proliferation of various cell types.
- In vitro studies have suggested that leptin deficiency could lead to impaired wound healing in the skin[27].
- The presence of excess adipose tissue can create a chronic inflammatory state, delaying the normal wound healing process and increasing the risk of infection.

7. Subcutaneous Fat:

- Changes in subcutaneous fat distribution, which often occurs in obesity, can affect the appearance and health of the skin. Uneven distribution of fat can

lead to skin folds and creases, increasing the risk of intertrigo and other dermatological issues.

8. Microcirculation:

- Obesity affects microcirculation in the skin.
- The increased pressure from excess adipose tissue can compress blood vessels, reducing blood flow to the skin. This can impact nutrient and oxygen delivery to skin cells, affecting their health and function.

❖ Cutaneous manifestations associated with obesity:

Obesity is a complex and multifactorial condition that affects individuals of all ages, including children and adolescents. Apart from its well-known metabolic and cardiovascular consequences, obesity also exerts significant effects on the skin, leading to various cutaneous manifestations. In pediatric populations, obesity-related skin alterations are increasingly recognized and can have substantial impacts on the overall health and well-being of affected individuals.

Understanding these cutaneous manifestations is crucial for healthcare providers to recognize and manage obesity-related skin conditions effectively. In this context, this discussion focuses on the dermatological implications of pediatric obesity, exploring the key cutaneous manifestations associated with insulin resistance, increased androgen levels, infections, and inflammatory conditions. Understanding these dermatological consequences is vital for healthcare professionals to improve their diagnostic, therapeutic, and preventive approaches for obesity-related skin issues in pediatric patients.

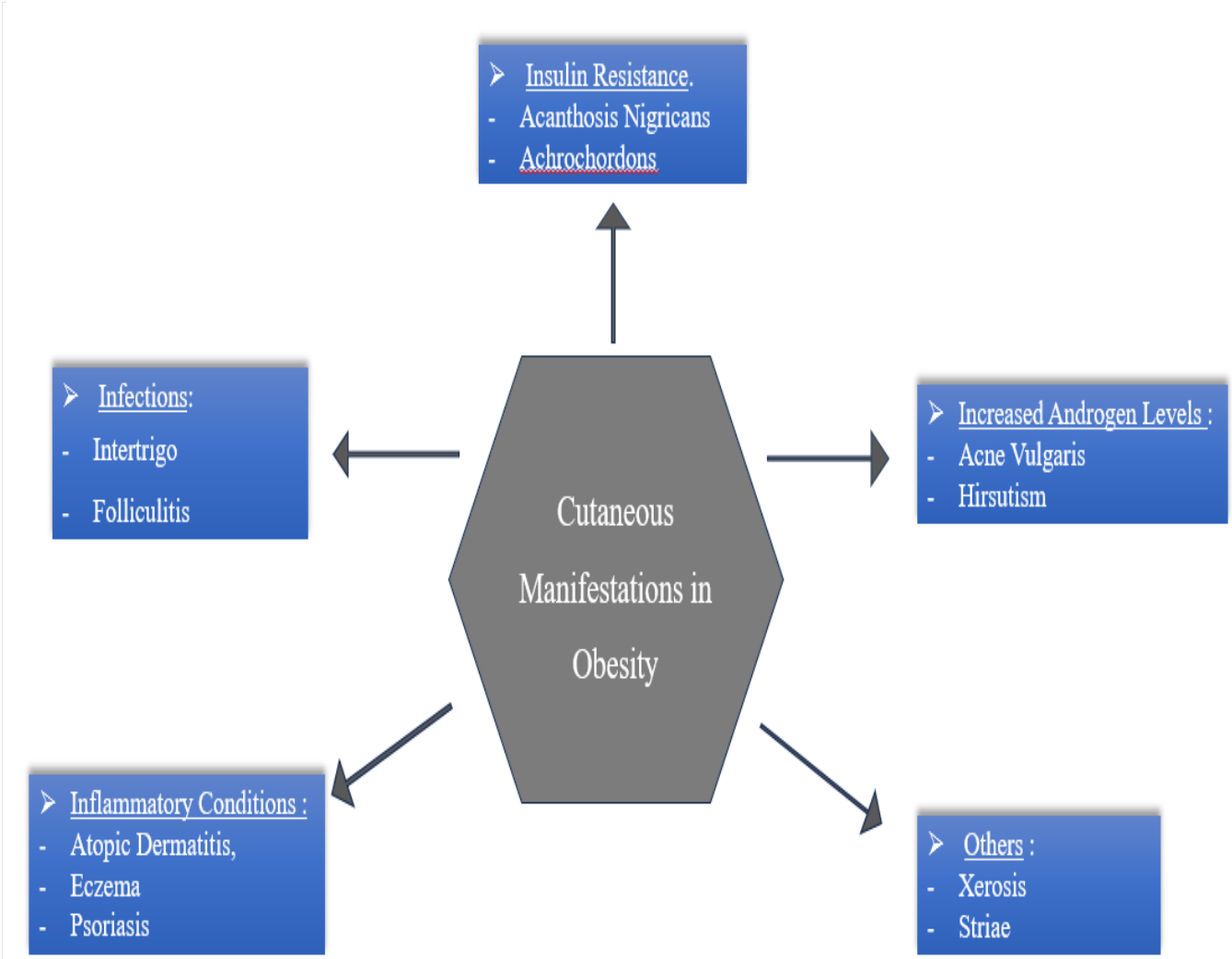


Figure 1 : Categorization of cutaneous manifestations of obesity.

1) Insulin Resistance :

➢ Acanthosis nigricans

AN is the most common and earliest cutaneous manifestation observed in obesity. It is the most common dermatologic manifestation of pediatric obesity, occurring in 66% of overweight adolescents and in 56% to 92% of children and adolescents with DM2[28,29].

It manifests as symmetrical, velvety, light brown to black plaques with poorly defined edges, which tend to accentuate the skin's natural markings. It commonly occurs in skin folds, particularly in the armpits, the back of the neck, the sides of the neck, the groin, and the genital area. Less frequently, they may appear on the face, inner thighs, elbows, knees, around the belly button, eyelids, knuckles, palms, soles, nipples, and areolas.

In its initial stages, the lesions may exhibit redness and itching, which gradually progresses to form plaques which have rough and wart-like in texture. Despite being localized to specific areas, the affected skin blends seamlessly into the surrounding skin, lacking distinct boundaries. Acanthosis nigricans can occasionally affect the lips and mucous membranes of the mouth and upper respiratory tract, though such cases are uncommon. In severe instances,

individuals may experience diffuse hair loss and nail dystrophy. The presence of acanthosis nigricans can also indicate an underlying systemic involvement.

Acanthosis nigricans (AN) typically appears during puberty and is most frequently linked to obesity and skin tags in individuals aged 12 to 30 years[30]. AN is an important clinical indicator of both obesity and insulin resistance. Obesity greatly contributes to secondary insulin resistance, which results in compensatory hyperinsulinemia. This hyperinsulinemia leads to increased binding of insulin to IGF-1 receptors, stimulating the proliferation of keratinocytes and fibroblasts[31]. This process is a key factor in the development acanthosis nigricans in obesity. The severity of AN and the occurrence of skin tags in young obese individuals are strongly correlated with the level of excess weight and the HOMA-IR levels.

➢ Acrochordon:

Acrochordons, commonly known as skin tags, are soft, coffee-colored, pedunculated papules typically found in areas such as the neck, armpits, and groin. They often appear alongside acanthosis nigricans and are closely associated with obesity, metabolic syndrome, hormonal imbalances, and

excessive skin rubbing. These growths form in skin folds and are generally small, though some can exceed one centimeter in size. Patients with more than ten acrochordons have been observed to have elevated leptin levels, suggesting a link to metabolic disturbances[32]. Skin-on-skin friction is believed to contribute to the development of acrochordons.

2) Increase in Androgen levels :

➢ **Acne Vulgaris :**

Acne is a chronic inflammatory skin disease. It is due to abnormal functioning of sebaceous and sweat glands with a large number of bacteria colonizing in the pilosebaceous unit. On examination, comedones, papules, pustules, and in severe conditions, even nodules and cysts appear over the face, neck, shoulders, upper back, and chest.Acne at prepubertal ages may be the first physical sign of pubertal maturation. However, mid-childhood acne in 1–7-year-olds is uncommon and can be associated with overweight or obesity.

In a multicenter study, obesity was found to be significantly associated with acne in children, with 65% of the study participants exhibiting acne[33].

➢ Hirsutism

Hirsutism, or excessive hair growth, is frequently linked with obesity due to hormonal imbalances, specifically elevated androgens and insulin resistance. Obesity is marked by chronic low-grade inflammation, which can disturb hormonal equilibrium and promote hirsutism. Increased insulin levels commonly seen in obesity can further boost androgen production, worsening hirsutism symptoms[34].

➢ Hidradenitis Suppurativa

Obesity increases the risk and severity of Hidradenitis suppurativa (HS) by promoting inflammation and creating conditions that worsen it. Obesity creates conditions that promote inflammation, such as increased mechanical stress and friction in skin folds, which can trigger or exacerbate HS. Additionally, obesity is linked to systemic inflammation, which can further aggravate the local inflammation in HS lesions. The hormonal and metabolic changes associated with obesity also play a role in the development and persistence of HS.

3) Cutaneous Infections :

The increased susceptibility to skin infections in obese individuals arises from a combination of factors, including a proinflammatory state, compromised cell-mediated immune responses, and anatomical factors[35]. Obesity is associated with larger skin folds, increased friction, and higher levels of sweating, creating a humid environment conducive to inflammation and skin conditions such as intertrigo. Intertrigo, characterized by pustules on a reddened base, is particularly common in obese children and can be exacerbated by heat, friction, and moisture.

Obese individuals also have a heightened risk of Candida Albicans infections due to the warm, moist skin folds providing an ideal environment for fungal growth[36]. Furthermore, folliculitis, an inflammation of hair follicles often caused by Staphylococcus aureus, is more prevalent in obese individuals, possibly due to impaired skin barrier function and the humid environment[37].

Management of these infections typically involves topical antibiotics or antifungals, along with measures to reduce friction and moisture. It is crucial for obese individuals to maintain good hygiene and manage their weight to prevent these complications.

Other infections, such as pachyonychia, furunculosis, anthrax caused by staphylococci, and erythrasma caused by Corynebacterium minutisimi, are also more common in obese patients, along with a higher tendency to develop cellulitis in the extremities and vascular ulcers due to factors like lymphedema and venous insufficiency[38].

4) Inflammatory conditions:

Obesity increases the risk of developing inflammatory skin conditions such as atopic dermatitis, eczema, and psoriasis. Although atopic dermatitis is a common childhood inflammatory skin condition, it is associated with epidermal barrier alteration and alteration with immunity[39]. Obesity exacerbates the inflammatory state in early childhood by inducing inflammation through cytokines produced from adipose tissue.

Adipocytes secrete various adipokines, including leptin, adiponectin, plasminogen activator inhibitor-1, interleukin-6, and tissue necrosis factor-alpha, which contribute to the inflammatory state of obesity [40]. Obese children are more likely to have atopic dermatitis compared to normal-weight children.

Chronic sleep disruption, common in children with atopic dermatitis, is associated with poor school performance, low self-esteem, and familial stress, which are also linked to obesity, further exacerbating symptoms. Additionally, children with atopic dermatitis have a higher risk of infections, as their immune dysfunction and barrier disruption make the skin more susceptible to bacterial infiltration, especially Staphylococcus aureus[41].

The link between obesity,eczema and psoriasis, with a higher prevalence of obesity seen in women and obese children. Research has since shown that these conditions share common inflammatory pathways and excess cytokines. Obesity, characterized by chronic low-grade inflammation, is associated with elevated levels of proinflammatory cytokines like TNF-α, IL-6, and acute phase proteins such as C-reactive protein, which may explain its association with psoriasis and eczema [42]. Identifying obesity early and promoting healthy lifestyle habits are crucial for treatment and improving outcomes.

Preventive measures for obesity should start during maternal pre-pregnancy or pregnancy, as excessive weight gain during pregnancy and being overweight before pregnancy are related to a higher risk of developing inflammatory skin conditions in children[43,44].

➢ Other common skin manifestations associated with obesity :

▪ Plantar Hyperkeratosis

Childhood obesity can affect the foot's shape, potentially resulting in discomfort or alterations due to increased pressure on the small bones in the front of the foot[45]. Obese children before puberty also exhibited physical changes in foot structure. These pressures on the foot can lead to plantar hyperkeratosis, a thickening of the skin on the soles, which is considered a sign of severe obesity.

▪ Xerosis

Xerosis, or dry skin, is one of the earliest and one of the common findings of obesity. It is associated with increased water permeability of the skin, leading to drier skin[46].

A study by Tollefson et al. (2012) investigated the prevalence of xerosis in obese children and found that it was significantly higher compared to non-obese children. The study also noted that xerosis was more prevalent in children with higher body mass index (BMI) percentiles. This suggests a correlation between obesity and xerosis in pediatric populations[47].

Monteiro Rodrigues et al. observed that gaining excess weight leads to adaptive physiological changes in the skin. In the study it was found that individuals who were morbidly obese had higher levels of transepidermal water loss (TEWL) compared to those who were overweight or had obesity class I and II. This higher TEWL also resulted in lower levels of epidermal hydration[48].

- **Striae :**

The presence of striae directly correlates with obesity. It is a linear atrophic patch to plaque formed due to excessive stretching of the skin.It occurs due to dermal damage induced by stretching and breakdown of the connective tissue. It is distributed perpendicular to the force of greatest tension. Striae are commonly found on the breasts, buttocks, abdomen, and thighs. They are commonly seen in adolescents, growth spurts, pregnancy, weight lifters, and feature of metabolic. These marks are initially erythematous, but they later turn violet and become white depressed patches. These appear to be a type of dermal scarring with aberrant healing response and replacement of collagen[49].

❖ Obesity Prevention

Childhood obesity is a pressing concern with long-lasting effects on health. It raises the risk of developing serious metabolic conditions like type 2 diabetes and cardiovascular disease. Socially and emotionally, obese children may face challenges like low self-esteem and depression, which can persist into adulthood. To combat this, prevention efforts should begin early, ideally before birth. Maternal gestational diabetes exposure and central obesity are linked to increased childhood adiposity and cardiometabolic issues. Babies with higher birth weights are more likely to be obese later on. Monitoring BMI in young children with maternal overweight or high birth weight is vital for preventing future obesity. Unhealthy behaviors like longer screen time, eating junk foods increases obesity risk, especially in children of overweight mothers. Indigenous schoolchildren have lower obesity rates, suggesting healthier lifestyles play a role. Encouraging healthy habits, such as life style modifications,regular physical activity and balanced diets rich in fiber and vegetable protein, can help reduce obesity rates.

.

METHODOLOGY

METHODOLOGY

SOURCE OF DATA :

- All the patients, from 5 – 18 years of age, attending outpatient and inpatient department of Dermatology, Venereology and Leprosy, Pediatrics of B.L.D.E (Deemed to be University) Shri. B.M. Patil Medical College Hospital and Research Centre, Vijayapura, were enrolled for the study.

Period of study:

- The study was conducted during the period of August 2022 to Febrauary 2024.

Study design:

- A hospital-based prospective cross-sectional study.

Sample size:

With an anticipated Proportion of 73.8% , the study would require a sample size of 78 with a 95% level of confidence and 10% absolute precision**.**

Formula used

$$n=\frac{z^2\ p*q}{d^2}$$

Where Z= Z statistic at α level of significance

d^2= Absolute error

P= Proportion rate

q= 100-p

METHOD OF COLLECTION OF DATA:

Patients from 5 – 18 years of age were enrolled for the study.

Inclusion criteria:

- Overweight, obese children and adolescents of the age between 5 to 18 years.

Exclusion criteria:

- Children less than 5 years of age.
- Non- Obese children.

METHOD OF STUDY:

- Paediatric population with overweight and obesity of age ranging from 5-18 years was included in the study.
- Cutaneous examination was looked for skin findings.
- BMI was calculated and plotted using BMI Growth Plots (WHO and IAP combined). Based on the values obtained from the BMI growth chart, the child will be categorized as overweight or obese.
- Investigations such as serum fasting insulin and fasting glucose were done.
- HOMA-IR was estimated based on the fasting insulin and glucose values, and assessed to look for correlation with BMI.

METHODOLOGY :

Overweight and obese children of age between 5-18 years will be examined to look for Cutaneous manifestations. Serum fasting glucose and Insulin levels will be done and insulin resistance (IR) is assessed using homeostasis model assessment (HOMA). HOMA-(IR) will be calculated using the formula HOMA-IR = [Serum Fasting Glucose x Serum fasting insulin] /22.5. The values obtained will be correlated with the cutaneous findings.

STATISTICAL ANALYSIS:

- The data obtained will be entered in Microsoft Excel Sheet, and statistical analysis will be performed using statistical package JMP SAS
- Result will be presented as Mean (Median) ± SD, counts and percentages and diagrams.

ETHICAL CLEARANCE:

- Institutional ethical committee clearance was undertaken for the study.

RESULTS

Results

Distribution of children according to age:

Age distribution among a total of 78 children showed 41 cases in the 10-15 years age group, 21 cases above 15years years age group, and 16 cases in the 5-10 years age group. (Table 1)

The mean age among the cases **13.55±2.87 years.** (Pvalue<0.001)

Table 2 : Distribution of Cases according to age.

Age	No of children	Percentage
5-10	16	20.5
10-15	41	52.6
>15	21	26.9
Total	78	100

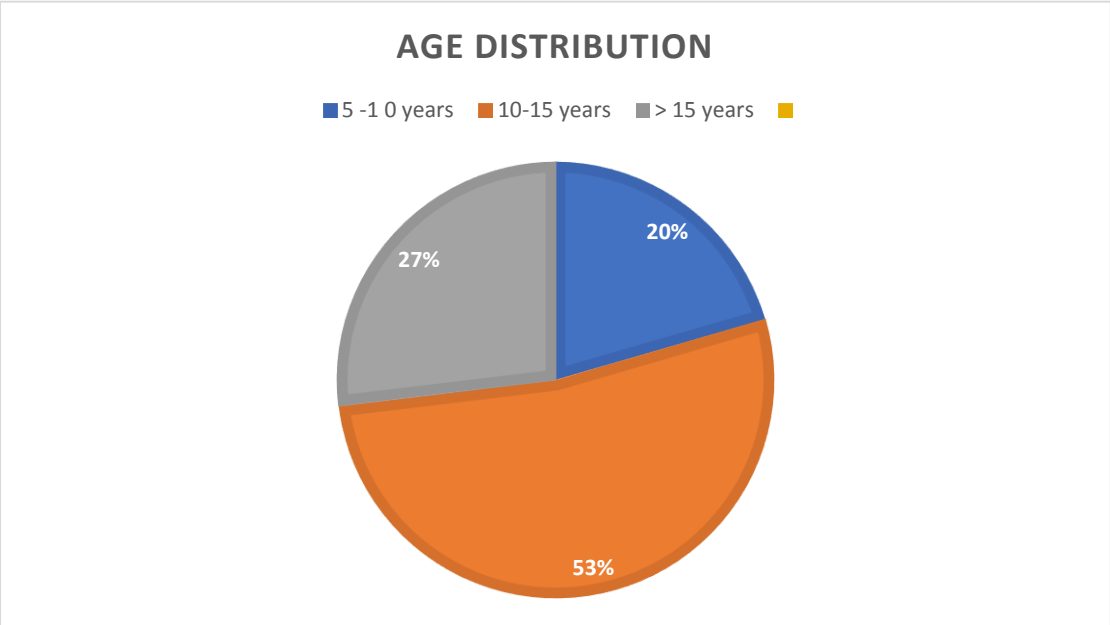


Figure 2 : Distribution of Cases according to age

Gender distribution :

Among 78 cases, 49 were males, while 29 were females.

Table 3 : Distribution of Cases according to gender

Gender	No of children	Percentage
Male	49	62.8
Female	29	37.2
Total	78	100.0

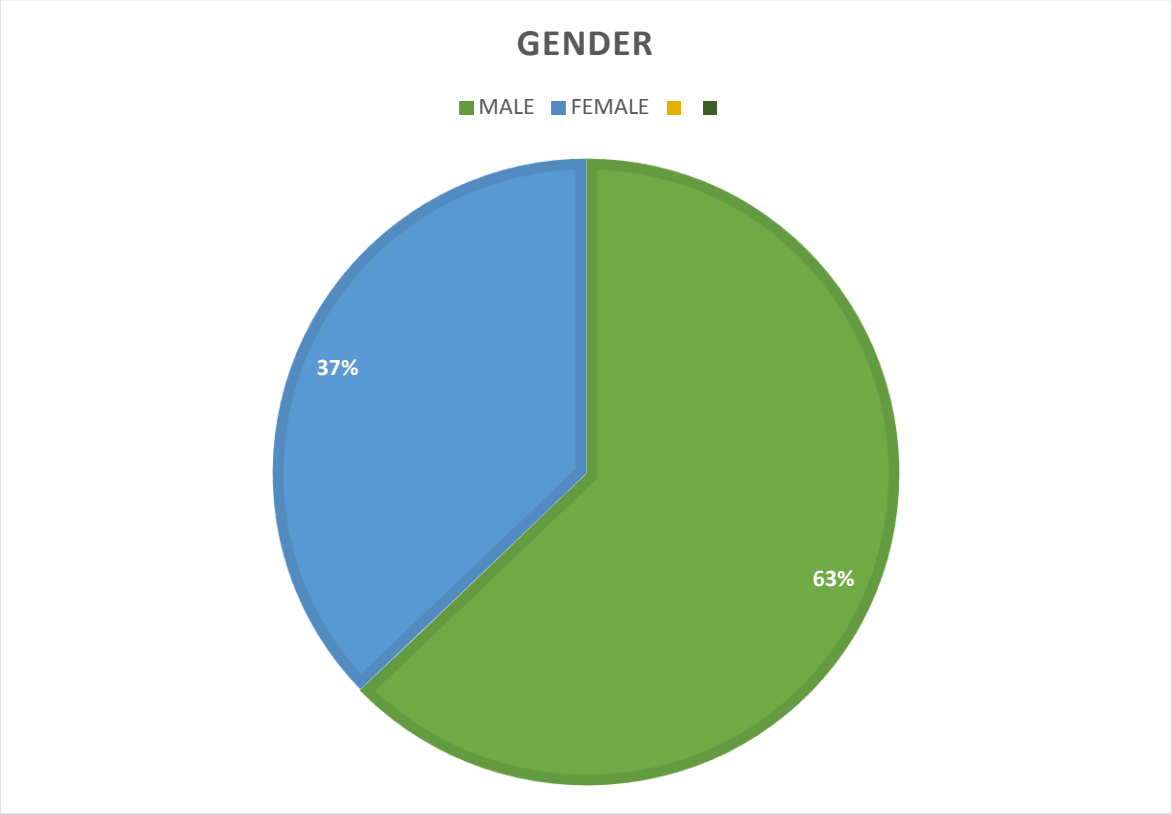


Figure 3 : Distribution of Cases according to Gender

Distribution of children according to BMI:

According to BMI values, the majority of individuals were obese, accounting for 65 cases, which is 85.5% of the total. There were also 13 cases categorized as overweight, making up 16.7% of the total cases. (Table 3)

Mean **: 28.69±3.82**; P-value <0.001

Table 4: Distribution of Cases according to BMI (In Centiles)

BMI (In Centiles)	No of children	Percentage
85-95 (Overweight)	13	16.7
>95 (Obese)	65	85.5
Total	78	100.0

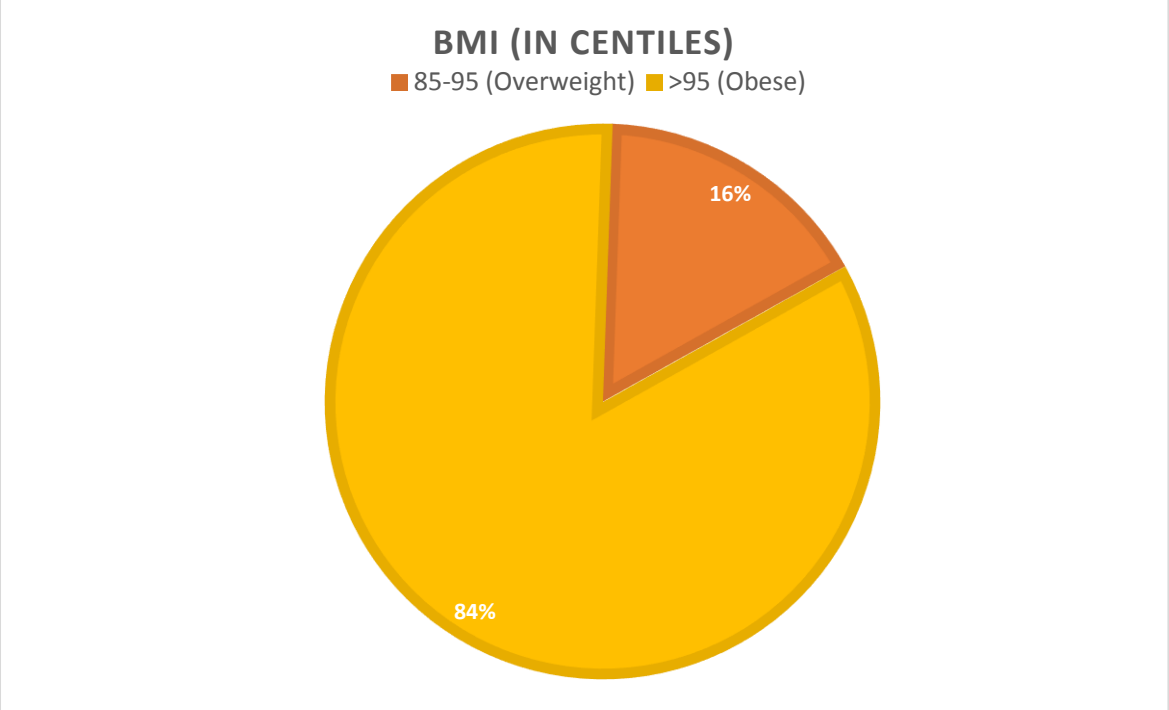


Figure 4: Distribution of Cases according to BMI (In Centiles)

Distribution of cutaneous manifestations among Children:

Among these, Acanthosis Nigricans was the most common cutaneous finding observed in 68 cases. Other most common skin manifestations include cutaneous infections seen in 63 cases, striae in 51 cases, acrochordons in 31 cases, acne in 25 cases, xerosis in 17 cases.

Table 5: Distribution of cutaneous manifestations among Children

	No of children	Percentage
Acanthosis nigricans	68	89.1
Striae	51	73.1
Hirsutism	1	1.3
Gynaecomastia	5	6.4
Acrochordons	31	39.7
Acne	25	32.1
Keratosis Pilaris	14	17.9
Xerosis	17	21.8
Skin Infections Dermatophytosis (41)	63	78.3

Bacterial Infections (9) Viral Infections (3) Parasitic (2) Intertrigo (8)		
Others	5	10

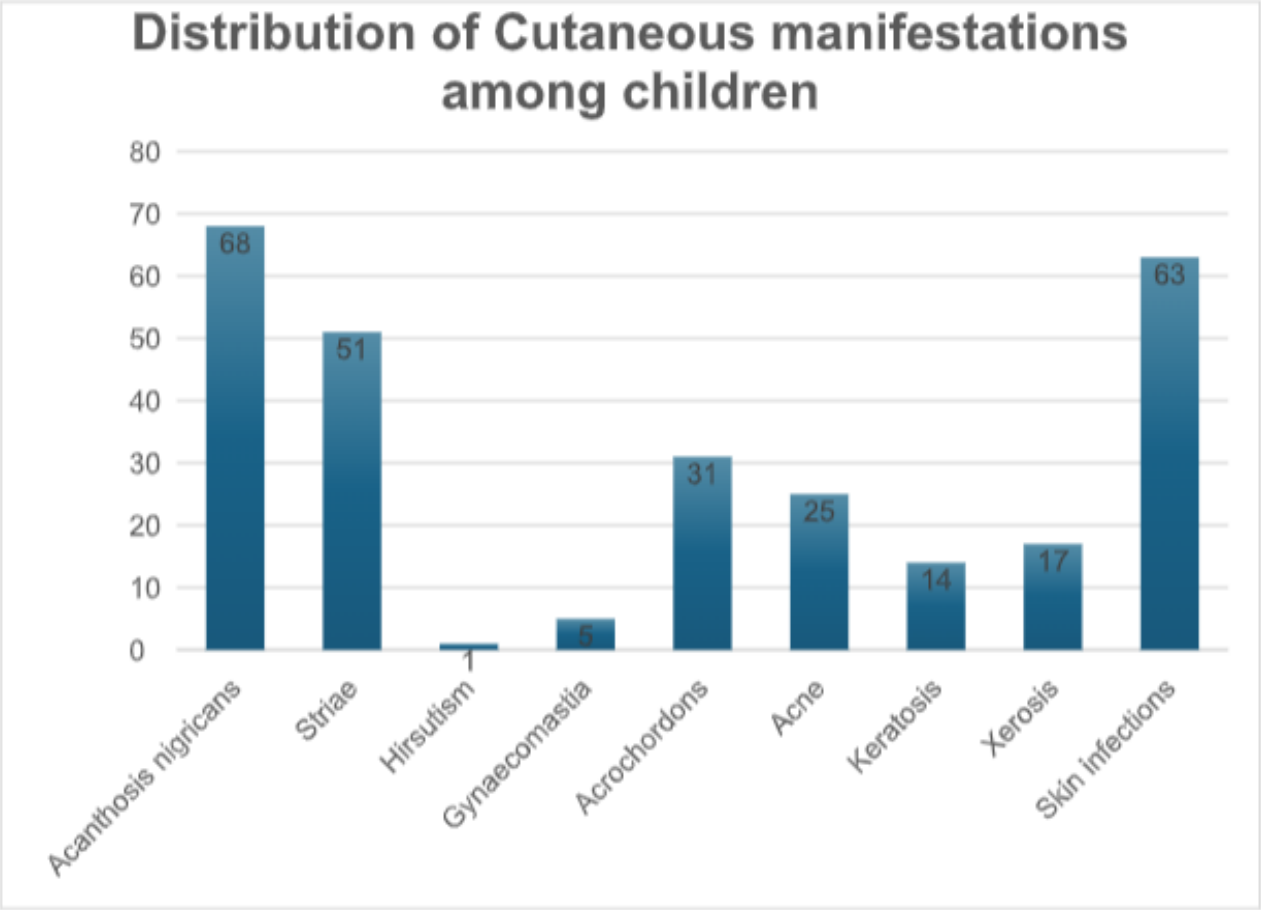


Figure 5 : Distribution of cutaneous manifestations among Children

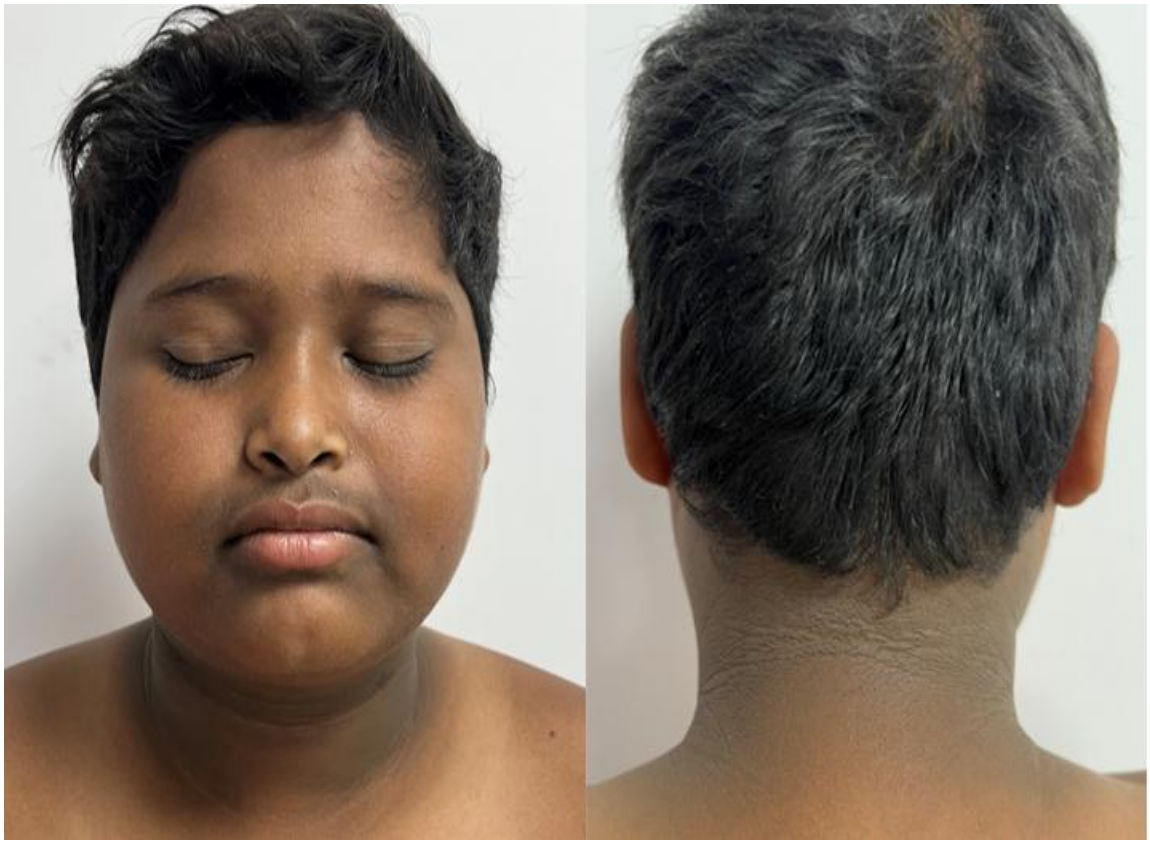


Figure 6 : Acanthosis Nigricans in an Obese child present over Face, nape of neck.

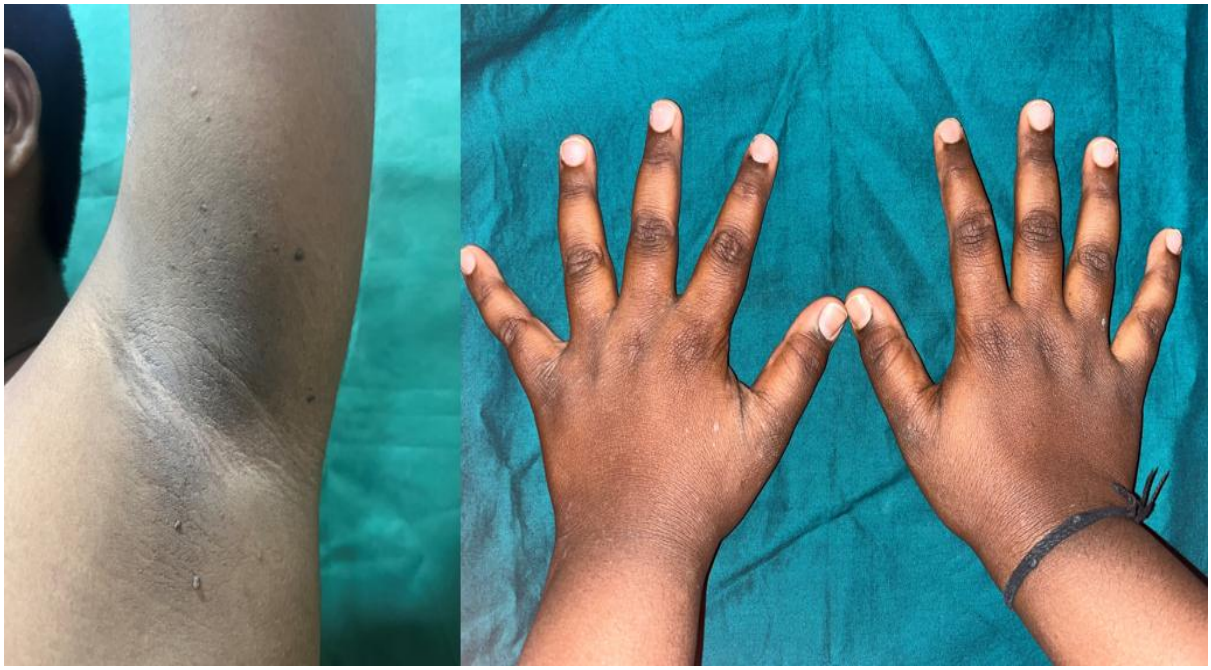


Figure 7 : Axilla showing : AN and Achrochordons. , AN over Knuckles.

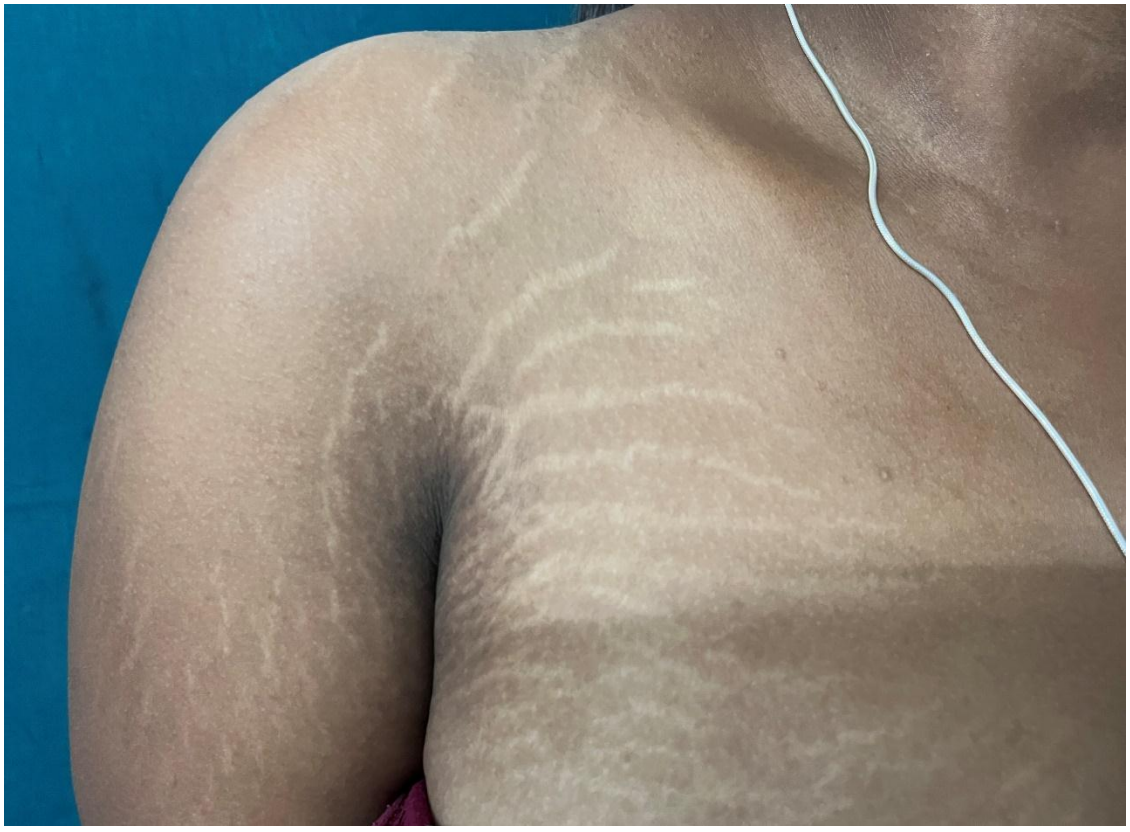


Figure 8 : Striae Distensiae – over arms, shoulders, chest.

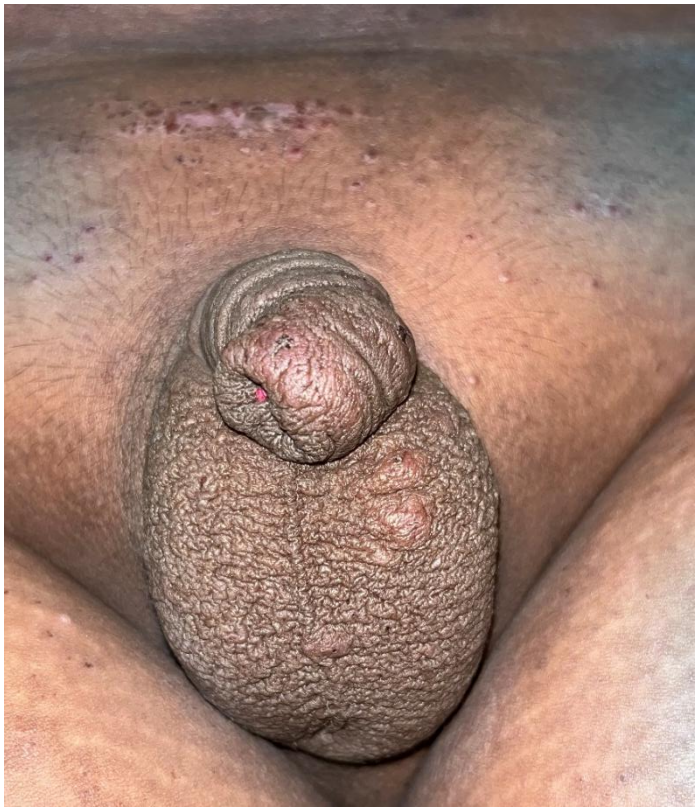


Figure 9: Nodular Scabioies – over Scrotum.

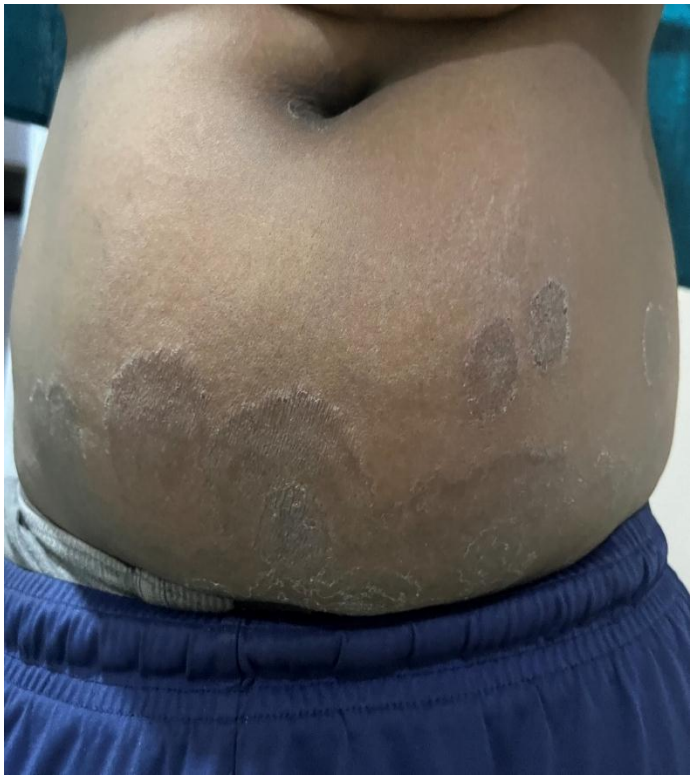


Figure 10 : Tinea Corporis – lower Abdomen

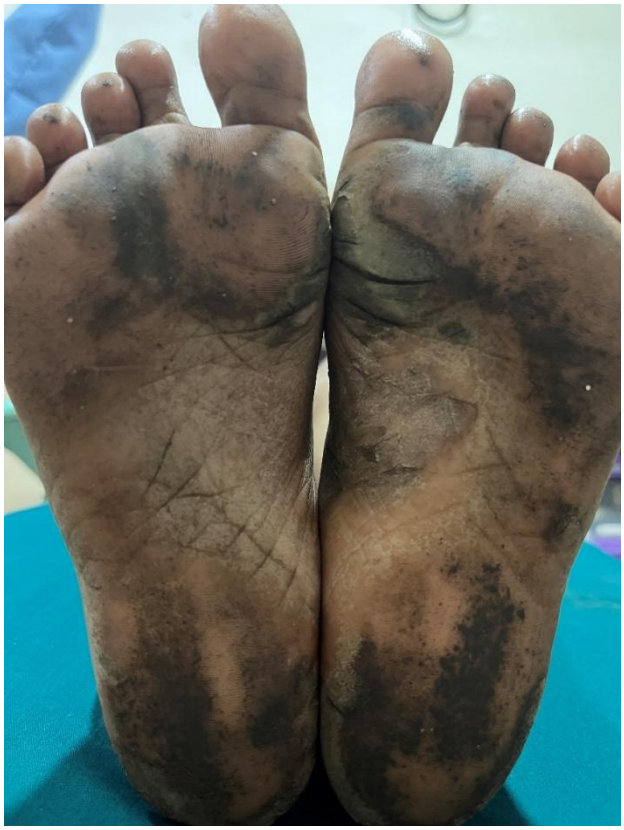


Figure 11: Plantar Eczema over foot,.

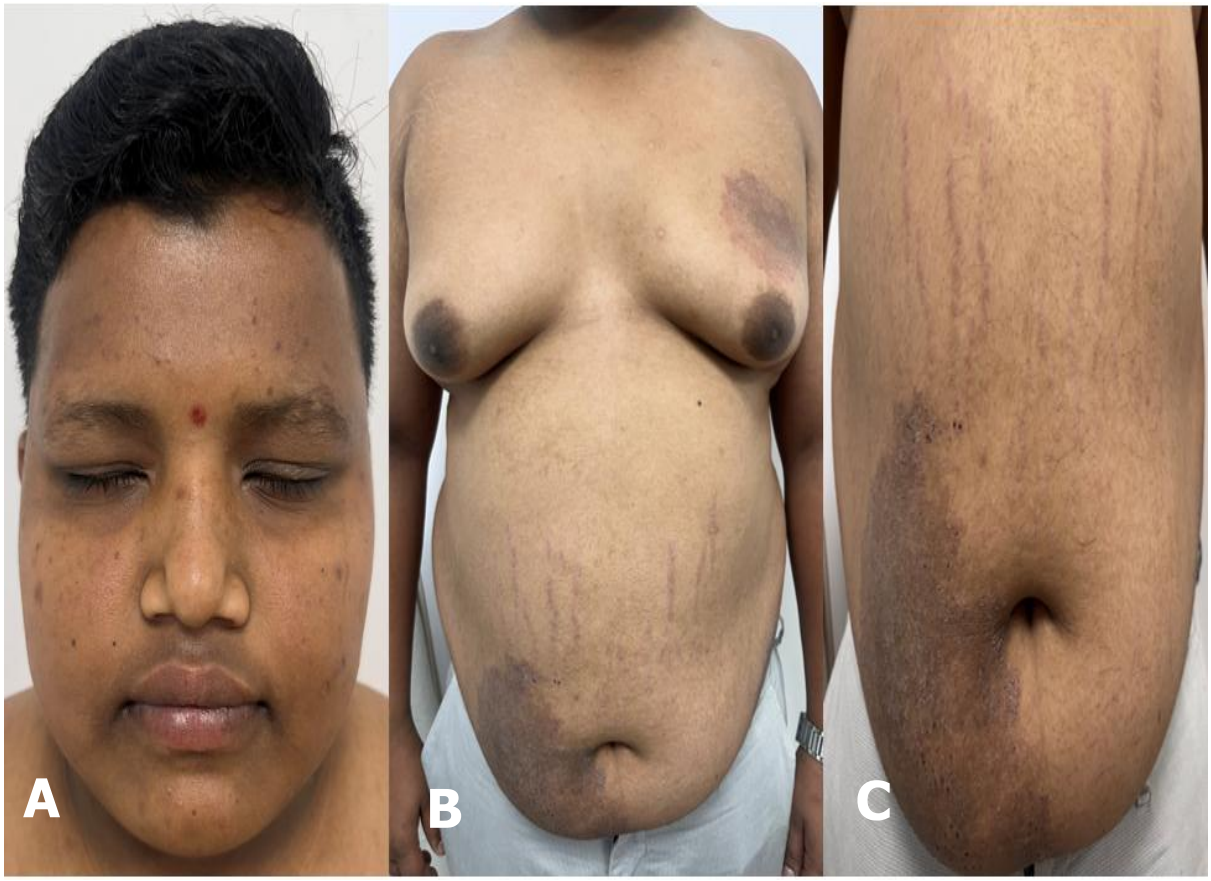


Figure 12 : A. Acne Vulgaris, B. Central obesity, Gyanecomastia C. Abdomen shoing – Striae and Tinea Lesions.

Distribution of patients according to HOMA IR :

HOMA-IR was estimated in all patients using fasting blood sugar and fasting insulin levels.

The mean HOMA-IR among cases was **4.61±2.06**

HOMA-IR >2.6 was observed among 46 accounting for 59% cases.

Table no 5 : Distribution of patients according to HOMA IR

HOMA-IR	No of children	Percentage
>2.6	**46**	**59**
< 2.6	32	41
Total	78	100.0

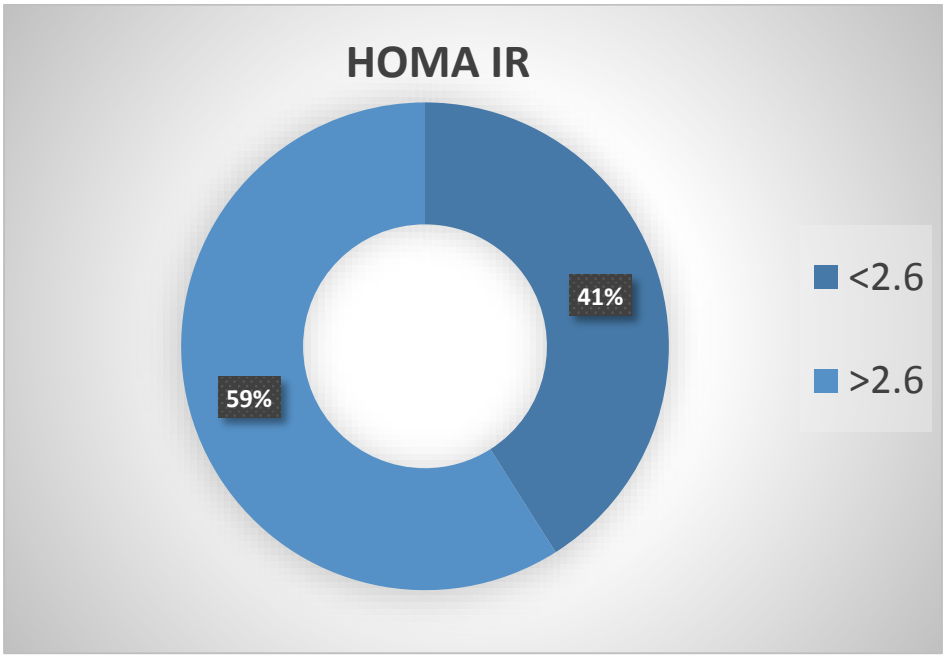


Figure 13 : Distribution of Cases according to HOMA I

Correlation of cutaneous manifestations and investigations among HOMA>2.6

Frequency Tables :

1. Acanthosis nigricans

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	N	4	8.7	8.7	8.7
	P	42	91.3	91.3	100.0
	Total	46	100.0	100.0	

2. STRIAE

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	N	12	26.1	26.1	26.1
	P	34	73.9	73.9	100.0
	Total	46	100.0	100.0	

3. ACHROCHORDONS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	N	25	54.3	54.3	54.3
	P	21	45.7	45.7	100.0
	Total	46	100.0	100.0	

4. FOLLICULITIS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	N	71	91.0	91.0	91.0
	P	7	9.0	9.0	100.0
	Total	78	100.0	100.0	

5. KERATOSIS PILARIS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	N	63	80.8	80.8	82.1
	P	14	17.9	17.9	100.0
	Total	78	100.0	100.0	

6. ACNE VULGARIS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	N	30	65.2	65.2	65.2
	P	16	34.8	34.8	100.0
	Total	46	100.0	100.0	

7. ATOPIC DERMATITIS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	N	71	91.0	91.0	92.3
	P	6	7.7	7.7	100.0
	Total	78	100.0	100.0	

8. GYNECOMASTIA

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	N	41	89.1	89.1	89.1
	P	5	10.9	10.9	100.0
	Total	46	100.0	100.0	

9. HIRSUTISM

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	N	45	97.8	97.8	97.8
	P	1	2.2	2.2	100.0
	Total	46	100.0	100.0	

DISCUSSION

DISCUSSION

Cutaneous manifestations in obesity are more common with earlier onset, longer duration, and greater severity of the condition, and are further increased when obesity is associated with diabetes or insulin resistance[50].

Among the 78 cases present in this study, the maximum number of cases was in the age group of **10-15years** (53%), followed by in 15-18 years (27%). The mean age among cases was **13.55±2.87 years (**Table 1). Most of the cases were males (49 males, 62.8 %) while 29 cases (37.2%) were females. (Table 2) The maximum numbers of cases among males were in the age group of 10-15 years (33 cases; 44%), while in females it was in age group 15-18 years (50.79%).

Out of 78 cases, the majority were classified as obese, with 65 cases (83.3%), followed by 13 cases (16.7%) classified as overweight (Table 3). Overweight cases were predominantly seen in individuals 10-15 years of age and obesity was observed in individuals above 10 years of age, accounting for 49.2% of the obese cases. Both overweight and obesity were more common in males, with prevalence rates of 46.2% for overweight and 66.2% for obesity, respectively.

The overall incidence of cutaneous manifestations among children revealed that the highest number of cases were in the 10-15 years age group.

In a study done by Gulanikar et al, on Cutaneous manifestations of Paediatric Obesity: Relation with insulin resistance showed that among 138 paediatric individuals, majority were males, had a higher BMI and belonged to the age group of 5-18 years. Similar findings were also observed in Mrinal et al prospective study in 100 obese children, in which 61 cases were males and 39 females. Mean age of the cases included was 11.3±1.3 years[50].

The most common cutaneous finding seen in our study was Acanthosis nigricans (87.2%). The individuals with acanthosis nigricans had more than one body site involved. Most patients had acanthosis nigricans over neck and axilla. However, a few patients had facial acanthosis, acanthosis nigricans over dorsum of hand (knuckles) and over cubital fossa area. Nithun et al in his study mentioned that most common site involved was neck (90%), which has been found to be affected in more than 90% of cases of AN in several studies[51].

Acanthosis nigricans (AN) is considered the earliest and most common cutaneous finding in obesity. It correlates with underlying systemic involvement, particularly diabetes and insulin resistance. Obesity, characterized by a hyperinsulinemia state, involves elevated insulin levels that act on insulin-like growth factor receptors on keratinocytes and fibroblasts, stimulating their

growth and proliferation. This excessive stimulation leads to the formation of AN.

The second most common cutaneous finding in this study was cutaneous infections constituting up to 78.3% of the total cases. Majority of which was seen among obese individuals. Amongst skin infections, 41 paediatric individuals (37.2%) were affected with dermatophyte infections, 9 patients (7%) had bacterial infections, 3 patients (3.8%) had viral infections, and 2 patients (2.6%) had parasitic infestations. Skin infections are more common in obese individuals primarily due to increased sweating, loose skin folds, and friction in body folds. These conditions predispose an individual to develop intertrigo and secondary infections.

Similar findings were also noted in a population-based study by Mirmani and Sunkwad A et al. where skin infections predominantly dermatophytosis, intertrigo and bacterial infections were seen more commonly among obese children.

Cutaneous findings such as striae (73.1%), acrochordons (39.7.7%) were noted in many individuals in this study. Striae were more commonly observed in obese cases, predominantly on the abdomen, shoulders, and thighs. Majority of patients with striae also had higher grades of acanthosis nigricans (grades 3 and 4) and acrochordons. Hsu et al. discovered that 40% of children with moderate

to severe obesity had striae, with a higher incidence in those who had been obese for a longer period[52].

Similarly, acne (32.1%), xerosis (21.8%), keratosis pilaris (17.9%), gynaecomastia (6.4%) and hirsutism (1.3%) and gynaecomastia were also seen in the study population (Table 4). Other less commonly associated cutaneous findings were phrynoderma, lichen planus, prurigo simplex, IBR, vitiligo, etc.

The overall association between BMI and cutaneous manifestations showed that cases with higher BMI (>95 percentile) showed more cutaneous manifestations compared to BMI (85-95 percentile) with statistically significant difference. ($P < 0.05$). 59% of the cases had HOMA-IR values greater than 2.6 and showed more cutaneous manifestations compared to those with HOMA-IR less than 2.6. The data indicates that individuals with higher BMI and elevated HOMA-IR values had significantly more cutaneous manifestations compared to those with lower BMI and HOMA-IR values.

In a study by Gomez et al. on skin disorders in overweight and obese patients and their relationship with insulin, a similar relationship was observed between skin diso, obesity, and insulin levels[53,54]. Similar findings were observed by Gulanikar AD et al. and study done by Boza et al., where dermatoses showed a statistically significant relationship with obesity[55].

Obesity is closely associated with various skin changes and serve as indicators for various underlying metabolic conditions like insulin resistance and diabetes in both children and adults. With the increasing prevalence of obesity, understanding these skin conditions is essential for paediatrician and dermatologists to identify these cutaneous findings for early diagnosis and prevention of future risk of developing metabolic complications.

CONCLUSION

❖ Conclusion :

Obesity is closely linked to a spectrum of dermatological alterations, which can serve as clinical markers of excessive adiposity. Effective dermatological care for obese patients is imperative due to the high prevalence of these manifestations, many of which are preventable and show good response to treatment, thereby enhancing patient quality of life. Insulin resistance (IR) is a prevalent condition among young obese individuals, conferring a heightened risk for metabolic syndrome (MS), type II diabetes mellitus (DM2), and cardiovascular disease. Enhancing insulin sensitivity presents a viable therapeutic strategy for addressing obesity and its dermatologic sequelae.

In children, obesity-related dermatological manifestations are increasingly recognized and can significantly impact overall health and psychosocial well-being. Early identification and management of these conditions are critical. Given the limited efficacy of lifestyle interventions and the paucity of approved pharmacotherapies for pediatric obesity, prevention remains paramount. Parental recognition

and intervention are essential in mitigating childhood obesity. In contrast to adults, children experience physiological insulin resistance during pubertal development, exacerbating their susceptibility to dermatologic conditions. Therefore, early prevention of obesity is crucial to mitigate the associated cutaneous complications.

SUMMARY

❖ Summary :

In this study of 78 cases, most were males, aged 10-15 years, with the majority being classified as obese. The most common skin manifestation was acanthosis nigricans (87.2%), frequently found on the neck and axilla, followed by cutaneous infections, primarily dermatophyte infections. Striae and acrochordons were also prevalent, particularly in individuals with higher grades of acanthosis nigricans. Other observed skin conditions included acne, xerosis, keratosis pilaris, gynaecomastia, and hirsutism.

A significant correlation was found between higher BMI, elevated HOMA-IR values, and the presence of more cutaneous manifestations. Individuals with a BMI above the 95th percentile exhibited more skin issues compared to those in the 85th to 95th percentile range. Similarly, 59% of cases with HOMA-IR values greater than 2.6 showed more skin manifestations compared to those with lower values.

These findings emphasize the importance of monitoring BMI and HOMA-IR levels to identify and manage skin issues in obese individuals, helping to

prevent future health complications. This understanding is crucial for pediatricians and dermatologists to ensure early diagnosis and better patient outcomes, highlighting the essential role of dermatologic assessment in the comprehensive care of obese individuals.

BIBLIOGRAPHY

1. Balke H, Nocito A. Vom Schönheitsideal zur Krankheit - eine Reise durch die Geschichte der Adipositas [A trip through the history of obesity]. Praxis (Bern 1994). 2013 Jan 16;102(2):77-83. doi: 10.1024/1661-8157/a001169. PMID: 23384950.
2. Raj M, Kumar RK. Obesity in children & adolescents. Indian J Med Res. 2010 Nov;132(5):598-607. PMID: 21150012; PMCID: PMC3028965.
3. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults. The Lancet. 2017 Dec 16;390(10113):2627-42
4. Ranjani H, Mehreen TS, Pradeepa R, et al. Epidemiology of childhood overweight & obesity in India: A systematic review. Indian J Med Res. 2016;143:160-74
5. GBD 2019 Risk Factor Collaborators. Global Burden of 87 Risk Factors in 204 Countries and Territories, 1990–2019: a systematic analysis for the global burden of disease study 2019. Lancet. 2020;396:1223– 49
6. Styne DM, Arslanian SA, Connor EL, et al. Pediatric obesity assessment, treatment, and prevention: an endocrine society clinical practice guideline. J Clin Endocrinol Metab. 2017;102:709-57
7. Hampl SE, Hassink SG, Skinner AC, et al. Clinical Practice Guideline for the Evaluation and Treatment of Children and Adolescents With Obesity.

Pediatrics. 2023 Feb 1;151(2) doi: 10.1542/peds.2022-060640. Erratum in: Pediatrics. 2024 Jan 1;153(1) doi: 10.1542/peds.2023-064612. PMID: 36622115.

8. Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, et al. CDC growth charts: United States. Adv Data. 2000;314:1-27
9. Khadilkar V, Yadav S, Agrawal KK, et al. Revised IAP growth charts for height, weight and body mass index for 5- to 18-year-old Indian children. Indian Pediatr. 2015;52:47-55
10. Bouchard C, Tremblay A, Després JP, et al. The response to long-term overfeeding in identical twins. N Engl J Med. 1990 May 24;322(21):1477-82. doi: 10.1056/NEJM199005243222101. PMID: 2336074.
11. Swinburn BA, Caterson I, Seidell JC, et al. Diet, nutrition and the prevention of excess weight gain and obesity. Public Health Nutr. 2004 Feb;7(1A):123-46 doi: 10.1079/PHN2003585. PMID: 14972057.
12. Bays HE, Gonzalez-Campoy JM, Bray GA et al. Pathogenic potential of adipose tissue and metabolic consequences of adipocyte hypertrophy and increased visceral adiposity. Expert Rev Cardiovasc Ther 2008;6:343–68
13. Lumeng CN, Saltiel AR. Inflammatory links between obesity and metabolic disease. J Clin Invest. 2011;121(6):2111-7. doi: 10.1172/JCI57132. PMID: 21633179.

14. Shoelson, S. E., Herrero, L., & Naaz, A. (2007). Obesity, inflammation, and insulin resistance. Gastroenterology, 132(6), 2169–2180. https://doi.org/10.1053/j.gastro.2007.03.059

15. Lakshmi C. Hormone therapy in acne. Indian J Dermatol Venereol Leprol. 2013 May-Jun;79(3):322-37. doi: 10.4103/0378-6323.110765. PMID: 23619437.

16. Mainieri F, Tagi VM, Chiarelli F. Insulin resistance in children. Curr Opin Pediatr. 2022 Aug 1;34(4):400-406. doi:10.1097/MOP.0000000000001151. Epub 2022 Jul 5. PMID: 35796641.

17. Zhao X, An X, Yang C, Sun W, Ji H, Lian F. The crucial role and mechanism of insulin resistance in metabolic disease. Front Endocrinol (Lausanne). 2023 Mar 28;14:1149239. doi: 10.3389/fendo.2023.1149239. PMID: 37056675; PMCID: PMC10086443.

18. Gutch M, Kumar S, Razi SM, Gupta KK, Gupta A. Assessment of insulin sensitivity/resistance. Indian J Endocrinol Metab. 2015 Jan-Feb;19(1):160-4. doi: 10.4103/2230-8210.146874. PMID: 25593845; PMCID: PMC4287763.

19. Fox C, Bernardino L, Cochran J, Essig M, Bridges KG. Inappropriate Use of Homeostasis Model Assessment Cutoff Values for Diagnosing Insulin Resistance in Pediatric Studies. J Am Osteopath Assoc. 2017 Nov

1;117(11):689-96. doi: 10.7556/jaoa.2017.135. PMID: 29084322; PMCID: PMC5868754.

20. Caroppo F, Galderisi A, Ventura L, Belloni Fortina A. Metabolic syndrome and insulin resistance in pre-pubertal children with psoriasis. Eur J Pediatr. 2021 Jun;180(6):1739-1745. doi: 10.1007/s00431-020-03924-w. Epub 2021 Jan 22. PMID: 33483797; PMCID: PMC8105196.

21. Farage MA, Miller KW, Elsner P, Maibach HI. Functional and physiological characteristics of the aging skin. Aging Clin Exp Res. 2008 Jun;20(3):195-200. doi: 10.1007/BF03324769. PMID: 18594185.

22. de Farias Pires T, Azambuja AP, Horimoto AR, et al. A population-based study of the stratum corneum moisture. Clin Cosmet Investig Dermatol. 2016;9:79-87

23. Yosipovitch G, DeVore A, Dawn A. Obesity and the skin: skin physiology and skin manifestations of obesity. J Am Acad Dermatol. 2007;56(6):901-16. quiz 917-920

24. Das S, Reynolds RV. Recent advances in acne pathogenesis: implications for therapy. Am J Clin Dermatol. 2014;15(6):479-88

25. Cramer MN, Jay O. Explained variance in the thermoregulatory responses to exercise: the independent roles of biophysical and fitness/fatness-related factors. J Appl Physiol (1985). 2015;119(9):982-89

26. Rockson SG. Obesity and lymphatic dysfunction. Lymphat Res Biol. 2015 Jun;13(2):100- 02. doi: 10.1089/lrb.2015.29000.sgr. PMID: 26091213.

27. Fantuzzi G. Adipose tissue, adipokines, and inflammation. J Allergy Clin Immunol. 2005;115(5):911-12 doi:10.1016/j.jaci.2005.02.023.

28. Stuart CA, Pate CJ, Peters EJ. Prevalence of acanthosis nigricans in an unselected population. Am J Med. 1989;87:269-72

29. Yamazaki H, Ito S, Yoshida H. Acanthosis nigricans is a reliable cutaneous marker of insulin resistance in obese Japanese children. Pediatr Int. 2003;45:701-05

30. Simmonds M, Llewellyn A, Owen C, Woolacott N. Predicting adult obesity from childhood obesity: a systematic review and meta-analysis. Obes Rev. 2016;17(2):95-107.

31. Phiske MM. An approach to acanthosis nigricans. Indian Dermatol Online J. 2014 Jul;5(3):239-49. doi: 10.4103/2229-5178.137765. PMID: 25165638; PMCID: PMC4144206.

32. El Safoury OS, Abdel Hay RM, Fawzy MM, Kadry D, Amin IM, Abu Zeid OM, Rashed LA. Skin tags, leptin, metabolic syndrome and change of the lifestyle. Indian J Dermatol Venereol Leprol. 2011;77:577-81

33. Liy-Wong C, Kim M, Kirkorian AY, et al. Hidradenitis suppurativa in the pediatric population: an international, multicenter, retrospective, cross-

sectional study of 481 pediatric patients. JAMA Dermatol. 2021;157:385-91

34. Pasquali R, Gambineri A. Insulin-sensitizing agents in polycystic ovary syndrome. Eur J Endocrinol. 2006;154(6):763-75

35. Falagas ME, Athanasoulia AP, Peppas G, et al. Effect of body mass index on the outcome of infections: a systematic review. Obes Rev. 2009;10:280-89

36. Miró EM, Sánchez NP. Cutaneous manifestations of infectious diseases. In: Atlas of Dermatology in Internal Medicine. New York: Springer; 2012. 77-119.

37. Sreeramoju P, Porbandarwalla NS, Arango J, et al. Recurrent skin and soft tissue infections due to methicillin-resistant Staphylococcus aureus requiring operative debridement. Am J Surg. 2011;201:216-220.

38. Baselga Torres E, Torres-Pradilla M. Manifestaciones cutáneas en niños con diabetes mellitus y obesidad. Actas Dermosifiliogr. 2014;105:546-557.

39. Huang AH, Roh YS, Sutaria N, et al. Real-world comorbidities of atopic dermatitis in the pediatric ambulatory population in the United States. J Am Acad Dermatol. 2021;85:893-900.

40. Yudkin JS, Stehouwer CD, Emeis JJ, et al. C-reactive protein in healthy subjects: associations with obesity, insulin resistance, and endothelial

dysfunction: a potential role for cytokines originating from adipose tissue? Arterioscler Thromb Vasc Biol. 1999;19:972-978.

41. Silverberg JI, Kleiman E, Lev-Tov H, et al. Association between obesity and atopic dermatitis in childhood: a case-control study. J Allergy Clin Immunol. 2011;127:1180-1186

42. Rana JS, Nieuwdorp M, Jukema JW, Kastelein JJ. Cardiovascular metabolic syndrome: an interplay of obesity, inflammation, diabetes and coronary heart disease. Diabetes Obes Metab. 2007;9:218-232.

43. Drucker AM, Pope EI, Field AE, et al. Association between maternal pre-pregnancy body mass index, gestational weight gain, and offspring atopic dermatitis: a prospective cohort study. J Allergy Clin Immunol Pract. 2019;7:96-102.e2.

44. Hrolfsdottir L, Schalkwijk CG, Birgisdottir BE, et al. Maternal diet, gestational weight gain, and inflammatory markers during pregnancy. Obesity. 2016;24:2133-2139.

45. Stolzman S, Irby MB, Callahan AB, Skelton JA. Pes planus and pediatric obesity: a systematic review of the literature. Clin Obes. 2015 Apr;5(2):52-59. doi: 10.1111/cob.12091. Epub 2015 Mar 23. PMID: 25808780; PMCID: PMC4631254.

46. Nino M, Franzese A, Ruggiero Perrino N, Balato N. The effect of obesity on skin disease and epidermal permeability barrier status in children. Pediatr Dermatol. 2012;29(5):567-570.

47. Tollefson MM, Bruckner AL, Section On Dermatology. Atopic dermatitis: skin-directed management. Pediatrics. 2014;134(6) :e1735-44

48. Monteiro Rodrigues L, Palma L, Santos O, Almeida M, Bujan J, Tavares L. Excessive weight favours skin physiology-up to a point: another expression of the obesity paradox. Skin Pharmacol Physiol. 2017;30(2):94-101.

49. Unluhizarci K, Karaca Z, Kelestimur F. Role of insulin and insulin resistance in androgen excess disorders. World J Diabetes. 2021 May 15;12(5):616-629. doi: 10.4239/wjd.v12.i5.616. PMID: 33995849; PMCID: PMC8107978.

50. Gupta M. Cutaneous manifestations of obesity in children: a prospective study. Indian J Paediatr Dermatol. 2017;18:28-30.

51. Nithun TM, Ranugha PS, Betkerur JB, Shastry V. Association of acanthosis nigricans and insulin resistance in Indian children and youth-a HOMA2 IR based cross sectional study. Indian Dermatol Online J. 2019;10(3):272-78

52. Hsu HS, Chen W, Chen SC, Ko FD. Colored striae in obese children and adolescents. Zhonghua Min Guo Xiao Er Ke YiXue Hui Za Zhi. 1996;37(5):349-52.

53. Plascencia Gómez A, Vega Memije ME, Torres Tamayo M, Rodríguez Carreón AA. Dermatosis en pacientes con sobrepeso y obesidad y su relación con la insulina. Actas Dermosifiliogr. 2014;105:178-185.

54. Gulanikar AD, Abrol A. Cutaneous manifestations of Paediatric Obesity: Relation with insulin resistance. IP Indian J Clin Exp Dermatol 2021;7(1):8-13.

55. Boza JC, Trindade EN, Peruzzo J, Sachett L, Rech L, Cestari TF. Skin manifestations of obesity: A comparative study. J Eur Acad Dermatol Venereol 2012;26:1220-3.

Azadi Ka Amrit Mahotsav

BLDE

(DEEMED TO BE UNIVERSITY)

Declared as Deemed to be University u/s 3 of UGC Act, 1956

Accredited with 'A' Grade by NAAC (Cycle-2)

The Constituent College

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 702/2022-23

30/8/2022

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Friday, 26th August, 2022 at 3.30 p.m. in the Department of Pharmacology** scrutinizes the Synopsis of Post Graduate Student of BLDE (DU)'s Shri B.M.Patil Medical College Hospital & Research Centre from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "CUTANEOUS MANIFESTATIONS IN CHILDHOOD OBESITY: A PROSPECTIVE CROSS-SECTIONAL STUDY

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: Dr. Pooja Kotian

NAME OF THE GUIDE: Dr. Arun C. Inamadar, Professor & HoD, Dept. of Dermatology.

r. Santoshkumar Jeevangi
hairperson
EC, BLDE (DU),
IJAYAPURA
Chairman,
tional Ethical Committee,
(Deemed to be University)

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA
MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103, Karnataka

llowing documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

CONSENT FORM

B.L.D.E. (Deemed to be University) SHRI B.M PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA-586103

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION/ RESEARCH

TITLE OF THE PROJECT :- A STUDY OF CUTANEOUS MANIFESTATIONS IN OVERWEIGHT AND OBESE PEDIATRIC POPULATION

PG GUIDE **:-** DR. ARUN C. INAMADAR

PG STUDENT **:-** DR. POOJA KOTIAN

RISK AND DISCOMFORTS **:-** No

BENEFITS:

I understand that my participation in the study will have no direct benefit to me other than the potential benefit of the research and education.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subject to confidentiality. Information of sensitive personal nature will not be part of the medical record but will be stored in the investigations research file. If the data are used for publication in the medical literature or for teaching purposes, no name will be used, and other identifiers such as photographs will be used only with special written permission. I understand that I may see the photograph before giving permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time; Dr. POOJA KOTIAN at the department of DERMATOLOGY 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 the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw the consent and discontinue participation in the

study at any time without prejudice. I also understand that Dr. ARUN C INAMDAR may terminate my participation in the study after he has explained the reasons for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to a child resulting directly from the child's participation in this study, if such injury were reported promptly, the appropriate treatment would be available to the child. But no further compensation would be provided by the hospital. I understand that by my agreement to participate in this study and not waiving any of my legal rights.

I have explained to _____ the purpose of the research, the procedures required, and the possible risks to the best of my ability.

_____ _____

Dr. POOJA KOTIAN Date

(Investigator)

PARENTS / GUARDIAN CONSENT STATEMENT:

We confirm that DR POOJA KOTIAN is doing **A STUDY OF CUTANEOUS MANIFESTATIONS IN CHILDHOOD OBESITY**, has explained to us the purpose of the research and the study procedure. We are willing to give as much information as required for the study and consent for investigations and the possible discomforts as well as benefits. We have been explained all the above in detail in our own language, and we understand the same. Therefore, we agree to give consent for the child's participation as a subject in this research project.

Parents/ Guardian signature Date :

PROFORMA

- NAME
- PT ID
- DOB
- AGE
- SEX
- ADDRESS
- MOBILE NO

AGE AT WHICH THE SKIN LESIONS DEVELOPED :

FAMILY HISTORY

H/O SIMILAR COMPLAINTS IN SIBLINGS: YES NO

GENERAL PHYSICAL EXAMINATION:

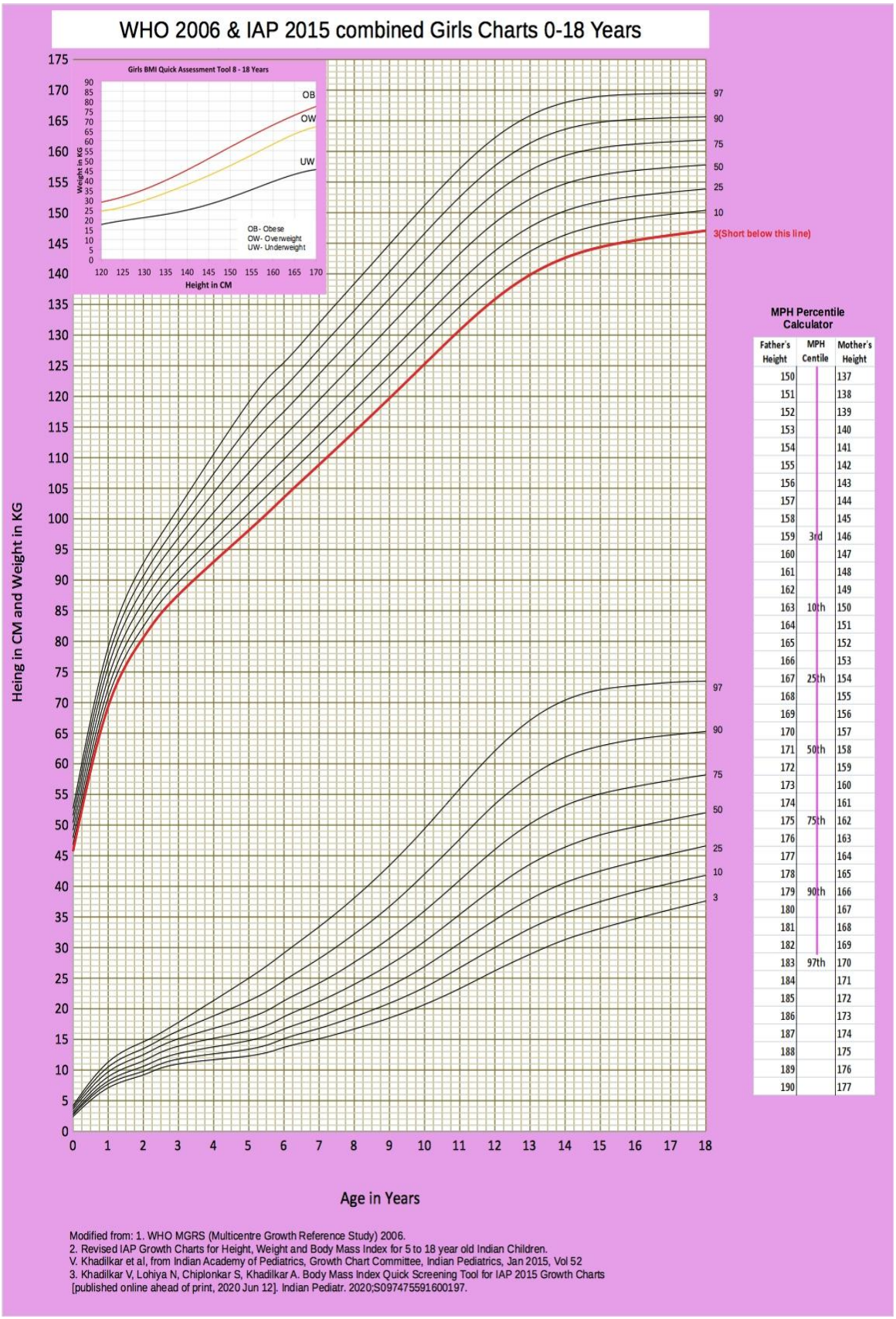
- WEIGHT: KG
- HEIGHT: CM
- BMI : KG/M^2

CUTANEOUS EXAMINATION:

INVESTIGATIONS:

- SERUM FASTING INSULIN LEVEL :
- SERUM FASTING GLUCOSE :
- HOMA – IR :

DIAGNOSIS



Father's Height	MPH Centile	Mother's Height
150		137
151		138
152		139
153		140
154		141
155		142
156		143
157		144
158		145
159	3rd	146
160		147
161		148
162		149
163	10th	150
164		151
165		152
166		153
167	25th	154
168		155
169		156
170		157
171	50th	158
172		159
173		160
174		161
175	75th	162
176		163
177		164
178		165
179	90th	166
180		167
181		168
182		169
183	97th	170
184		171
185		172
186		173
187		174
188		175
189		176
190		177

Figure 14 : WHO and IAP 2015 – Height and Weight Charts – Girls – (0-18 years)

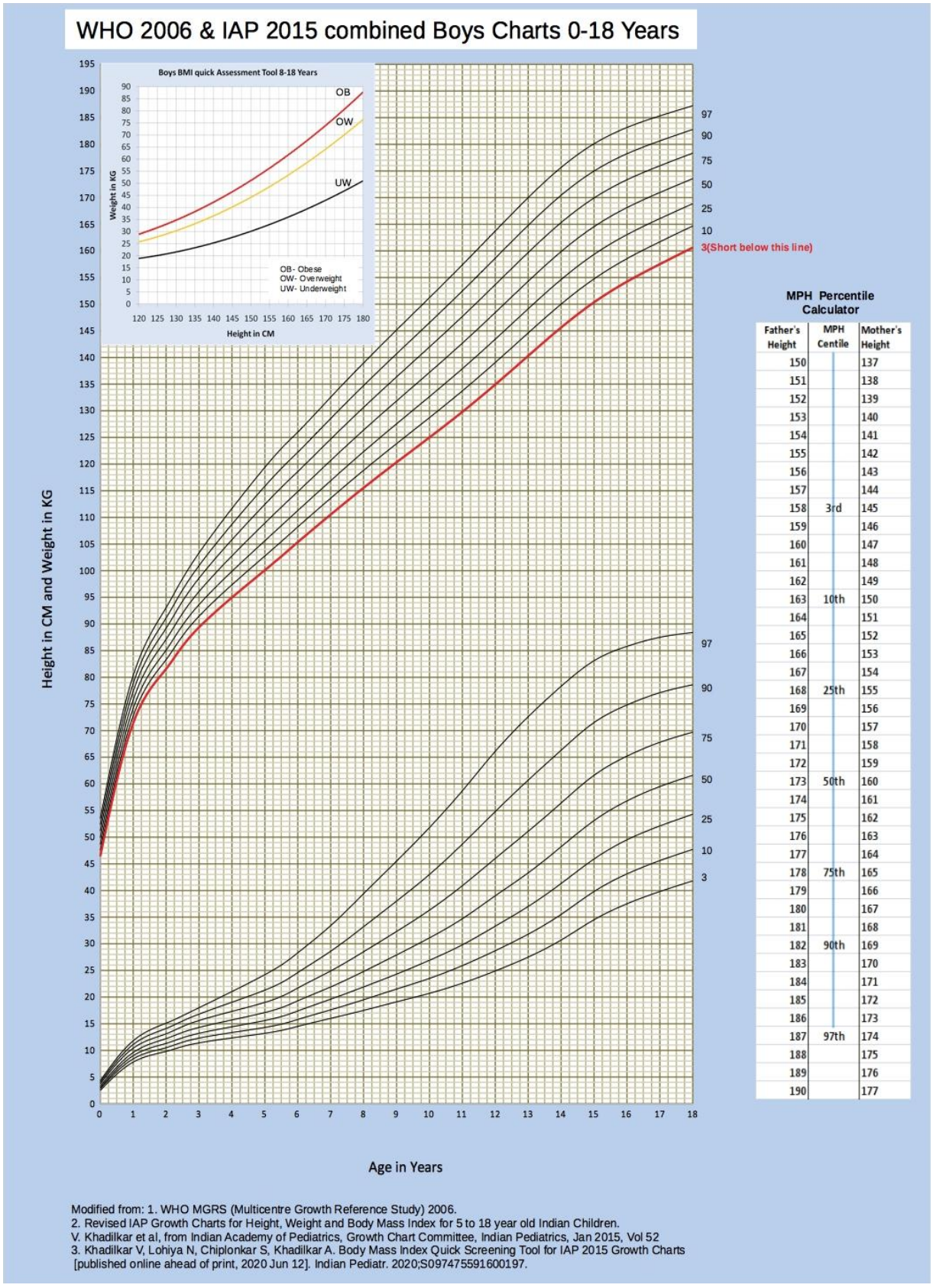


Figure 15 : WHO and IAP 2015 – Height and Weight Charts – Boys – (0-18 years)

KEY TO MASTERCHART

TC = TINEA CORPORIS/ CRURIS	AMN = ACQUIRED MELANOCYTIC NEVI
SD = STRIAE DISTENSIAE	CMN = CONGENITAL MELANOCYTIC NEVI
AV = ACNE VULGARIS	GYM = GYNAECOMASTIA
KP = KERATOSIS PILARIS	FOL = FOLLICULITIS
LP = LICHEN PLANUS	LN = LICHEN NITIDUS
ECZ = ECZEMA	HKD = HYPERKERATOSIS DERMATOSIS
AD = ATOPIC DERMATITIS	XER = XEROSIS
PIH = POST INFLAMMATORY HYPO/HYPERPIGMENTATION	SCAB = SCABIES
PMLE = POLYMORPHIC LIGHT ERUPTIONS	VV = VITILIGO VULGARIS
INT = INTERTRIGO	LSC = LICHEN SIMPLEX CHRONICUS

P.VER = PITYRIASIS VERSICOLOR	P.ALBA = PITYRIASIS ALBA
H SCAR = HYPERTROPHIC SCAR	A SCAR = ATROPHIC SCAR
CALM = CAFÉ AU LAIT MACULES/ PATCHES	WAR = WARTS
AN = ACANTHOSIS NIGRICANS	KE = KERATOLYSIS EXFOLIATA

MASTERCHART

Name	Age	Sex	FH - Obesity	Age -skin lesions developed	Weight (Kg)	HEIGHT (cm)	BMI Index	85-95 percentile (overweight)	>95 percentile (Obese)	Fasting Insulin	FBS	HOMA IR	Diagnosis	TINEA	Trichorrhexis	Acanthosis nigricans	STRIAE	CMN	AMN	INTERTRIGO	ECZEMA	HKD	Atopic Dermatitis	P. ALBA	SCABIES	Folliculitis	Keratosis Pilaris	ALOPECIA AREATA	P V eisicolor	IBK	PIH	Café au lait	Lichenoid PMLE	Prurigo Simplex	Phrynoderma	Gyaenaecomastia	POLYTHELIA	Sebortheic Capitis	PPD	VITILIGO VULGARIS	HIRSUTISM	XEROSIS	ACNE VULGARIS	HYPERTROPHIC SCAR	Atrophic scar	Lichen Planus	LPN	Keloid	Warts	Lichen nitidus
Sahil Dhanwadkar	14 M		P	13	84	168	29.76	0	1	13	98	3.1	PPD,AN,SD,CMN	N	P	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Sanika Kamble	14 F		P	13	55	138	28.8	0	1	10.9	86	2.3	TC+AN+SD	P	P	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Abhishek Hosur	14 M		P	13	74	132	32.03	0	1	16.42	103	4.2	PMLE+ANs+SD	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Avinash Kalmadi	15 M		P	12	70	167	25.02	1	0	16.05	96	3.8	AMN+CALM+AN+SD+GYM	N	P	P	P	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Vaibhavi S Pattan	14 F		N	14	67	163	25.22	1	0	10.9	91	2.4	SC+AN+SD	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N
Vikas Harijan	14 M		P	14	61	146	28.62	0	1	9.46	97	2.3	VV+AN+SD	N	P	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Samarth Timea	15 M		N	13	62	156	25.48	0	1	16.27	94	3.8	TC+AN	P	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Shrilaxmi Atanur	15 F		N	13	62	156	25.48	1	0	12.28	103	3.1	TC+AN+HR	P	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Sumeet S Banjari	15 M		N	15	65	151	28.51	0	1	17.04	90	3.8	Scab+TC+AN	P	P	P	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Vinod Kumbar	17 M		P	16	82	161	31.63	0	1	10.68	102	2.7	TC+AN+SD	P	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Shrikanth Rebinal	17 M		P	16	80	164	29.74	0	1	15.37	96	3.6	Ecz+AN+SD+POLYT	N	P	P	P	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Soumya Hebbal	17 F		P	16	96	160	37.5	0	1	26	109	7	TC+AN+SD	P	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Rishikesh Talwar	9 M		N	9	45	130	26.63	0	1	6.83	93	1.6	Ecz+AN	N	N	P	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Akshatha Bagewa	17 F		P	16	55	138	28.8	0	1	48	122	15	TC+AN+SD HKD	P	N	P	P	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Karthik Nathikar	17 M		P	17	103	178	34.54	0	1	14	102	3.5	AA+CALM+AN+SD+GYM	N	P	P	P	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Harish irappa	14 M		P	13	97	174	32.04	0	1	27.24	102	6.9	TC+SD	P	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Srujan gowda	10 M		N	10	48	134	26.73	0	1	9.88	99	2.4	P.Alba+AN+Xer+PIH	N	N	P	N	N	N	N	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Sidhartha Gowda	11 M		N	11	68	160	26.79	0	1	10.85	98	2.6	TC+AN+SD+P.Alba+CMN	P	N	P	P	N	N	N	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Karthik mali	17 M		P	15	97	170	33.56	0	1	16.27	94	3.8	AN+SD+AMN	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Madan	15 M		P	15	67	163	25.22	1		10	94	2.3	G1AV+AN+SD	N	N	P		P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P								
Laxmi Anantpur A	12 F		N	10	53	141	26.66	0	1	12	91	2.7	AN+CALM+SD	P	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Aditi Awati	8 F		Y	8	49	138	25.2	0	1	8	94	1.9	FECZ	N	N	N	N	N	P	N		N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Shravya Laxman	11 F		N	11	68	160	25.78	0	1	10.85	98	2.6	TC+AN+SD+P.Alba+CMN	P	N	P	P	N	N	N	N	N	P	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
													G1AV+AMN+Hscar+CALM+AN																																											
Daneshwari rames	15 F		P	13	81	160	31.64	0	1	13	104	3.3		N	N	P	N	N	N	N	N	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Prathama Pawale	17 M		N	15	114	171	39	0	1	23.3	90	5.2	P.Alba+FolT+LSC+AN+G1AV	N	N	P	P	N	P	N	N	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P		N	N	N	N	N	N	N
Kavya Upadya	8 F		P	7	52	140	26.53	0	1	8	90	1.8	AD+HScar+KP	N	N	N	P	N	P	N	N	N	N	P		N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N
Pramod Parashur	11 M		N	11	50	143	24.5	0	1	9	92	2	SCAB+CMN+PIH+Grade1AV+AN	N	P	P	P	N	P	P	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Shravya Laxman	13 F		N	13	53	153	25.92	0	1	9	87	1.9	TC+AN+Striae+AMN	P	N	P	P	N	P	P		N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Harshita B	11 F		P	10	51	140	26.02	0	1	9	94	2.1	AD+AN+PIH+AMN	N	N	P	N	N	P	N	N	N	N	P	N	N	N	N	N	N	N	NP	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
Daksh Metagar	9 M		N	9	49	138	25.2	0	1	8.78	85	1.8	PS+PHRYM+AN+AMN+PIH+SD	N	P	P	P	N	P	P	N	N	N	P	P	N	N	N	N	N	N	NP	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N

31	Daksh Metagar	9	M	N	9	48	138	25.2	0	1	8.78	85	1.8	PS+ PHRYN+ AN + AMN +PIH + SD	N	P	P	P	N	P	N	N	N	N	N	N	N	N	N	N	N	P	N	N	P	P		N	N	N	N	N	N	N	N	N	N	N	N	N	N
32	Soubhagya B	18	F	P	16	83	156	34.1	0	1	14	102	3.5	IBR + HKD + AN + AMN	N	P	P	P	N	P	N	N	P	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
33	Praveen Lamani	15	M	P	14	73	168	26	1	0	89	28	6.3	G2 AV + TC+ KP + Striae + AN	P	P	P	P	N	P	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	P	N	N	N	N	N	N	N
34	Shravan	10	M	N	10	45	138	24	0	1	7	99	1.5	AN + P VER	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
35	Rakshita	17	F	P	15	58	153	24.78	1	0	8	94	1.9	AN + SD	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
36	Prajwal Danigond	16	M	N	16	86	171	29.41	0	1	11	98	2.7	TC + AMN + AN + SD + HYPER scar	P	N	P	P	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N
37	Daneshwari Kaval	15	F	N	15	81	160	31.64	0	1	16	101	4	G3 AV + AN + HYPER scar + AMN + CAl	N	N	P	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	P	P	N	N	N	N	N	N
38	Samarth Ibrahimp	16	M	P	16	68	159	26.9	1	0	7	91	1.6	TC+ AN + SD + Acne + PIH	P	N	P	P	N	N	N	N	N	N	N	N	N	P	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N
39	Srajana Despande	17	F	P	16	65	154	27.41	1	0	11	98	2.7	V + AN + AMN + SD + Xer	N	N	P	P	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	P	N	N	N	N	N	N	N	N
40	Kusum	17	F	P	15	91	158	36.45	0	1	16	98	3.9	AV + AN + SD + ATR scar + CALM	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	P	N	P	N	N	N	N	N
41	Shivanand Umara	14	M	P	13	70	151	30.7	0	1	18	103	4.6	LP + PIH + AN+ CALM + CMN	N	N	P	N	P	N	N	N	N	N	N	N	N	N	N	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N
42	Shriharsha	11	M	N	11	57	147	26.3	0	1				AN + TC + ECZ	P	N	P	N	N	N	N	P	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
43	Sumeet Rathod	12	M	N	12	60	147	27.7	0	1	10.35	96	2.5	AN+ PIH+ HYPER scar+ ACHRO	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N
44	Ningraj	12	M	N	12	65	156	26.71	0	1	12.35	93	2.8	AN + XER + PIH	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N
45	Samruddhi	14	F	P	14	52	145	24.7	1		42.7	96	10	DPN+AN+ SD + CMN+ ACHRO	N	P	P	P	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N
46	Mahesh	18	M	N	16	77	163	28.98	0	1	23	102	5.8	TC + AN+ SD+ CALM+ HYPER scar	P	P	P	P	N	N	N	N	N	N	N	N	N	P	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N
47	Ayesha	10	F	P	10	54	150	24	0	1	8	92	1.8	AN+P alba + PIH + SD +KEL + CMN	N	N	P	P	P	N	N	N	N	N	P	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N
48	Durga	8	F	N	8	40	118	28.73	0	1	7	97	1.5	AN+P alba + PIH + SD + ECZ	N	N	P	P	P	N	N	N	P	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
49	Veena	12	F	P	11	65	144	31.35	0	1	14	107	3.7	AV + AN + SD + PIH+ AMN	N	P	P	N	N	P	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
50	Pruthvi	10	M	N	10	53	136	28.65	0	1	18	101	4.5	TC + AN + SD +PIH	P	N	p	P	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
51	Sandeep	14	M	P	12	66	158	25.2	1	0	9	97	2.2	TC + AN + POLYT + AV + SD+ CALM+ H	P	P	P	P	N	P	N	N	N	N	N	N	N	N	N	N	N	P	P	N	N	N	N	P	N	N	N	N	N	P	N	N	N	N	N	N	N
52	Laxmi Bandi	15	F	N	15	78	158	30.84	0	1	23	102	5.8	Vart + AN + SD + Achro + AV	N	P	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	P	N
53	Darshan	16	M	P	15	124	176	40.03	0	1	24	97	5.7	TC + AN + SD +Atro scar+Int +AcHRO	P	P	P	P	P	N	P	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	P	N	N	N	N	N
54	Kiran sajjan	9	M	P	9	132	44	25.2	0	1	14.3	93	3.3	AN+KP + SD +ATR scar + AMN	N	P	P	P	N	P	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N
55	Hashim kalar	13	M	P	12	74	154	31.2	0	1	17	102	4.3	AMN + Warts + SD + KP	N	N	N	P	N	P	N	N	N	N	N	N	P		N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N
56	Chandan Koli	16	M	N	16	75	162	28.58	0	1	8	93	1.3	TC + SD + Fol + Keloid + KP+ AV	P	N	N	P	N	N	N	N	N	N	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	P	N	N
57	Satish Kumar	15	M	N	14	78	159	31.24	0	1	16	91	3.6	AV + Warts + KP + SD	N	N	N	P	N	N	P	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	P	N
58	Karthik C	14	M	P	13	70	160	27.34	0	1	23.5	87	5	Hyper scar + AMN + AV + AN + Achro + S	N	N	P	P	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	P	N	N	P	N	N	N
59	Ashish D	16	M	N	15	80	163	31.24	0	1	19	94	4.4	AN + Striae + PIH + AMN + Ecz+ Xer	N	P	P	P	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N
60	Sanjana	16	F	P	14	64	154	26.9	1	0	21	94	4.9	AN + Striae + PIH + AMN + AV + Xerso	N	N	P	P	P	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	P	P	N	N	N	N	N	N	N
61	Chandrashekar	14	M	P	14	74	156	30.1	0	1	13.2	93	3	TC + SD + CALM	P	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
62	Krishna K	16	M	P	15	80	167	28.69	0	1	25.21	97	6	TC + AMN + SD +Fol + SD+ AV + HKD + X	P	P	P	P	N	P	N	N	P	N	N	N	P	P	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	P	N	N	N	N	N	N	N
63	Suniita	13	F	N	12	60	152	25.97	0	1	14	97	3.4	AN + SD + AV + PIH + Xer + TC	P	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	P	P	N	N	N	N	N	N	N
64	Prajwal chavan	15	M	P	14	70	167	25.1	1	0	12	87	2.6	AN + SD + hyper scar + AV + Xer	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	P	P	P	N	N	N	N	N	N
65	Sufiyan K	15	M	P	13	64	155	26.64	0	1	24	97	5.2	AN + SD + AV + PIH + Xer + Achro + P alt	P	P	P	P	P	N	N	N	N	N	P	N	N	P	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	P	N	P	N	N	N	N	N
66	Amit	14	M	N	14	64	158	25.64	0	1	21.5	94	5	AN + Striae + AV + P Alba+ Xer + TC	P	P	P	P	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	P	N	N	N	N	N	N	N
67	Amogh	7	M	N	7	44	125	28.16	0	1	16	92	3.6	AN + PIH + AMN + CALM + LN + Achr + F	N	P	P	N	N	P	N	N	N	N	P	N	N	N	N	N	N	P	P	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P
68	Vasavi Uppin	15	F	P	13	81	160	31.64	0	1	8.5	80	1.7	An + Achr+ SD + CALM + PN + PIH + Stri	N	P	P	P	N	N	P	N	N	N		N	N	P	N	N	N	P	P	N	N	N	N	N	P	N	N	N	P	P	N	N	N	N	N	N	N
69	Rakesh	8	M	N	8	57	140	29.08	0	1	9	90	2	AMN + SD + AV + P Alba+ Xer + KE	N	N	N	P	N	P	N	N	N	N	P	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N

70	Prasad	9	M	N	9	57	149	25.67	0	1	9	84	1.9	AN + SD + AV + P Alba+ Xer+ TC + Achro	P	P	P	P	N	P	N	N	N	N	P	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	P	P	N	P	N	N	N	N	N
71	Ambrish	16	M	P	16	67	161	25.85	0	1	11	84	2.3	AN + SD+ AV + Fol + Xer + TC + AchrO + /	P	P	P	P	N	N	N	N	N	N	N	N	P	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	P	P	N	P	N	N	N	N	N
72	Swaroop	9	M	N	9	57	147	26.3	0	1	11	92	2.5	AN + SD+ P Alba+ Xer+ CALM + KP	N	N	N	P	N	N	N	N	N	N	P	N	N	P	N	N	N	N	P	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N
73	Nikethan	15	M	P	15	85	168	30.12	0	1	21	92	4.8	AN + SD + Int + XeR + AV	N	P	P	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N
74	Shashidar	14	F	N	13	60	152	25.97	1	0	8	84	1.7	AN + SD+ P Alba+ Xer+ CALM	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N
75	Soumya Y	15	F	P	15	90	160	35.16	0	1	21	89	4.6	AN + SD + chelitis + PIH + Xer+ atrop scal	N	N	P	P	N	P	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	P	N	N	P	N	N	N	N	N
76	Mahalaxmi	10	F	P	10	65	148	29.65	0	1	11	88	2.4	AMN + SD +Fol	N	N	N	P	N	P	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N
77	Shivakumar	16	M	P	15	103	157	41.79	0	1	27	88	5.9	TC + AMN + SD +Fol + AV +AN	P	P	P	P	N	P	N	N	N	N	N	N	P	N	N	N	N	P	N	N	N	N	P	N	P	N	N	N	P	P	N	P	N	N	N	N	N
78	Riya	10	F	P	10	132	44	25.2	0	1	18	90	4	AN + SD + Int+ Xer + AV	N	P	P	N	N	N	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N
79	Rahul	14	M	N	13	70	160	27.34	0	1	7	84	1.5	AN + SD + PIH + AMN + Ecz + XeR	N	P	P	P	N	N	P	P	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	P	N	N	N	N	N	N	N	N