A STUDY OF METABOLIC SYNDROME IN PSORIATIC PATIENTS

Dissertation submitted for

M.D DEGREE BRANCH –XIII

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IRUNGALUR, TRICHY

TH TAMILNADU DR.MGR MEDICAL UNIVERSITY,

CHENNAI

APRIL-2016

CERTIFICATE

This is to certify that dissertation titled **"A STUDY OF METABOLIC SYNDROME IN PSORIATIC PATIENTS"** is a bonafide work done by **DR.J.SELVI** under my guidance and supervision in the Department of Biochemistry, Chennai Medical College Hospital and Research centre, Irungalur , Trichy during her post graduate course from 2013- 2016

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DECLARATION

I, **DR.J.SELVI** hereby solemnly declare that the dissertation title "**A STUDY OF METABOLIC SYNDROME IN PSORIATIC PATIENTS"** was done by me at Department of dermatology in Chennai Medical College Hospital and Research Centre, Irungalur, Trichy, under the supervision and Guidance of my professor and Head of the Department **Dr. Kalavathy Ponniraivan, MD.** This dissertation is submitted to Tamil Nadu Dr. M.G.R Medical University, towards partial fulfillment of requirement for the award of M.D. Degree (Branch- XIII) in Biochemistry.

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The work done by **DR.J.SELVI** on **"A STUDY OF METABOLIC SYNDROME IN PSORIATIC PATIENTS"** is under my supervision and I assure that this candidate will abide by the rules of the Ethical Committee.

Prof. Dr. Kalavathy Ponniraivan, MD.

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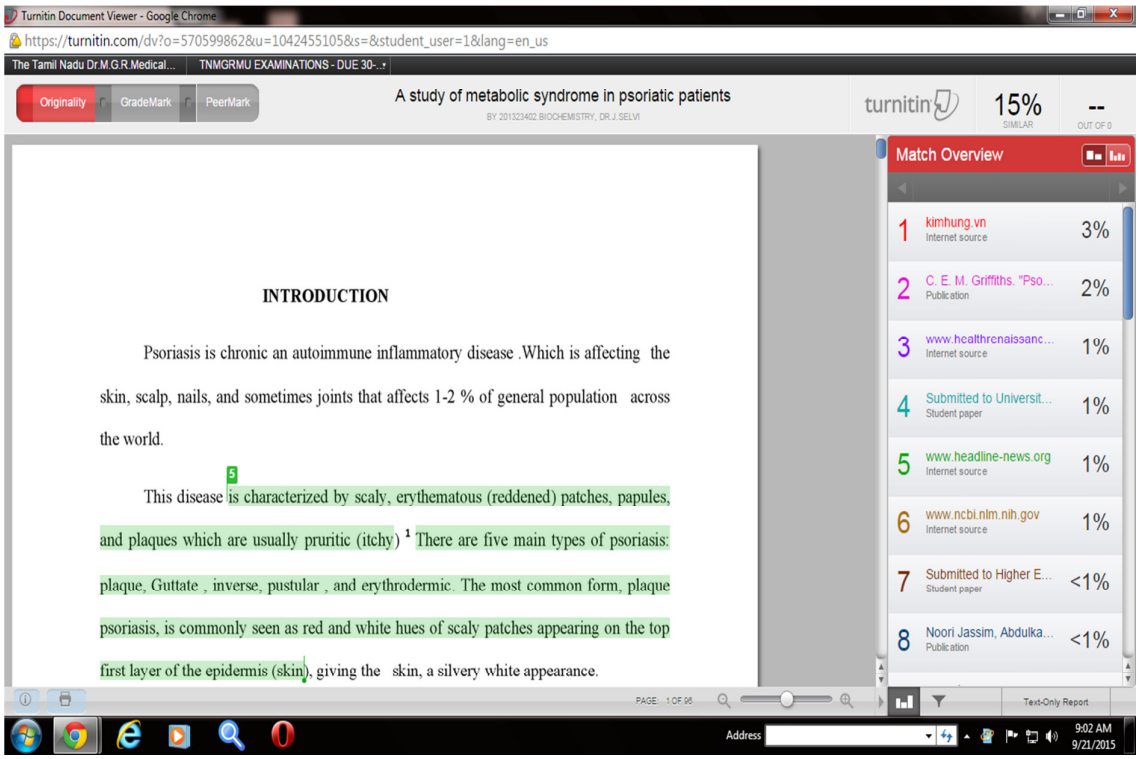

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CONTENTS

ABBREVIATIONS

WHO - World Health Organization

MS - Metabolic Syndrome

IDF - International Federation of Diabetes

NCEP - National Cholesterol Education Programme

CHD- Coronary Heart Disease

CVD- Cardio Vascular disease

IL- Interleukin

TNF- Tumour necrosis factor

BSA - Body Surface Area

DM - Diabetes Mellitus

PASI - Psoriasis Area Severity Index

PA - Psoriatic Arthritis

TC - Total Cholesterol

TAG – Triacylglycerol

HDL – High Density Lipoprotein

LDL - Low Density Lipoprotein

VLDL – Very Low Density Lipoprotein

FT3 - Free Triiodothyronine

FT4 - Free Tetraiodothyronine

TSH - Thyroid stimulating hormone

ABSTRACT

Title: A STUDY OF METABOLIC SYNDROME IN PSORIATIC PATIENTS

Background: Psoriasis is a chronic immune mediated inflammatory disorder of the skin and joints. Recent studies have shown increased prevalence of traditional cardiovascular risk factors such as diabetes mellitus, hypertension and metabolic syndrome. Thyroid gland hormones cause an increase of epidermal growth factor level which has an important role in keratinocyte proliferation, which may be involved in psoriasis disease.

Aims& Objectives:: To study the relationship between psoriasis and metabolic syndrome and to correlate the same with thyroid profile.

Materials and Methods: This study was conducted in Chennai Medical College Hospital and Research Centre, Irungalur, Trichy. Hundred psoriatic patients in the age group of 20-80 years in, and 30 controls were selected for this study. Fasting blood glucose by GLUCOSE OXIDASE AND PEROXIDASE method, serum lipid profile by enzymatic method and serum thyroid profile by ENZYME LINKED IMMUNOSORBANT ASSAY method.

Results: Our study shows that prevalence of metabolic syndrome in cases of psoriasis to be out of 37%, compared to controls among patients with other dermatological conditions to be 3.3%, ($p < 0.001$) and shows the FT3 (pg/ml) in cases Mean to be 2.117 and in Controls Mean was 2.037, Mean difference (0.0803), p value (0.53) .Shows the mean of FT4 (pg/ml) in cases Mean (1.284), Controls Mean (1.303),and the Mean difference was (-0.0193), p value (0.809) likewise TSH (mIU/ml) in cases Mean to be (3.580), in Controls Mean (2.277), and the Mean difference was (1.303), p value (0.001).

Conclusion: Patients of psoriasis have higher prevalence of metabolic syndrome and subclinical hypothyroidism than general population. Therefore identification of metabolic syndrome and thyroid profile testing can be done routinely for better management of psoriasis.

Key words: Metabolic Syndrome, Psoriasis, Thyroid hormones, inflammatory mediators.

INTRODUCTION

Psoriasis is chronic an autoimmune inflammatory disease .Which is affecting the skin, scalp, nails, and sometimes joints that affects 1-2 % of general population across the world.

This disease is characterized by scaly, erythematous (reddened) patches, papules, and plaques which are usually pruritic (itchy) [1]. There are five main types of psoriasis: plaque, Guttate, inverse, pustular, and erythrodermic. The most common form, plaque psoriasis, is commonly seen as red and white hues of scaly patches appearing on the top first layer of the epidermis (skin), giving the skin, a silvery white appearance.

Psoriasis is usually diagnosed based on the appearance of the skin; there are no special blood tests or diagnostic procedures.[2] Usually a skin biopsy, or scraping, may be needed to rule out other disorders and to confirm the diagnosis. Biopsy from the skin will show clubbed rete pegs if positive for psoriasis. Another sign of psoriasis is Auspitz's sign that when the plaques are scraped, one can see pinpoint bleeding from the skin below .

Psoriasis is their severe form affects quality of life is the affected patients like any other chronic illness like hypertension, type 2 diabetes and depression[3].

National Psoriasis Foundation survey of 426 psoriasis patients, 71 percent reported the disease was a significant problem in everyday life [4]. It is now thought to be a systemic disease with health implications beyond the skin manifestations.[5]

The Metabolic syndrome which is characterized by obesity, hypertension, dyslipidemia and impaired glucose tolerance [7].

The different analysis have suggested that psoriatic patients have an increased risk of myocardial infarction, stroke, vascular inflammation and atherosclerotic conditions independent of traditional risk factors for cardiovascular disease.[6] Correlation between psoriasis and Metabolic Syndrome and the effect it has on the patient's health and on the efficacy and safety of treatment options, it is important that patients undergo appropriate screening as part of routine medical care [8, 9].

This disease is a common, chronic relapsing skin disease. Some endocrinological disturbances exacerbate the disease. Psoriasis is an early sign of hypothyroidism and sometimes associated with more severe form of hypothyroidism [10].

AIMS AND OBJECTIVES

AIM:

1. To study the relationship between psoriasis and metabolic syndrome in psoriatic patients by performing lipid profile and plasma glucose levels.

2. To study the correlation of psoriasis with Thyroid dysfunction by estimating FT3, FT4 and TSH in psoriatic patients.

OBJECTIVES

1) To study the prevalence of metabolic syndrome in different types of Psoriasis.

2) To study the prevalence of Thyroid dysfunction among Psoriatic patients.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

1. Farber et al 1977; Identified in childhood psoriasis the familial incidence is greater when compared to adult onset psoriasis [11].

2. **Krueger et al 1984;** The hyperproliferation of epidermis , terminal differentiation are the fundamental abnormalities in psoriatic skin, there is an inflammatory process involving cytokines, chemokines, antigen-presenting cells .

3. **Ettehadi et al 1994;** The Tumour Necrosis Factor -alpha appears to be a critical cytokine in the Psoriasis pathogenesis, where it is crucial for keratinocytes hyperproliferation, endothelial cell regulation, and function of memory T cells

4. Lakka et al in 2002; Systemic inflammation which is correlated with metabolic syndrome, with proinflammatory cytokines such as tumor necrosis factor α and C reactive protein levels being elevated compared to those without metabolic syndrome[12].

5. Feldman et al 2004; Showed the psoriatic severity namely mild psoriasis, moderate, and severe based on affected body surface area (BSA) (<3% , 3-10% and 10%) [13] respectively.

6. **Gordon and Ruderman 2006;** Demonstrated that inflammatory mediator levels are increased in psoriatic lesions, when compared with normal skin of non psoriatic individuals.

7. Sommer et al in 2006; Showed that an increased frequency of metabolic syndrome and it's components amongst subjects with psoriasis [14].

8. Ludwig et al in 2007; studies showed Increased frequency of metabolic syndrome in psoriasis leading in turn to risk of cardiovascular disease and increasing mortality rate[15].

9. Gisondi et al in 2007; Demonstrated that the association would not to be related to age, sex or the kind of psoriasis [16].

10. Sterry et al in 2007; Identified that tobacco addiction, obesity, physical activity, depression, poor food habits and psychological stress responsible for the Metabolic Syndrome and increasing risk of coronary artery disease[17].

11. Setty et al in 2007; Found that healthy food could have beneficial effects on psoriasis and shows the strong associations among obesity, weight gain, and psoriasis [18].

12. Rakesh et al 2008; Analysis concluded that psoriatic patients self – conscious inconvenienced by the shedding of the skin live in a fear of relapse and avoid social interactions[19].

13. Gelfand et al in 2009; suggested that psoriasis is linked with an increased frequency of adverse outcomes like myocardial infarction, stroke and cardiovascular death [20].

14. Mehta et al in 2009; Identified that, severe psoriasis can be a risk factor for atherosclerotic disease [21].

15. Abuabara et al in 2010; Identified with severe psoriatic patients die about 5 years younger when compared to without psoriatic groups , cardiovascular death which is the most common cause of excess mortality in these patients [22].

16. Prey in 2010; There is some dispute, regard to its relationship to the severity and duration of the disease [23].

17. Mebazaa et al in 2010; Found prevalence of metabolic syndrome increased in females with psoriasis [24].

18. Zindancy I et alin 2012; Demonstrated that Metabolic Syndrome which is more among women than men owing to higher BMI and Waist circumference [25].

19. Shapiro et al in 2012; Authors suggested that the Metabolic Syndrome in psoriasis due to chronic presence of systemic inflammation, certain pro inflammatory cytokines and immunological mediators [26].

20. Ozer African et al in 2004; Concluded that the PASI (Psoriasis Area Severity Index) scores were higher among psoriatic patients caused by the direct or indirect effects of thyroid hormones [27].

21. Shraddha Madanagobalane et al in 2012; Suggested that Metabolic syndrome is more frequent in patients with psoriasis and they also found that no relationship between disease severity and presence of metabolic syndrome [28].

22. Sristi Lakshmi et al in 2014; Concluded that there is no close relation between psoriasis and metabolic syndrome in south Indian patients [29].

One of the common, chronic disfiguring inflammatory and proliferative condition of the skin is psoriasis, both genetic and environmental factors can influence a major role. Psoriasis is in duration, periodicity of flares and extent morphological variants are common[30].

INCIDENCE AND PREVALENCE:

In China, psoriasis is essential to affect the population, but the disease is very rare or nonexistent in Inuits, Samoans or Latin American Indians. It is very common in East than West Africa. Climate also appears to affect psoriasis.

AGE OF ONSET:

Lomholt's reported age of onset of Psoriasis in childhood in his study in the Faroe Islands**.** In a US study the average age of onset was 28years, the analysis reported in China, the mean age of onset was 36 years.

SEX EFFECTS:

Psoriasis equally affects males and females. The variety of analysis indicated that age of onset of Psoriasis is younger in females. One Indian survey Sristi Lakshmi et al in 2014 reported that proportion of metabolic syndrome was significantly higher in females than males [29.48,49].

ETIOLOGY:

GENETIC EPIDEMIOLOGY:

Many evidence suggested, psoriasis which has an important genetic component. Lomholt's classic epidemiological study of psoriasis in Foroe Island in 1963 showed that Psoriatic prevalence was greater in first and second degree relatives of sufferers.

ENVIRONMENTAL RISK FACTORS:

Environmental factors linked to psoriasis for both the disease initiating process and exacerbation of pre-existing disease.

TRAUMA:

Physical, chemical, electrical, surgical, inflammatory & infective insults recognised to induce lesions of psoriasis called as Koebner's phenomenon**.**

INFECTION:

Streptococcal infection is important in chronic plaque psoriasis**.**

DRUGS:

Many reports suggested that, antimalarials drugs, beta blockers , angiotensin–converting enzyme inhibitors , non steroidal anti inflammatory drugs (NSAIDS), and withdrawal of corticosteroids, these drugs are favorable for the onset and exacerbation of psoriasis

SUNLIGHT:

Recent work indicated that severely photosensitive psoriasis is predominantly female, distinct from polymorphic light eruption and strongly associated with HLA-CW6, family history and early age of onset.

PSYCHOGENIC FACTORS:

Several studies suggested that the impact of psoriasis on physical and mental components of the quality of life index similar to other major diseases including heart disease and arthritis

SMOKING AND ALCOHOL:

Both smoking and alcohol have detrimental effect on psoriasis.

HIV AND AIDS:

The relation between severe psoriasis, psoriatic arthropathy and HIV infection is recognised.

CARDINAL CHARACTERISTICS OF PSORIATIC LESIONS:

1. Epidermal hyperproliferation with loss of differentiation,
2. Dilatation and proliferation of dermal blood vessels,
3. Accumulation of inflammatory cells.

FACTORS ALTERED IN PSORIATIC SKIN:

1. The Growth factors
2. The Cytokines
3. The Inflammatory mediators
4. Other biological markers.

PATHOGENETIC MECHANISMS:

EPIDERMAL PROLIFERATION:

Proliferation of keratinocytes illustrated in psoriasis because of an increase in the proliferating cell component in the basal levels of the epidermis, and not due to shortened cell cycle time. Multiple growth factors are the mediator of these events [37].

VASCULAR CHANGES:

Epidermal keratinocytes are the primary sources in angiogenic activity and dermal capillaries accelerate the inflammatory process through the expression of molecules involved in leukocyte homing, stimulated by inflammatory mediators.

MOLECULAR GENETICS:

PSORS 1 is a genetic determinant of the psoriasis, which probably accounts for 30-50% of the heritability of the disease and has been replicated in all linkage studies. PSORS 1 is located within the major histo compatability complex (MHC) on chromosome 6p.Guttate psoriasis is strongly PSORS 1 associated, whereas palmoplantar pustulosis and late onset (> 50 years of age) psoriasis vulgaris are not associated.

Most of the studies published in western populations with only little information about Indians [31]. Chablani et al in 1992 in a study of 67 psoriasis patient from western India found association with the A1, B17 and Cw6, but not with B13 antigens [32].

Pitchappan et al, in 1989 reported association of HLA Bw57 and DR7 with psoriasis vulgaris in south India [33]. Rani et al, in 1998 showed that Cw FN

X010602 was the main allele that had high frequency in psoriasis patients in India[38] .Indian studies reported lower familial incidence of the disease [34]. Bedi at al in 1995 reported positive family history of psoriasis in 14% of their patients [35, 39]. While Kaur et al in 1997 reported family history in only 2% of their patients [36, 40].

INFLAMMATION AND IMMUNOLOGY:

Most commonly the T lymphocytes which play a major role in development of plaques of psoriasis. This includes.

1. The Early influx of T cells expanding lesions
2. Strong association with the MHC, particularly HLA –CW6
3. Ablative action of anti-T cell therapy.2.Increased antigen presentation in psoriatic plaques.
4. Anecdotel of development of psoriasis in syngeneic bone marrow transplant.

KOEBNER AND REVERSE KOEBNER PHENOMENA:

The koebner phenomena usually occurs 7-14 days after injury.

CLINICAL FEATURES:

Acute Guttate attack of psoriasis occurs in childhood. The Most common form of psoriasis occurs before fourth decade of life. The dorsal tongue exhibit geographic, annular white patches. Nail changes include 'oil spots'nail pitting, distal oncholysis and accumulation of subungual debris [30].Thirty percent or more psoriatic patients have inflammatory arthritis, commonly presents as an

asymmetric oligoarthritis affecting distal or proximal interphalangeal joints. Psoriatic patients appear to be an increased risk for developing obesity, diabetes mellitus, hyperlipidemia, hypertension and cardiovascular disease [41, 48, 49] .

CLINICAL VARIANTS:

1. PLAQUE TYPE

2. GUTTATE TYPE

3. PUSTULAR TYPE

4. ERYTHRODERMIC TYPE

1. PLAQUE PSORIASIS:

It is well demarcated, erythematous plaques with an adherent, silver to white – colored scale.

AUSPIT'Z SIGN: The pinpoint bleeding on the skin may be seen while the adherent scales are removed.

WORONOFF'S RING: Plaques have surrounding hypopigmentation called as WORONOFF'S RING.

GUTTATE PSORIASIS [30]:

It occurs more commonly in young adults, and it presents with multiple small, 'drop shaped 'erythematous scaly plaques diffusely on the body, most frequently on the trunk.

PUSTULAR PSORIASIS:

It is characterized by superficial pustules .It may be localized on the palms and soles like palmoplantar pustulosis or may be generalized.

ERYTHRODERMIC VARIANT:

It is characterized by diffuse erythroderma with fine scaling.

DIAGNOSIS:

The psoriatic diagnosis is usually based on clinical findings. Evidence are supporting the diagnosis includes typical morphology and anatomic locations of the skin lesions, presence of nail lesions or arthritis and a positive family history of psoriasis .Skin biopsy useful in atypical cases.

PSORIASIS WITH METABOILC SYNDROME:

Variety of surveys have reported that elevated level of serum immunological markers, like Interleukin -6, Interleukin-2 receptor, TNF-alpha and ICAM-1, in psoriasis being a systemic immunologic disorder [54,55,57,].

METABOLIC SYNDROME:

The other names are Insulin Resistance Syndrome, Syndrome X, which consists of metabolic abnormalities with increased risk of coronary heart disease and diabetes mellitus.

NCEP: ATP 2001 CRITEARIA FOR METABOLIC SYNDROME:

1. Fasting Plasma glucose: is ≥ 100mg/dl or in specific medication or previously diagnosed Type 2 diabetes mellitus.

2. Hypertension: Blood Pressure is ≥ 130 mmHg of systolic or ≥ 85 mmHg of diastolic or in specific medication.

3. Hypertriglyceridemia: Triglyceride level is ≥ 150 mg/dl or with specific medication

4. Low HDL –c: is <40 mg/dl in males, <50mg/dl in females or with specific medication.

5. Obesity mainly central: Waist Circumference is > 102cm in males, >88 cm in Females.

EPIDEMIOLOGY [42]: Worldwide the metabolic syndrome is in Native Americans, 60% of females age 45-49 & 45% of male's age 45-49 years. The rising prevalence in children and severity of obesity is initiating features of the metabolic syndrome in a younger ages.

RISK FACTORS:

OVERWEIGHT/OBESITY:

Body mass is positively related to fasting triglycerides concentrations, plasma cholesterol and blood pressure, inversely related to HDL –cholesterol. Waist circumference is a significantly better index of insulin resistance than Waist / hip ratio or BMI. A cut of value for waist circumference of < 100cm rules out insulin resistance in both male and female with optimal sensitivity and specificity. Body mass index of > 30 Kg/m^2 is considered as "obesity" and it plays a major role in atherosclerotic progression **[43, 44].**

STRESS:

Prolonged stress which is an underlying cause of metabolic syndrome by the hormonal balance of the hypothalamic – pituitary – adrenal axis.(HPA-AXIS).A dysfunctional HPA-axis causes high cortisol levels , which causes rising glucose and insulin levels, which in turn results insulin mediated effects on adipose tissue, promoting visceral obesity , insulin resistance , dyslipidemia and hypertension , with direct effects on bone , causing " low turnover " osteoporosis [45].

SEDENTARY LIFESTYLE:

MS components are associated with a sedentary lifestyle, included increased plasma glucose, high blood pressure increased triglyceride, reduced HDL cholesterol and increased adipose tissue.

AGING:

In US study 44 percentages of MS affected people age were older than 50 years. In this study an increased percentage of women have the MS than men over age 50 [42].

TYPE 2 DIABETES MELLITUS [42]:

Base on both the National Cholesterol Education Programme and International Diabetes Foundation definitions of the Metabolic Syndrome Type 2 diabetes mellitus is included. In MS the risk of type 2 diabetes mellitus is increased three to five fold.

HYPERINSULINEMIA AND INSULIN RESISTANCE:

Insulin resistance refers to the decreased rate of glucose uptake mediated by insulin .It was shown to be accompanied by increased levels of insulin and undesirable changes in cardiovascular risk factors like high levels of triglycerides , decreased HDL cholesterol , and development of hypertension. In addition adipose tissue is now recognised to be a source of a number of inflammatory cytokines (interleukin -6), tumour necrosis factor α (TNF- α), and growth factors (Heparin binding epidermal growth factor (HB-EGF) hormone like substances (leptin, adiponectin, resistin) [46].

HYPERTENSION:

Cardiovascular risk increases both with increasing systolic and diastolic pressure cholesterol is a continuous variable like blood pressure and there is no comprehensible cut –off value doubles the risk of CHD at any given concentration of cholesterol.

HYPER TRIGLYCERIDEMIA:

Hypertriglyceridemia state promotes the oxidative and proinflammatory milieu enhancing expression of adhesion molecule formation of foam cell and intoxication of smooth muscle cell. Following hydrolysis, chylomicrons which are exogenously derived, VLDL cholesterol secreted endogenously enriched remnant by products enters the endothelial space. Hypertriglyceridemia increases reverse cholesterol transport that 10% lowering of TGL concentration decreases the risk of CHD by 23% [47,56] .

CHD (CORONARY HEART DISEASE):

The Metabolic Syndrome components are increased risk for coronary heart disease [50, 51].

LIPODYSTROPHY:

Both congenital and acquired lipodystrophy can give rise to insulin resistance and other components of the MS.

PATHOPHYSIOLOGY OF METABOLIC SYNDROME:

Which is the most common for there a development of visceral fat and which the adipocytes that can increases the plasma TNF alpha level and alter the level of adiponectin , resistin .Chronic inflammation contributes to an increased risk of hypertension, atherosclerosis and diabetes .

CLINICAL FEATURES:

SYMPTOMS AND SIGNS:

The metabolic syndrome is typically not associated with symptoms. On physical examination presence of elevated blood pressure and expanded waist circumference and other biochemical abnormalities including increased triglycerides, decreases HDL cholesterol and increased fasting blood sugar.

OTHER ASSOCIATED CONDITIONS:

Those alterations include increased level of apo B , apo C-III, Plasminogen activator I, Fibrinogen level , Homocysteine level , Asymmetric level of

dimethylarginine, increased white blood cell count, increased level of Inflammatory Cytokines, raised C-Reactive Protein level, Microalbuminuria , Hyperuricemia, Non-Alcoholic Fatty Liver Disease , Poly Cystic Ovarian Disease and Obstructive Sleep Apnea [52,53] .

DIAGNOSIS:

Blood pressure and waist circumference measurements provide information necessary for the diagnosis.

LABORATORY TESTS:

Fasting lipid profile and blood glucose are needed to determine the metabolic syndrome. Measurements of other biomarkers associate with insulin resistance, included, Plasma fibrinogen, hsC- Reactive Protein, urinary microalbumin, serum uric acid and liver function tests.

THYROID DYSFUNCTION IN PSORIASIS:

Psoriasis can be exacerbated by endocrinological disturbances especially due to thyroid hormones. The thyroid hormones T3 and T4 increase in leads to epidermal hyperplasia [27]**.** Antithyroid hormonal drugs like propylthiouracil, anti thyroid preparation, elevated number of cytotoxic T cells, suppressor cells, and lowered level of lymphocytes in psoriatic plaque type. Receptors of Triiodothyronine can play a major role in the keratin synthesis, an anti thyroid drug like Propylthiouracil, which may affects the synthetic process of keratin by binding with Triiodothyronine nuclear receptors.

Thyroid hormones have hyper proliferative effect on the skin by EGF. The skin is most important tissue for thyroid hormones like Triiodothyronine and Tetraiodothyronine and these hormones can increase the level of EGF (Epidermal growth factor) that accelerate the proliferation of epidermis. When thyroid function is low, prolactin increases with psoriasis .Prolactin increases cell division and sebum formation whereas darkness and stress increase it .This may be the connection between sunlight and the alleviation of psoriasis[27].

To our knowledge there are few studies in India on Psoriasis related with Metabolic Syndrome and thyroid dysfunction. Hence it is proposed to study the recent prevalence of metabolic syndrome in psoriatic patients. It is also proposed to find out the association of thyroid dysfunction in psoriatic Patients.

MATERIALS AND METHODS

MATERIALS AND METHODS

STUDY DESIGN **:** Case and control study

PLACE OF STUDY **:** Department of Bio Chemistry

The CMCH & RC (Chennai Medical College Hospital and Research Centre), Trichy.

PERIOD OF STUDY: JANUARY 2014-FEBRAUARY 2015

SAMPLE SIZE **:** 100 Cases and 30 Controls.

(Cases- psoriatic patients) (Controls – non psoriatic patients)

AGE **:** 20-80 years

SEX **:** Both females and males.

GEOGRAPHICAL DISTRIBUTION: Both urban and rural areas.

ETHICAL CONSIDERATIONS

The necessary approval was obtained to conduct the study from the CMCH & RC, ethical committee, Trichy. Patients were given an explanation about the purpose of the study and informed written consent was obtained, confidentiality about their results was assured. Their participation was optional.

SELECTION OF CASES AND CONTROLS:

Hundred psoriatic patients in the age group of 20-80 years in CMCH & RC, Irungalur , Trichy, and 30 non psoriatic patients other than psoriasis who were all attending in Dermatology department ,in the same age group as control were

selected for the study. All the patients were included as cases evaluated and diagnosed as psoriasis on the basis of history, clinical findings and skin biopsy.

CASES:

Inclusion Criteria:

1. Patients with psoriasis (age groups 20-80 years)
2. psoriatic Patients with metabolic syndrome
3. Psoriatic arthritic patients

Exclusion Criteria: 1. Psoriatic patients < 20 years.

2. Psoriatic patients with associated chronic autoimmune disorders like Systemic Lupus Erythematous, Rheumatoid arthritis, Asthma.

CONTROLS:

Inclusion Criteria: Patients attending the Dermatology Department suffering from skin diseases other than psoriasis.

Exclusion Criteria: Non psoriatic patients with chronic autoimmune disorders like Systemic Lupus Erythematous, Rheumatoid arthritis, Asthma.

STUDY PROTOCOL:

Informed consent obtaining from the subjects who were all included for study and patients were subjected to history taking and the clinical examination.

DETAILED HISTORY:

A detailed history was elicited for

- Duration of the disease
- Severity and Symptoms of disorder
- Arthritic pain

- Smoking habits, consumption of alcohol details and diet habits .
- Co-morbid diseases and concomitant drugs intake.
- Native treatment.
- Treatment before hospitalization.

CLINICAL EXAMINATION:

A thorough physical examination was done to look for local and systemic features. Psoriatic involvement was assessed using Body Surface Area (BSA) [30].

The national psoriasis foundation defines mild, moderate and severe psoriasis

MILD **:** If affected upto 3 percentage of the body, especially in isolated patches on the elbows, hands, knees feet and scalp. This can be controlled by topical therapy.

MODERATE TYPE **:** In this type the body's surface affected from 3percentage – 10percentage, especially on the scalp, arms, legs, torso and other areas. Treatments for this type are topical agents, phototherapy, systemic medications may be given.

SEVERE PSORIASIS **:** It affects the body surface more than 10%. It may be extensive with plaques, pustules or erythroderma. Treatment for this type are Phototherapy, systemic medications, or a combination of these, with or without a topical agent , are necessary to achieve adequate results.

ANTHROPOMETRIC MEASUREMENTS:

Height in cm, weight in kg, waist circumference in cm and blood pressure in mmHg measurements were done.

1. Based on weight and height calculations the Body Mass Index (BMI) was determined by using the following equation

BMI = Weight in Kilogram / height in meters Square

According to rule of India, a BMI from 23 to 24.9 is overweight, a Body Mass Index is above or equal to 25 is moderate obesity and a Body Mass Index is above or equal to 30 in severe type of obesity.

2. Measurement of waist circumference by the measuring tape which placed at the level of the iliac crest snugly around the abdomen.
3. More than ninety cm waist circumference in men and above 80 cm for women was considered as obesity.
4. The average of two measurements of blood pressure was recorded in right arm and was taken in the sitting posture
5. **INVESTIGATIONS:**

1. Complete Blood Count
2. Fasting blood glucose
3. Fasting plasma lipid (TC, TGL, LDL-c, HDL-c)
4. Serum Thyroid profile.
5. LFT (S. AST,ALT,GGT, Bilirubin levels were also done)

COLLECTION OF SPECIMENS

Informed consent was obtained for each patient and control groups prior to the study. 5ml of venous blood samples were collected in clot activator coated polypropylene tubes by venue puncture under strict aseptic precaution as soon as the subjects got admitted as per the inclusion

criteria. Serum was separated by centrifugation for 10 minutes at 3500 rpm. 8-12 hours fasting samples were collected from all subjects during their hospital stay and analysis of total cholesterol, triacylglycerol and high density lipoprotein were done.

SAMPLE STORAGE:

The specimens were freezed at -20°C for storage until analysis.

The separated serum was analysed for the following tests:

1. **Fasting Blood Glucose**
2. **2 . Serum lipid profile**

i. Serum Total Cholesterol

ii. Serum Triglycerides

iii. Serum VLDL-c

iv. Serum LDL–c

v. Serum HDL–c

3. Thyroid profile

i. Free Triiodothyronine (T3)

ii. Free Thyroxine (T4)

iii. Thyroid Stimulating Hormone (TSH)

iv.

QUANTITATIVE ESTIMATION OF FASTING BLOOD GLUCOSE:

METHODOLOGY:

GLUCOSE OXIDASE – PEROXIDASE METHOD (**END POINT METHOD**)

PRINCIPLE:

In serum /plasma glucose is oxidized by glucose oxidase (GOD) enzyme and to produce gluconic acid with the liberation of hydrogen peroxide then it is converted to water and nascent oxygen due to peroxidase (POD) enzyme.

An oxygen acceptor 4- Aminoantipyrine, which takes up the oxygen and together with phenol forms a chromogen (pink colored) then it, can be measured at 505 nm.

Glucose + $o_{2\ (nascent\ oxygen)}$ + H_2O $\xrightarrow{GOD}$ Gluconic acid + H_2O_2

H_2O_2 + phenol + 4-Aminoantipyrine $\xrightarrow{POD}$ quinoneimine complex (Red)+ H_2O

GLUCOSE REAGENTS:

1. Phosphate buffer (Ph 7.5) : 0.1 mol/L
2. 4-Aminoantipyrine : 5.0 mmol/L
3. Peroxidase : >1.5 KU/L
4. Glucose Oxidase : >15 KU/L
5. Phenol : 5.0 mmol/L

Glucose Standard (concentration: 100 mg /dl)

ASSAY PROCEDURE: (FULLY AUTOMATED ANALYZER)

Pipette into test tubes and labelled them as Blank (B), Standard (S) and (T) as follows:

S.NO	REAGENT	BLANK	STANDARD	TEST
1.	reagents of GLUCOSE	1.0 ml	1.0 ml	1.0 ml
2.	GLUCOSE Std	_	10 µl	_
3.	SPECIMEN	_	_	10 µl

Reaction temperature at 37◦C.

Mixed well then the absorbance of Standard (S) and Test (T) against Blank (B) read at 505 nm or with green filter (500- 540 nm) .

CALCULATION OF RESULTS

$$\text{Glucose conc (mg/dl)} = \frac{\Delta \text{ Abs for Test}}{\Delta \text{ Abs for Standard}} \times 100$$

Storage:

The reagents were stored at 2-8◦c.

Reference value:

FBS: 70- 100 mille gram/dl

PPBS : < 140 mille gram /dl

LIPID PROFILE

QUANTITATIVE ESTIMATION OF SERUM

TOTAL CHOLESTEROL

METHODOLOGY:

Cholesterol oxidase / peroxidase

PRINCIPLE:

Cholesterol esters are hydrolyzed to produce cholesterol. Then, free cholesterol takes part in two coupled reactions that permit to measure cholesterol photometrically.

The reaction sequence is as follows:

c- ester + H_2O $\xrightarrow{\text{Cholesterase}}$ Cholesterol + FA

Cholesterol + ½ O2 + H2O $\xrightarrow{\text{CHOD}}$ Cholestenone + hydrogen peroxide

2H2O2 + 4- AMAT + Phenol $\xrightarrow{\text{Peroxidase}}$ chromogen (Quinoneimine) +4H2O

Cholesterol Reagent:

Pipes	: 35 mmol / liter
Sodium cholate	: o.5 mmol / liter
Phenol	: 28mmol/ liter
Cholesterol esterase	: > 0.2 Units/ mille liter
Cholesterol oxidase	: >0.1U/mille liter
POD	: more than 0.8U/mille liter
4- AMAT	: 0.5mmol/liter
pH	: 7.0

Std (5ml): TC 200mg/dl

The reagents were stored at 2ºC-8ºC

PREPARATION OF WORKING SOLUTION:

The reagents are allowed to attain room temperature.

PROCEDURE:

The sample and working solution are brought to room temperature prior to use. 3 test tubes named as blank (B), Standard (S), Test (T). 1 ml of working reagent added in 3 test tubes then 10µL of sample was added in'T' and 10µL of standard was added in 'S'. After mixing then incubated in room temperature for 10 minutes.

	Blank	**Standard**	**Test**
Distilled water	**10µL**	**--**	**--**
Reagent	**1mL**	**1mL**	**1ml**
Standard	**--**	**10µL**	**--**
Sample	**--**	**--**	**10µL**

CALCULATIONS:

$$\frac{\text{Sample absorbance}}{\text{Standard absorbance}} \times 200 = \text{Sample concentration (mg/dl)}$$

LINEARITY:

This method is linear upto 1000mg/dl.

REFERENCE VALUES:

Serum TC :(mg/dl)

Desirable value : up to 200

Borderline High value: 200 - 239

High value : > 240

QUANTITATIVE ESTIMATION OF SERUM TRIGLYCERIDES

METHODOLOGY:

Glycerol-3- phosphate oxidase (GPO)

PRINCIPLE OF THE METHOD:

The estimation of triglycerides by the enzyme of lipoprotein lipase. Quinoneimine is an Indicator which is generated by hydrogen peroxide under the catalytic action of peroxidase from 4-aminoantipyrine and 4- Chlorophenol.

The reaction sequence:

$$\text{Triglycerides} \xrightarrow{\text{Lipoprotein lipase}} \text{Glycerol + FA}$$

$$\text{Glycerol + Adenosinetriphosphate} \xrightarrow{\text{GK}} \text{Glycerol -3-phosphate + ADP}$$

$$\text{Glycerol -3-phosphate} + O_2 \xrightarrow{\text{GPO}} \text{DHAP + Hydrogen peroxide}$$

$$2H_2O_2 + \text{AMAT} + \text{4- Chlorophenol} \xrightarrow{\text{Peroxidase}} \text{Quinoeimine + HCl + } H_2O$$

REAGENTS:

4-chlorophenol	: 4 mille mol / Liter
ATP	: 2 mmol / Liter
Mg^{2+}	: 15mmol/Liter
Glycerolkinase	: ≥0.4 kU/ Liter
Lipoprotein lipase	: ≥ 2 kU/ Liter
Peroxidase	: ≥ 2 kU/Liter
4-Aminoantipyrine	: 0.5mmol/l
Glycerol -3- phosphate – oxidase	: ≥0.5 kU /L
Good's buffer pH 7.2	:50 mmol / l

Standard: Triglycerides 200mg/dl

The reagents were stored at 2ºC-8ºC

PREPARATION OF WORKING SOLUTION:

The reagents are allowed to attain room temperature.

PROCEDURE:

The sample and the working solution were brought to room temperature prior to use. 3 test tubes labeled like B, S, T. working reagent 1 ml added in all 3 tubes. 10micro Liter of sample was added in 'T' tube and standard 10µL was added in 'S'tube. After it mixed and incubated the tubes at room temperature for 10 minutes.

	Blank	**Standard**	**Test**
Distilled water	**10µl**	**--**	**--**
Reagent	**1ml**	**1ml**	**1ml**
Standard	**--**	**10µl**	**--**
Sample	**--**	**--**	**10µl**

CALCULATIONS:

$$\frac{\text{Sample absorbance}}{\text{Standard absorbance}} \times 200 = \text{Sample concentration (mg/dl)}$$

To correct for free glycerol, subtract 10mg/dl from the triglycerides value calculated above.

LINEARITY:

This method is to determine triglyceride concentration within a measuring range from 2-1000mg/dl.

REFERENCE VALUES:

Serum TGL: (mille gram/dl)

Normal : < 150

High : 150 – 199

Hypertriglyceridemia: 200-499

Very High : > 499

QUANTITATIVE ESTIMATION OF SERUM

LOW DENSITY LIPOPROTEIN CHOLESTEROL

METHODOLOGY:

Direct enzymatic method

PRINCIPLE OF THE METHOD:

LDL is selectively protected while non-LDL-lipoprotein are processed by enzyme, then LDL is released and LDL-cholesterol determined in a color producing due to enzymatic reaction.

Reaction sequence:

1. Low Density Lipoprotein + 1.reagnt ⟶ Protected LDL

   Chylomicrons , HDL, VLDL, $\xrightarrow{\text{CHE \&CHO}}$ Cholestenone +hydrogen peroxide

   $H2O2 \xrightarrow{\text{Catalase}} H_2O$

2. Protected LDL + 2. reagnt ⟶ LDL

   $\text{LDL-c} \xrightarrow{\text{CHE \&CHO}} \text{Cholestenone} + H_2O_2$

   $H2O2 + \text{4- AMAT} + \text{H-DAOS} \xrightarrow{\text{POD}} \text{Color}$

REAGENTS:

Reagent 1:

Cholesterol esterase	: ≥2.5 kU/L
Cholesterol oxidase	: ≥ 2.5 k UNITS/L
(H-DAOS)	: 0.5mmol/Liter
Buffer's pH 6.8	: 20 mille mol / Liter

Catalase : ≥500 kU/ Liter

pH of Good's buffer :7.0 :25 mille mol / Liter

Peroxidase : ≥15 kU/ Liter

4-Aminoantipyrine : 3.4mmol/ Liter

Calibrator: LDL- Cholesterol 132 mg/dL

The reagents were stored at 2ºC-8ºC

PREPARATION OF WORKING SOLUTION:

The reagents are allowed to attain room temperature.

PROCEDURE:

The sample and the working solution were brought to room temperature prior to use. Three test tubes named them as B, C, T. 280 µL of working reagent 1 added in all 3 tubes. 3.0µL of sample added in 'T' tube and 3.0 µL of calibrator added to 'C' tube. After mixed and tubes were incubated at room temperature for 5 minutes. Read the absorbance A_1, then 70 µL of working reagent 2 is added to 3 test tubes. It mixed and incubated the tubes for 5 minutes at room temperature. Read the absorbance A_2.

	BLA NK (µl)	CALIBRATOR (µl)	TEST (µl)
CALIBRATOR	_	**3.0**	**-**
SAMPLE	**-**	**-**	**3.0**
DISTILLED WATER	**3.0**	_	
REAGENT 1	**280**	**280**	**280**
Mix ,incubate in 37◦C for 5min,, read the absorbance (A1), then add:			
Reag: 2	**70**	**70**	**70**

Mixed well and incubated 5 min, in 37◦C, read absorbed (A2).

$\Delta A = [(A_2 - A_1)$ S or C] - [(A2-A1)B]

CALCULATIONS:

$$\frac{\Delta A \text{ Sample}}{\Delta A \text{ calibrator}} \times \text{conc .calib} = \text{Sample concentration(mg/dl)}$$

LINEARITY:

This method is to determine LDL-C concentration within a measuring range from 1- 400mg/dl.

REFERENCE VALUES: Serum LDL-c: (mg/dl)

Optimal : < 100

Near/above optimal: 100-129

Borderline High : 130- 159

High : 160- 189

Very high : >189

QUANTITATIVE ESTIMATION OF SERUM

HIGH DENSITY LIPOPROTEIN CHOLESTEROL

METHODOLOGY:

Direct enzymatic method

PRINCIPLE OF THE METHOD:

Human lipoproteins are used to form antigen-antibody complexes with chylomicrons, VLDL and LDL and that only HDL-c is determined by an enzymatic measurement of cholest.

The reaction sequence is as follows:

$$\text{LDL,VLDL, Chylomicrons} \xrightarrow{\text{Anti-human } \beta \text{ – Lipoprotein antibodies}} \text{Antigen – antibody complexes + HDL}$$

$$\text{HDL –c} + H_2O + O_2 \xrightarrow{\text{CHE \&Oxidase}} \text{Cholest-4-en-3-one + Fatty acid +} \; H_2O_2$$

$$H_2O_2 + \text{F- DAOS + 4- Aminoantipyrine} \xrightarrow{\text{POD}} \text{Blue colored Complex +} \; H_2O$$

Reagent 1:

The Ascorbate oxidase : 2, 250 Units / Liter

The Anti-human β - lipoprotein

Peroxidase : 2, 000 U/L

4-Aminoantipyrine : 0.75mmol/l

Buffer's pH 7.0 : 25 mmol / l

Reagent 2:

Buffer's pH	7.0	: 30 mille mol / liter
CHE		: 4000 Units /Liter
CHO		: 20000Units/Liter
(F-DAOS)		:0.8mmol/Liter

Calibrator: HDL-Cholesterol : 50.6 mg/dl

The reagents were stored at 2ºC-8ºC

PREPARATION OF WORKING SOLUTION:

The reagents were allowed to attain room temperature.

PROCEDURE:

The sample and the working solution were brought to room temperature prior to use. Three Test Tubes named as B, C, T. 240 µl of working reagent 1 is added to 3 test tubes. 2.4µl of sample was added to test tube labeled 'T' and 2.4 µl of calibrator is added to test tube labeled 'C'. After mixing tubes were incubated 5 minutes in room temperature then read absorbance A_1, then 60 µl of working reagent 2 was added to 3 tubes. After mixing incubated the tubes 5 minutes in room temperature then read the absorbance A_2.

	BLANK µl	CALIBRATOR µl	TEST µl
CALIBRATOR	_	**2.4**	**-**
SAMPLE	**-**	**-**	**2.4**
DISTILLED WATER	**2.4**	_	
REAGENT 1	**240**	**240**	**240**
Mix ,incubate 5min, in 37◦C then read absorbance (A1), and add:			
REAG: 2	**60**	**60**	**60**

$\Delta A = (A_2 - A_1)$ sample or calibrator.

CALCULATIONS:

$\frac{\Delta A \text{ Sample}}{\Delta A \text{ Calibrator}}$ × conc. Calib = Sample concentration (mg/dl)

LINEARITY:

This method is to estimate HDL-c concentration within a range of 1-180mg/deci liter.

REFERENCE VALUES:

Serum HDL-C: Males – 30-60 mg/dl

Females – 35-75 mg/dl

ESTIMATION OF THYROID PROFILE [58]:

ESTIMATION OF FT3 (FREE T3)

METHODOLOGY:

THE ELISA METHOD

PRINCIPLE:

Based on the competitive binding between FT3 in a test specimen and T3 –Peroxidase conjugate in a limited number of binding sites on the well coated with anti –T3 (Sheep) . The amount of T3 –Peroxidase conjugate bound to the well which is inversely proportional to the FT3concentration in the specimen.

Then the specimen was incubated and T3 –Peroxidase conjugate unbound enzyme conjugate is removed in the equilibrium state by washing procedure. Then TMB /Substrate solution added after that a blue color develops. The intensity of this blue color , then changes to yellow after stopping the reaction , this color is inversely proportional to the FT3 amount in the specimen.

Calibrator's absorbance and specimen is estimated by using ELISA microplate readers or automated ELISA systems (eg. HUMAN'S Huma - Reader or ELISYS line) . Specimen's concentration is extrapolated from a dose response curve generated by utilising serum calibrators of known concentration of antigen.

KIT COMPONENTS:

1. **STRIPS**

   8 - Well strips, coated by anti –T3 sheep)

2. **CALIBRATORS: (6 C , 2 ml per cal) ready for use, in human serum**

FT3 CONCNTRATION	Pg/ml
Calibrator 1	O (A)
Calibrator 2	1 (B)
Calibrator 3	3 (C)
Calibrator 4	5 (D)
Calibrator 5	8 (E)
Calibrator 6	16 (F)

3. ENZYME – ANTIGEN CONJUGATE: (13 ml)

Ready for use , coloured red T3 – HRP Conjugate in a protein stabilising matrix. - 1%.

4. WASH SOLUTION : (20 ml)

Concentrated of ca.1000ml

Buffered saline. - 250 mmol/l

5. SUBSTRATE :(in 14ml)

3, 3', 5,5', (TMB) – 0.5 g/l

Buffer (Sodium acetate) - 0.05mol/l

Urea H_2O_2 - 0.03%

6. STOP soln : (in 7.5ml)

H_2SO_4 -0.5 mol/l

Total concentration of preservative < 0.04%.

Stability of the reagents are able up to the expiry dates on the labels when stored in 2-8◦ C . Opened reagents can be used within 60 days.

MICROPLATE:

An aluminium bag sealed the microplate with a desiccant.

PREPARATION OF REAGNT:

- All the reagents brought to room temperature (15-25◦C) before use.
- Reagents always be stored about s 2- 8◦ C.
- **WASH solution (WS) :**
- Turbidity, which may appear in the concentrate , will dissolve on dilution.
- WS diluted with 1000 ml of fresh, deionised water then rinse vial several times.
- Stability in 15-25◦C up to 60 days

TESTING SAMPLE:

- The Serum
- Storage of specimens at 2-8◦C up to 5 days if up to 30 days stored at -20◦C.

PROCEDURE:

1st STEP	**WELL (MICROLITER)**	
	A1…..D2 CAL	**E2 SPECIMEN**
CAL –A-F; (duplicate)	**50**	_
SPECIMENS, CONTROLS; (duplicate)	_	**50**
CONJUGATE	**100**	**100**

Gently rock and cover the MIC by strip

Incubate at 20- 25◦C upto 60 min

Wash - 3 times

WASH	**300**	**300**
2nd STEP		
SUBSTRATE	**100**	**100**
No shaking MIC after SUB addition		
Incubate upto 15 min about 20…25◦C		
STOP	**50**	**50**
Then mixed		

Measurement of the absorbance as early as possible at **450nm** or within 30min, after reaction termination, reference wavelength using a of **630-690nm**.

VALIDATION OF THE TEST:

The calibration of highest absorbance :CAL- **A ≥ 1.3.**

CALCULATION:

The measured absorbance Plotted against the calibrator in a lin –lin graph. The Appropriate of plotted measuring points resulted in a calibration curve , from the analyte concentration in the sample can be determined.

REFERENCE VALUE:

	ADULT	PREGNANT
MEAN	**2.8picogram/ml**	**3.0 pg / ml**
STANDARD DEVIATION (S.D)	**0.7 pg /ml**	**0.6 pg /ml**
EXPECTED RANGE (≥2 S.D)	**1.4-4.2pg /ml**	**1.8-4.2pg/ml**

ESTIMATION OF FT4 (FREE T4)

METHODOLOGY:

THE ELISA METHOD

PRINCIPLE:

ELISA is used the competitive binding between FT4 in a test specimen and T4 –Peroxidase conjugation for the limited number of binding sites on the anti –T4(Sheep) coated well. The amount of conjugation of T4 –Peroxidase which is bound to the well is inversely proportional to the FT4 concentration in the specimen.

Then specimen incubated and T4 –Peroxidase conjugate unbound enzyme conjugate which is removed in the equilibrium state by washing procedure. TMB /Substrate solution is added then a blue color develops. The intensity of this blue color, which changes to yellow after the reaction stopped, this blue color is inversely proportional to the FT4 amount in the specimen.

Calibrators absorbance and specimen which is determined by using of ELISA microplate readers or automated systems of ELISA (for eg. HUMAN'S Huma - Reader or ELISYS line). Concentration of specimen is extrapolated from the dose response curve generated by utilising known antigen concentrations of serum calibrators

KIT:

MC STRIPS

8 - Well strips , coated by anti –T4sheep)

CALIBRATORS: (6 cal , 2 ml/ cal) ready for use, in human serum

FT4 CONCNTRATION	ng/ml
Calibrator 1	0 (A)
Calibrator 2	0.40 (B)
Calibrator 3	1.25 (C)
Calibrator 4	2.10 (D)
Calibrator 5	5.00 (E)
Calibrator 6	7.40 (F)

ENZYME – ANTIGEN CONJUGATE: (13 ml)

Ready for use, coloured green T4 – HRP Conjugate in a protein stabilising matrix. - 1%.

WASH SOLUTION : (20 ml)

Concentrated of ca.1000ml

Buffered saline. - 250 mmol/l

SUBSTRATE REAGENT :(14ml)

3, 3', 5,5', (TMB) – 0.5 g/l

Buffer (Sodium acetate) - 0.05mol/l

Urea H_2O_2 - 0.03%

STOP soln : (in 7.5ml)

H_2SO_4 -0.5 mol/l

PRESERVATIVES: Total concentration < 0.04%.

The reagents stability are able to stated up to expiry dates on the labels and stored at 2-8◦C .

Opened reagents have to be stored at 2- 8◦ C can be used up to 60 days.

MICROPLATE:

An aluminium bag Sealed with a desiccant.

PREPARATION OF REAGENT:

- Bring all reagents to room temperature (15-25◦C) before use.
- Reagents not in uses should always be stored at 2- 8◦ C.

WORKING WASH SOLUTION:

- Turbidity, which is Faint and may appear in the concentrate, completely dissolve on dilution.
- Dilute WS with 1000 ml of fresh, deionised water then rinse vial several times.
- Stability at 15-25◦C up to 60 days

SAMPLE:

- The Serum
- Storage of specimens for 5 days at 2-8◦C and at -20◦C up to 30 days.

PROCEDURE:

1st STEP	WELL (µl)	
	A1…..D2 CALIBRATORS	E2 SPECIMEN
CAL –A-F; in duplicate	50	_
SPECIMENS AND CONTROLS; (duplicate)	_	50
CONJUGATING AGENT	100	100

Gently rock and cover MIC with strip

Incubate at 20….25◦C up to 60 min

Wash - 3 times

WASH	300	300
2nd STEP		
SUBSTRATE	100	100
No shaking MIC after addition of SUB		
Incubated up to 15 min at 20…25◦C		
STOP	50	50
Then Mixed		

Absorbance measured at **450nm** as early as possible or within 30min, after reaction termination takes place, using a reference wavelength of **630-690nm**.

TEST VALIDATION:

Results are available if calibration of highest absorbance CAL- **A ≥ 1.3.**

TEST CALCULATION:

Measured absorbance plotted against calibrator in a lin graph. Plotted measuring points result in a calibration curve, from which the concentration of analyte in the sample can be determined.

REFERENCE VALUE:

	ADULT	**PREGNANT**
MEAN	**1.4nanogram /ml**	**1.5 ng / ml**
STANDARD DEVIATION (S.D)	**0.3nanogram /ml**	**0.37ng /ml**
EXPECTED RANGE (≥ 2 S.D)	**0.8-2.0nanogram /ml**	**0.8-2.2 ng/ ml**

ESTIMATION OF TSH

METHODOLOGY:

ENZYME LINKED IMMUNOSORBENT ASSAY (ELISA METHOD)

PRINCIPLE:

The ELISA which is used the principle of competitive binding between TSH in a test specimen and TSH –Peroxidase conjugated for the limited number of binding sites on the anti –TSH (Sheep) coated well. The amount of TSH –Peroxidase conjugate which is bound with the well and is inversely proportional to the TSH concentration in the specimen.

Incubated specimen and TSH –Peroxidase conjugation with unbound enzyme then conjugate is removed by washing procedure. Then TMB /Substrate solution is added and a blue color develops. This blue color intensity, which changes to yellow after stopping of the reaction and is inversely proportional to the TSH amount in the specimen.

Calibrator's absorbance and specimen which is determined by using of ELISA microplate readers or automated ELISA systems (eg.HUMAN'S Huma - Reader or ELISYS line). Concentration of Specimen is extrapolated from a dose response curve generated by known antigen concentrations of serum calibrators .

KIT :

MIC STRIPS

8 - Well strips , coated by anti –TSH sheep)

CALIBRATORS: (6cal , 2 ml/ cal)

TSH CONCNTRATION	Micro International Unit/mille litre
Calibrator 1	0 (A)
Calibrator 2	0.5 (B)
Calibrator 3	3.0 (C)
Calibrator 4	6.0 (D)
Calibrator 5	15.0 (E)
Calibrator 6	30.0(F)

ENZYME – ANTIGEN CONJUGATE: (13 ml)

Red coloured anti-TSH (goat), labelled with HRP.

WASH SOLUTION : (50 ml)

Concentrated for ca.1000ml – pH 6.25 ± 0.1

Tris buffered. - 10mmol/l

NaCl - 8gm/l

SUBSTRATE REAGENT :(13ml)

TMB – 1.2mmol/l

H_2O_2 - ≤ 6.0 mmol/l

STOP SOLUTION: (15ml)

Sulphuric acid -0.5 mol/l

PRESERVATIVES: Total concentration < 0.1%.

Reagents stability are up to the stated expiry dates on the labels ,when stored at 2-8◦C.

MICROPLATE:

An aluminium bag sealed with a desiccant.

PREPARATION OF REAGENTS:

- All reagents brought to the room temperature (15-25◦C) before use.

WORKING WASH SOLUTION:

- Turbidity, which is faint and may appear in the concentrate, will dissolve completely on dilution.
- WS 1 + 20 diluted with fresh, deionised water .eg 50 ml WS + 1000ml = 1050ml.

TET SAMPLE:

- Serum sample
- Storage of Specimens at 2-8◦C and at -20◦C, upto 5 days and 30 days respectively.

PROCEDURE:

Reagents and test sample must be at the room temperature before use.

1st STEP	**WELL (µl)**	
	A1…..D2 CALIBRATORS	**E2 SPECIMEN**
CAL –A-F; in duplicate	**50**	_
SAMPLE AND CONTROLS;(duplicate)	_	**50**
CONJUGATE	**100**	**100**

Gently rock it and cover MIC with strip

Incubation at 20….25◦C up to 60 min

Wash - 3 times

WASH	**300**	**300**
-2nd STEP		
SUBSTRATE	**100**	**100**
No shaking MIC after addition of SUB		
Incubation at 20…25◦C up to15 min		
STOP	**100**	**100**
Then mixed		

Absorbance measured at **450nm** as early as possible or within 30minutes, after terminating of reaction, by using of a reference wavelength of **630-690nm**.

TEST VALIDATION:

CALIBRATOR	RANGEOFACCEPTANCE(OD)
A	**< 0.05**
B	**>2.0 × absorbance CAL(A)**
C	**>3.0 × absorbance CAL(B)**
D	**>1.4 × absorbance CAL(C)**
E	**>1.9 × absorbance CAL(D)**
F	**>1.5 × absorbance CAL(E)**
F	**>1.2**

TEST CALCULATION:

Measured the absorbance potted against calibrator in a Slin graph. Then plotted measuring points result in a calibration curve, from which the concentration of analyte in the sample which can be determined.

REFERENCE VALUE:

NORMAL RANGE: 0.3 - 4.0 mIU/ml

RESULTS AND STATISTICAL ANALYSIS

RESULTS AND STATISTICS:

In this study 100 psoriatic patients (case) were enrolled over an eighteen months period, 30 non psoriatic patients, other skin disorders (controls). The majority of the patients were males (51%) and the mean age was (**45.85 years)**

Table 1: Age distribution of the study population (n=130)

Age group	Cases N (%)	Controls N (%)	Total N (%)
21- 30 years	**15 (15)**	**5 (16.7)**	**20 (15.4)**
31 - 40 years	**19 (19)**	**4 (13.3)**	**23 (17.7)**
41 – 50 years	**28 (28)**	**9 (30)**	**37 (28.5)**
51 – 60 years	**27 (27)**	**11 (36.7)**	**38 (29.2)**
>60 years	**11 (11)**	**1 (3.3)**	**12 (9.2)**
Total	**100 (100)**	**30 (100)**	**130 (100)**

Mean age: 45.85 years

Standard deviation: 2.99 years

Minimum: 20 years

Maximum: 75 years

Figure 1: Bar chart showing age distribution of the study population (n=130)

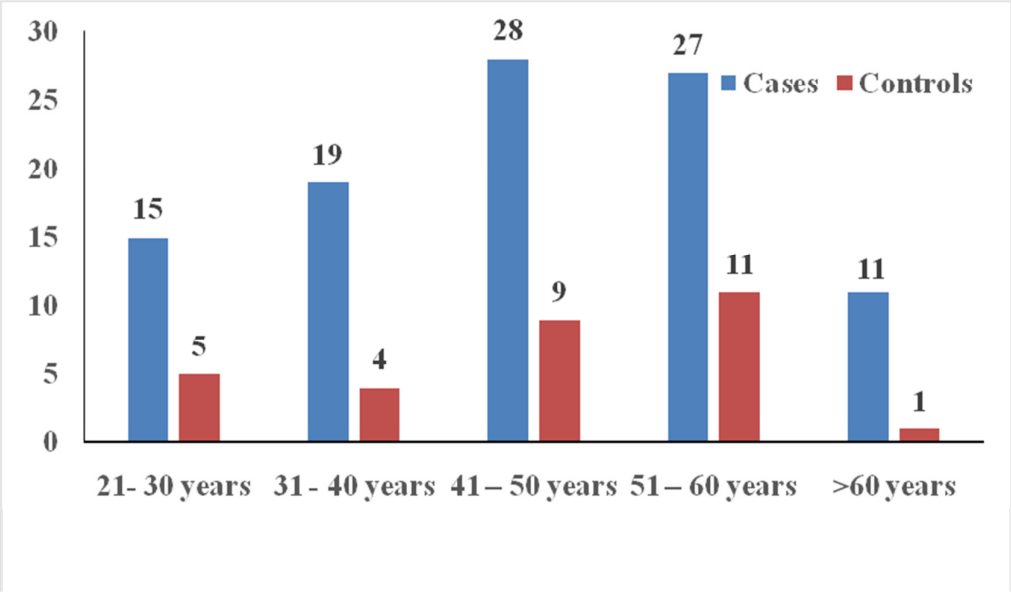

Table 2: Comparison of age among cases and controls (n=130) "T" test

Study Groups	Average Age	SD	Mean difference	p value	95% confidence interval
Cases	45.89	13.07	0.19	0.943	-5.10 to 5.48
Controls	45.70	12.06			

Comments: There was a very minimal age mean difference between the cases and the controls and this difference was not statistically significant. Hence both cases and controls were comparable.

Table 3: Gender distribution of the study population (n=130)

Gender	Cases N (%)	Controls N (%)	Total N (%)
Male	51 (51)	13 (43.3)	64 (49.2)
Female	49 (49)	17 (56.7)	66 (50.8)
Total	100 (100)	30 (100)	130 (100)

Chi square value: 0.543 **p value: 0.461**

Comments: Males and females were equally distributed in both cases and controls

Figure 2: Bar chart showing gender distribution of the study population among cases and controls(n=130)

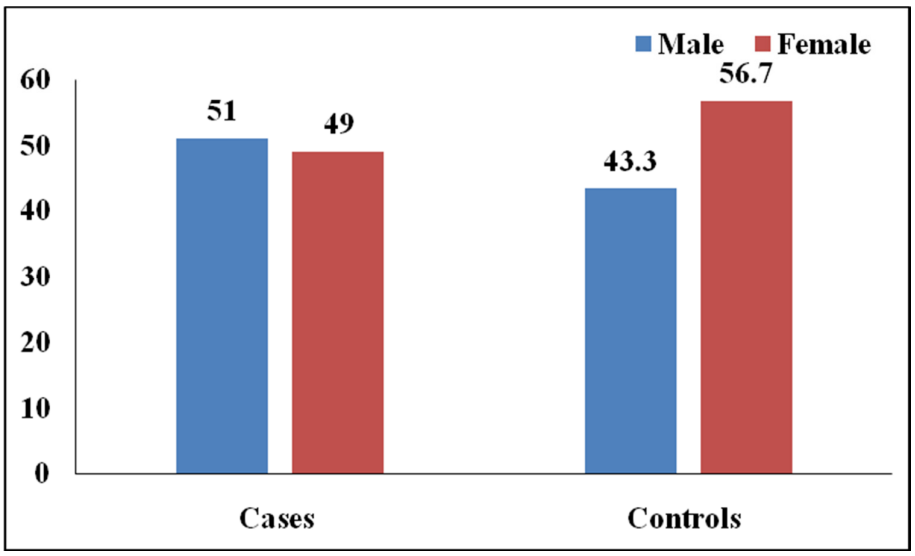

Table 4: Distribution of the Cases according to duration of Psoriasis (n=100)

Duration of Psoriasis (years)	Frequency	Percent
1 to 5	22	22.0
6 to 10	27	27.0
11 to 15	6	6.0
16 to 20	19	19.0
21 to 25	11	11.0
>25	15	15.0
Total	100	100.0

Mean disease duration: 16.06 years

Standard deviation: 11.20 years

Minimum: 1 year

Maximum: 50 years

Comments: About 50% of the cases had psoriasis for more than 10 years.

Figure 3: Bar chart showing Cases according to duration of Psoriasis (n=100)

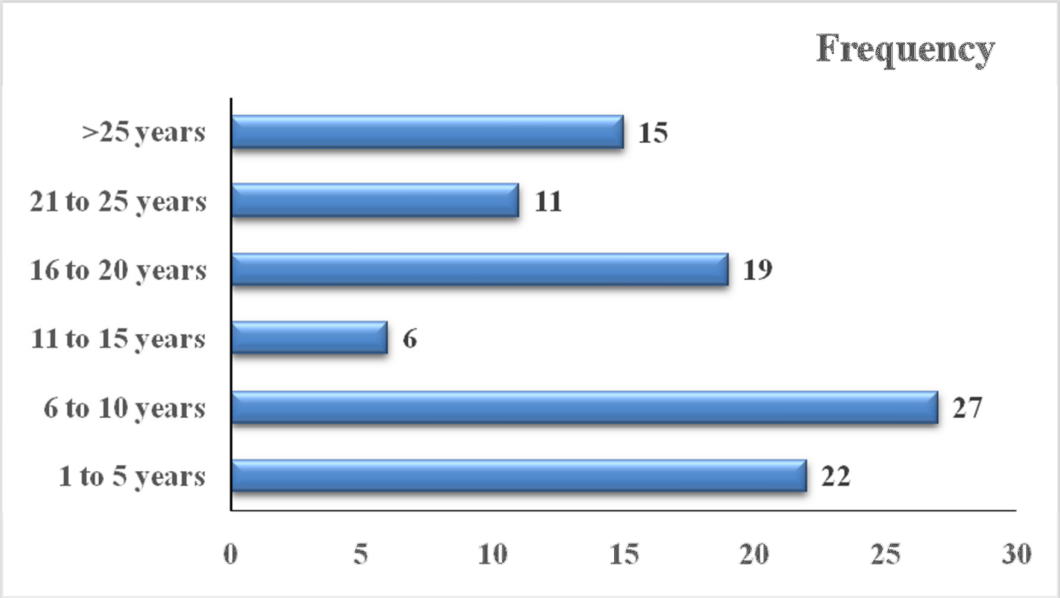

Table 5 : Distribution of cases according to marital status (n= 100)

Marital status	Frequency	Percent
Unmarried	**27**	**27.0**
Married – Non Consanguineous	**60**	**60.0**
Married – II degree Consanguineous	**3**	**3.0**
Married - III degree Consanguineous	**10**	**10.0**
Total	**100**	**100.0**

Comments: About two-third of the cases had a non- consanguineous marriage while 13% of cases had consanguineous marriage.

Figure 4: Pie chart showing Cases according to Marital status (n=100)

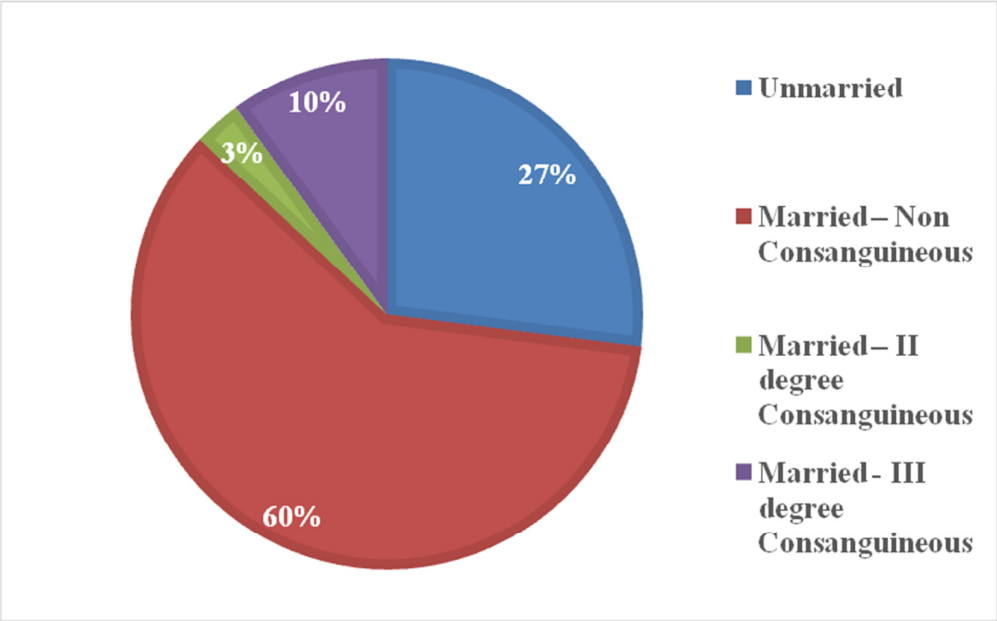

Table 6: Distribution of the Cases according to Type of diet (n=100)

Type of Diet	Frequency	Percent
Non-vegetarian diet	83	83
Vegetarian	17	17.0
Total	100	100.0

Comments: About 83% of cases were on non-vegetarian diet.

Figure 5: Doughnut chart showing Cases according to Type of diet (n=100)

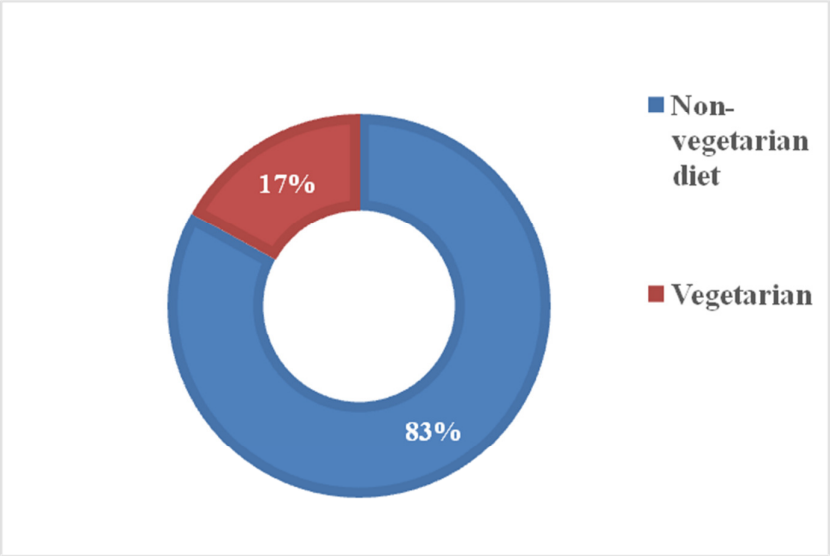

Table 7: Distribution of the Cases according to Type of residence (n=100)

Type of residence	Frequency	Percent
Urban	**57**	**57.0**
Rural	**43**	**43.0**
Total	**100**	**100.0**

Comments: More than half of the subjects were urban areas and 43% rural areas subjects.

Figure 6: Pie chart showing Cases according to type of residence (n=100)

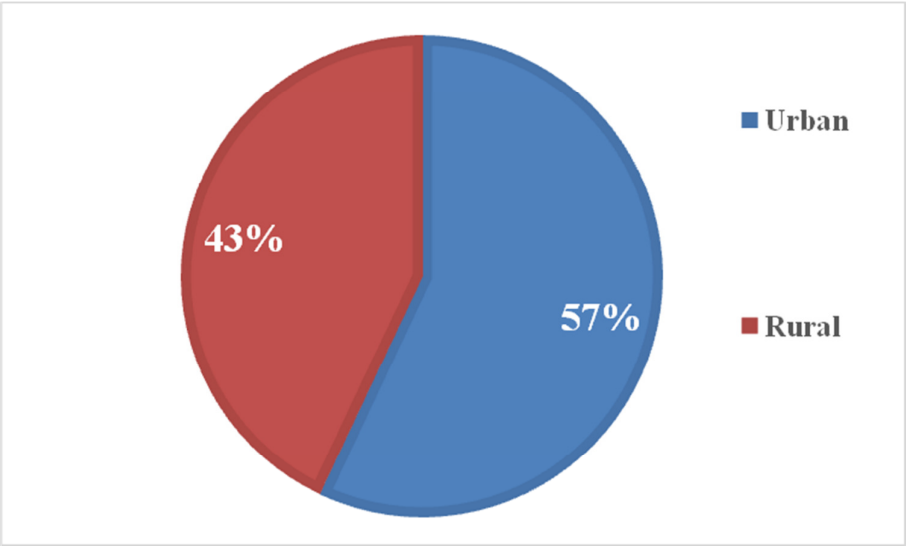

Table 8: Distribution of the Cases according to family history of psoriasis (n=100)

Family history	Frequency	Percent
Present	**0**	**0**
Absent	**100**	**100**
Total	**100**	**100**

Table 9: Distribution of the Cases according to type of psoriasis (n=100)

Type of Psoriasis	Frequency	Percent
Erythrodermic	**1**	**1.0**
Plaque	**99**	**99.0**
Total	**100**	**100.0**

Comments: Except a single case, all others had plaque type of psoriasis.

Table 10: Distribution of the Cases according to psoriatic arthritis/nail changes (n=100)

Psoriatic arthritis/ Nail changes	Frequency	Percent
Neither	**72**	**72.0**
Only Nail changes	**23**	**23.0**
Only Psoriatic arthritis	**1**	**1.0**
Both present	**4**	**4.0**
Total	**100**	**100.0**

Comments: Nail changes were present in about one-fourth of the cases while arthritis was observed only in 1 case. Both the changes were seen together in 4 patients.

Figure 7: Bar chart showing Cases according to psoriatic arthritis/nail changes (n=100)

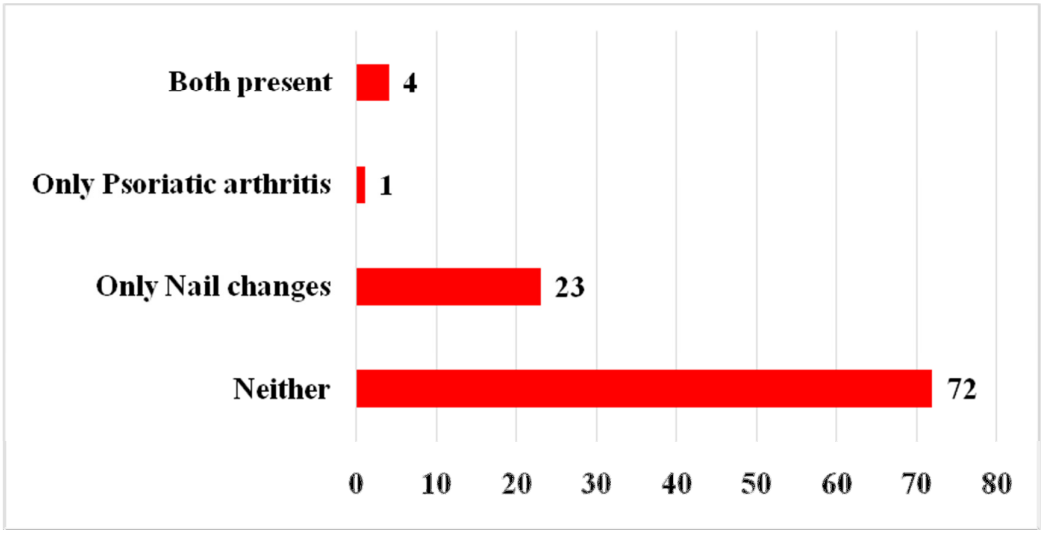

Table 11: Distribution of the Cases according to skin surface area involvement in psoriasis (n=100)

BSA score	Frequency	Percent
MILD-3%	50	50.0
MODERATE-3-10%	41	41.0
SEVERE >10%	9	9.0
Total	100	100.0

Comments: About half the cases had only mild involvement while 41% had moderate involvement of 3 to 10% of skin surface area.

Figure 8: Bar chart showing Cases according to skin surface area involvement in psoriasis (n=100)

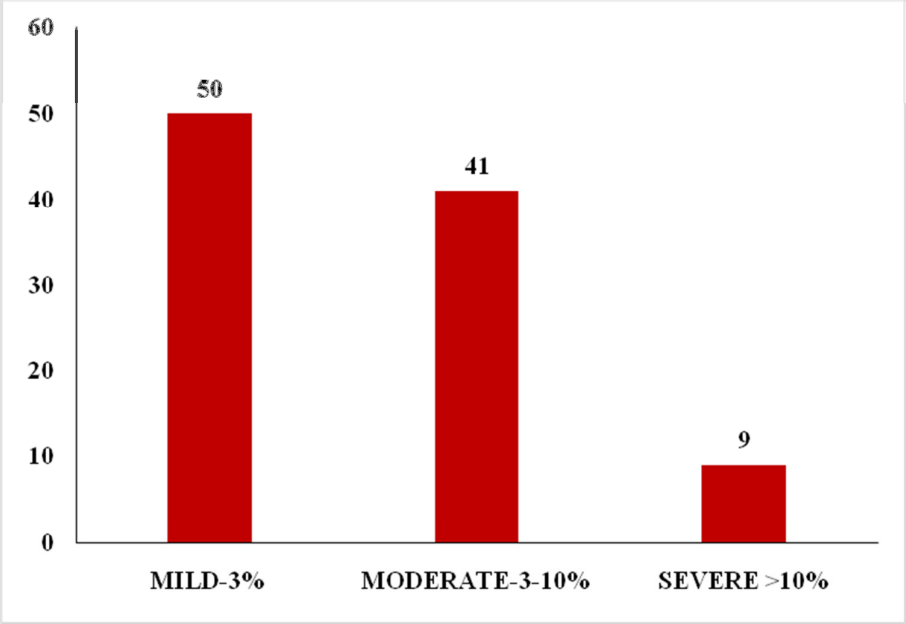

Table 12: Distribution of the Cases according to socio-economic status (Total number of population =100)

SE status	The Frequency	%
Low Socio Economic class	72	72
Middle class	28	28
Total	100	100.0

Comments: About three-fourth of the cases belong to lower socio-economic status while others were from middle class.

Figure 9: Pie chart showing Cases according to socio-economic status (n=100)

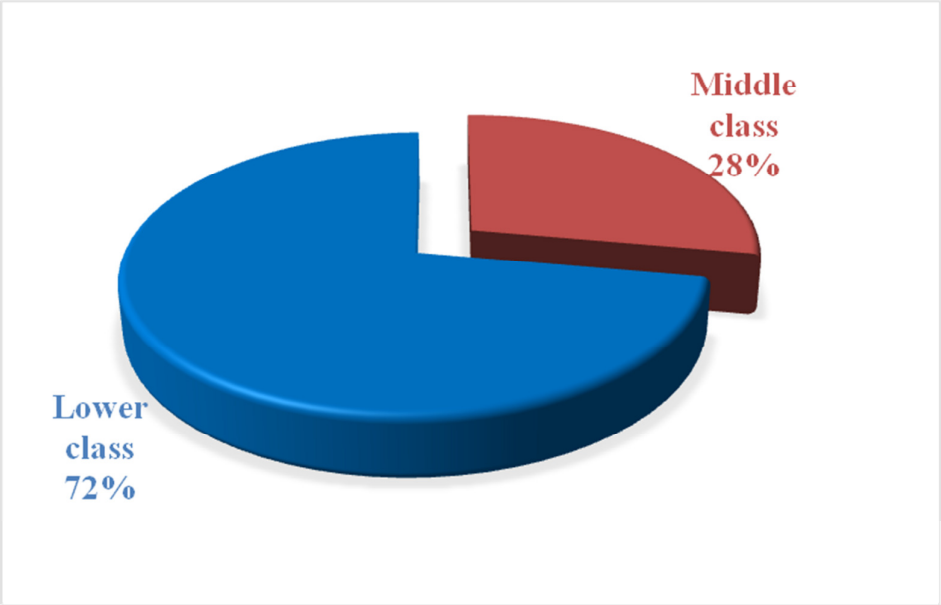

Table 13: Distribution of the Cases according to smoking status (n=100)

Smoking status	Frequency	Percent
Smoker	25	25
Not a smoker	75	75
Total	100	100.0

Comments: About three-fourth of the cases were non-smokers while others were addicted to smoking.

Figure 10: Doughnut chart showing Cases according to smoking status (n=100)

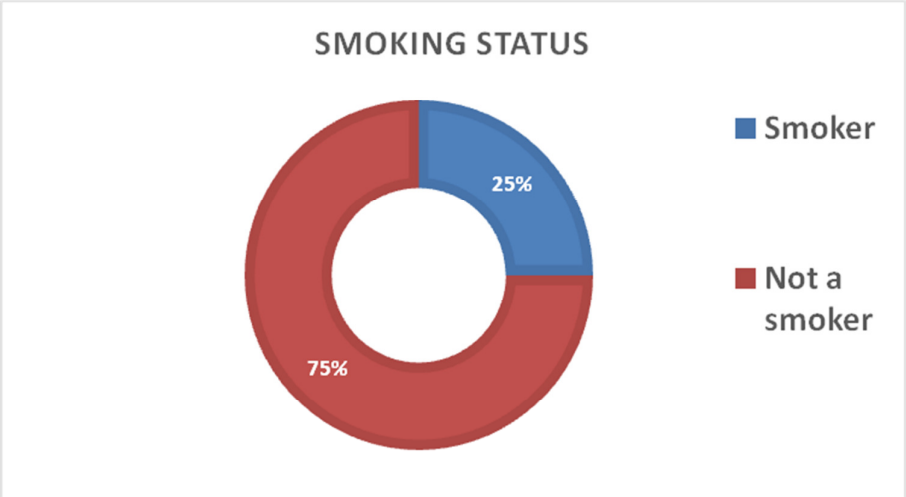

Table 14: Distribution of the Cases according to alcohol usage (n=100)

Alcohol use	Frequency	Percent
Present	**17**	**17**
Absent	**83**	**83**
Total	**100**	**100.0**

Comments: 17% of cases reported use of alcohol in one form or the other.

Figure 11: Pie chart showing Cases according to alcohol usage (n=100)

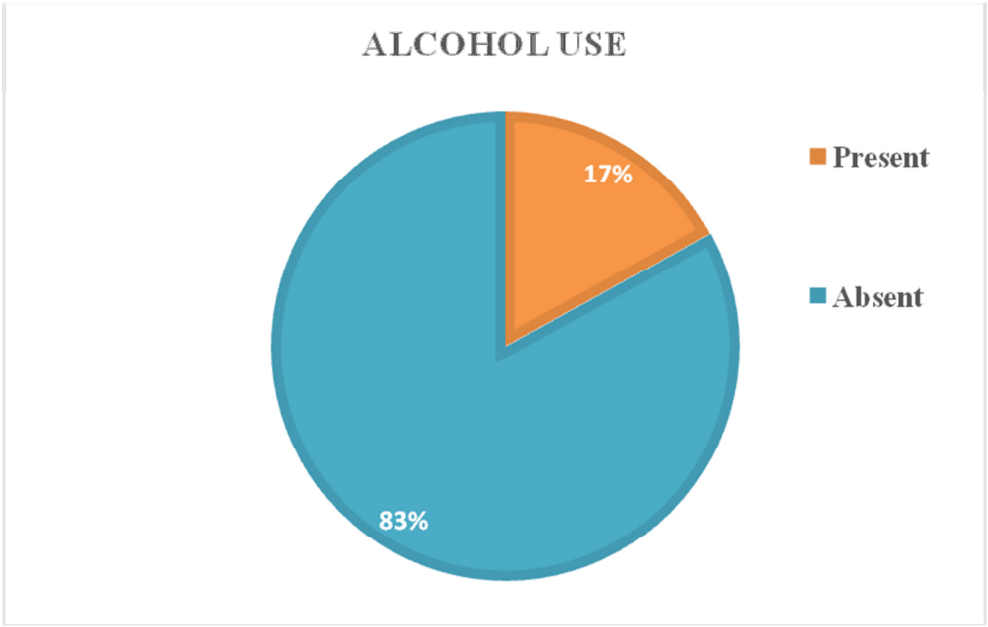

Table 15: Distribution of the study population according to anthropometric parameters (n=130)

Statistic	Height (cm)	Weight (Kg)	BMI	Waist Circumference (cm)	Hip Circumference (cm)	Waist Hip Ratio
Mean	158.05	59.48	22.770	77.35	79.25	.9759
Std. Deviation	4.934	8.277	6.4220	6.580	6.524	.01427
Minimum	145	42	11.2	56	58	0.95
Maximum	171	90	49.4	104	106	1.13

Table 16: Distribution of the study population according to biochemical parameters (n=130)

Statistics	FBG (milligram /dl)	TC (mg/dl) (milligram/dl	Triglycerides (milligram/dl	VLDL-c (milligram/ dl)	LDL -c (milligram/ dl)	HDL -c (milligram/dl)
Mean	104.19	189.87	159.92	31.67	118.13	40.09
Std. Deviation	30.806	36.881	73.803	14.708	32.493	6.671
Minimum	72	108	62	12	64	23
Maximum	276	282	368	73	198	57

Table 17: The Metabolic Syndrome prevalence among psoriatic patients and controls (n=130)

Parameter	Group	Frequency	Percentage
Waist circumference (in centimeter)	Normal	111	85.4%
	Elevated (>90 for males, >80 for females)	19	14.6%
Fasting Blood Glucose (mg/dl)*	Normal (<100)	91	70%
	Elevated (>100)	39	30%
Serum Triglyceride level (mg/dl)**	Normal	73	56.2%
	Elevated (>=150)	57	43.8%
Serum HDL level (mg/dl)***	Normal	40	30.8%
	Reduced HDL (<40 for males,<50 for females)	90	69.2%
Blood pressure (mm Hg)****	Normal	97	74.6%
	Elevated (SBP>+130 and/or DBP>=85)	33	25.4%

Table 18: Distribution of Metabolic Syndrome components among psoriatic and non psoriatic subjects (n=130)

Components		Group		p value (Chi-square test)
		Cases N (%)	Controls N (%)	
Waist circumference (in centimeter)	Normal	86 (86%)	25 (83.3%)	0.717
	Elevated	14 (14%)	5 (16.7%)	
Fasting Blood Glucose (mg/dl)*	Normal	62 (62%)	29 (96.7%)	<0.001
	Elevated	38 (38%)	1 (3.3%)	
Serum Triglyceride level (mg/dl)**	Normal	45 (45%)	28 (93.3%)	<0.001
	Elevated	55 (55%)	2 (6.7%)	
Serum HDL level (mg/dl)*	Normal	29 (29%)	11 (36.7%)	0.425
	Reduced	71 (71%)	19 (63.3%)	
Blood pressure (mm Hg)**	Normal	68 (68%)	29 (96.7%)	0.002
	Elevated	32 (32%)	1 (3.3%)	

Comments:

1. Central obesity was almost equal in both cases and controls (14% versus 16.percentage) , ($p>0.05$).

2. Elevated Fasting Blood glucose was common in Cases (38%) than in controls (3.3%) and also elevated serum triglyceride level was more in cases (55%) than controls (6.7%) and this differences were statistically significant ($p<0.05$).

3. Reduced serum HDL level was more in cases (71%) than controls (63.3%) and it's p value- ($p>0.05$).

4. Elevated blood pressure was common in Cases (32%) than in controls (3.3%) and these difference were statistically significant ($p<0.05$).

5. In Cases, the components in descending order of prevalence are Reduced HDL > Raised level of Triglycerides > increased level of fasting glucose > Raised level of blood pressure > Central obesity.

6. In controls, the components in descending order of prevalence are Reduced HDL > obesity > Elevated level of Triglycerides > Elevated blood pressure & Elevated fasting glucose.

Figure 12: Bar chart showing the Metabolic Syndrome prevalence among study population and control group (n=130)

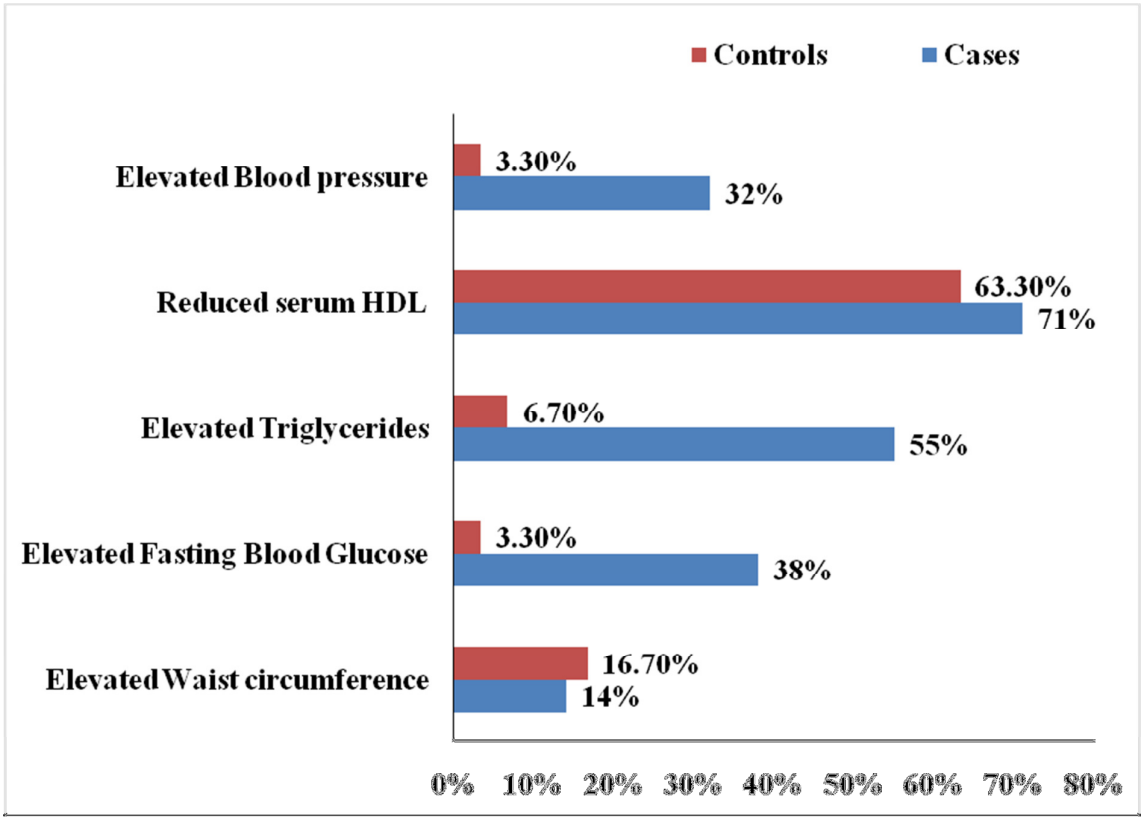

Table 19: Based on NCEP ATP III criteria MS components in both Psoriatic and non Psoriatic individuals: (Total number =130)

Criteria	Metabolic syndrome	Frequency	Percentage
Modified NCEP Adult Treatment Panel III (ATP III)	Present	38	29.2 %
	Absent	92	70.8 %

Comments: Prevalence of metabolic syndrome was 29.2% among the study population

Table 20: Distribution of study population based on the individual MS components among cases and controls (n=130)

Number of Components	Frequency	Percent
No components	18	13.8
1	43	33.1
2	31	23.8
3	21	16.2
4	15	11.5
5 (All components)	2	1.5
Total	130	100.0

Comments:

1. About 14% of the study population were free from any MS component .

2. About 86% had one or more components of metabolic syndrome, while 1.5% were suffering from all 5 components of metabolic syndrome according to NCEP ATP III criteria.

Figure 13: Bar chart showed that the Metabolic Syndrome in both study and control groups (n=130)

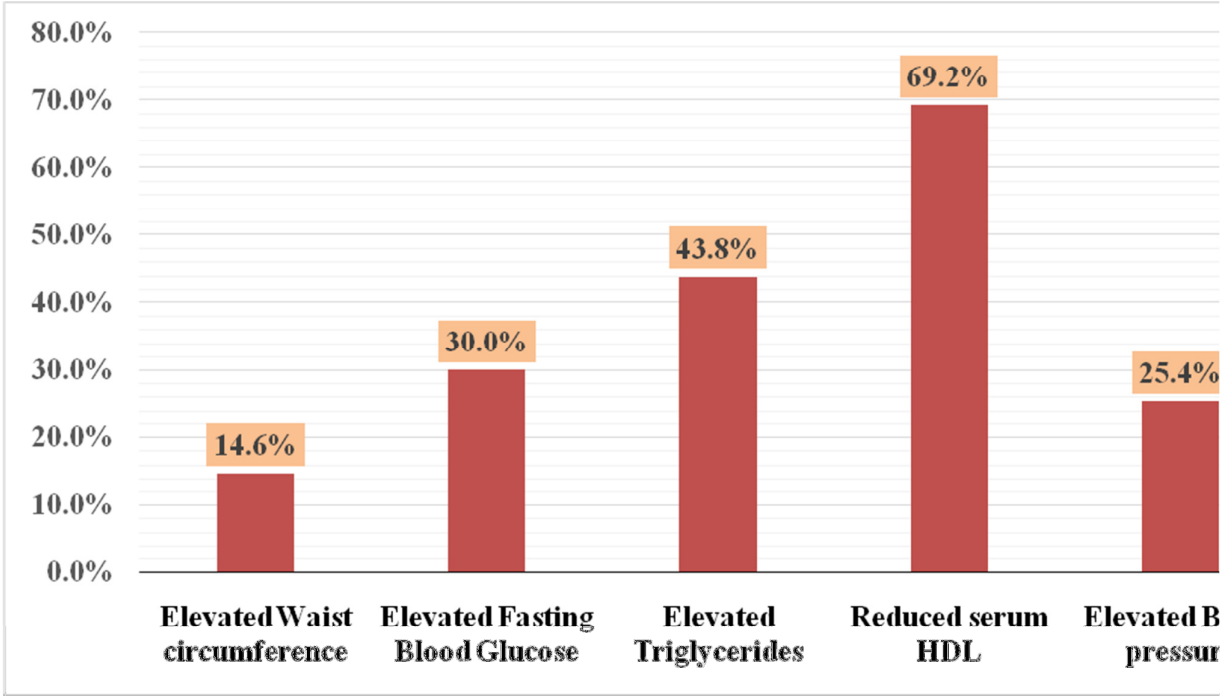

Table 21: Distribution of the metabolic syndrome among cases and controls (n=130)

Metabolic syndrome	Cases N (%)	Controls N (%)	Total N (%)
Present	37 (37)	1 (3.3)	38 (29.2)
Absent	63 (63)	29 (96.7)	92 (70.8)
Total	100 (100)	30 (100)	130 (100)

Chi square value: 0.543 **p value: <0.001**

ODDS RATIO: 17.032 **p value: 0.006**

Odds ratio: 2.22 to 130.25

Comments:

1. It's prevalence was very high among cases (Thirty seven%) in comparison to controls (3.3%) and this difference was statistically significant.

2. Cases had 17 times higher odds of having metabolic syndrome than controls and this risk was also statistically significant.

Figure 14: Stacked Bar chart showing prevalence of Metabolic Syndrome among cases and controls (n=130)

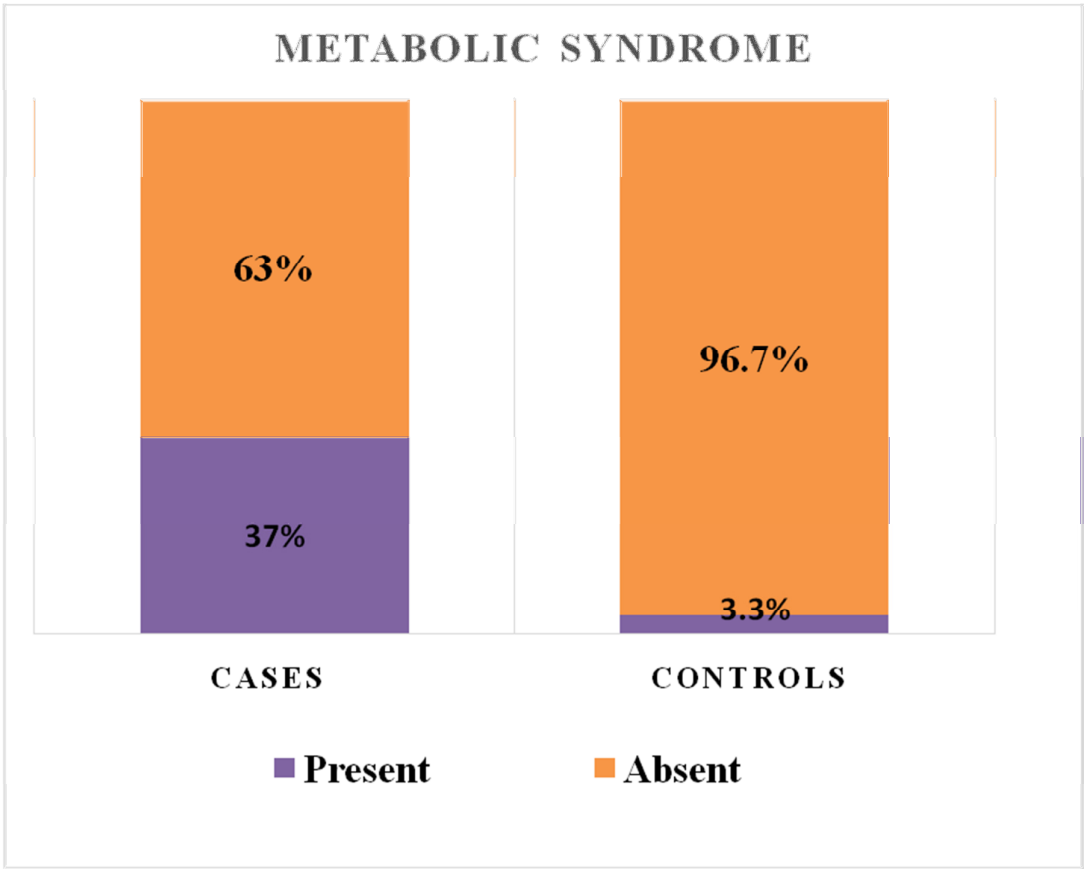

Figure 15: Pie chart showing MS in study population (n=100)

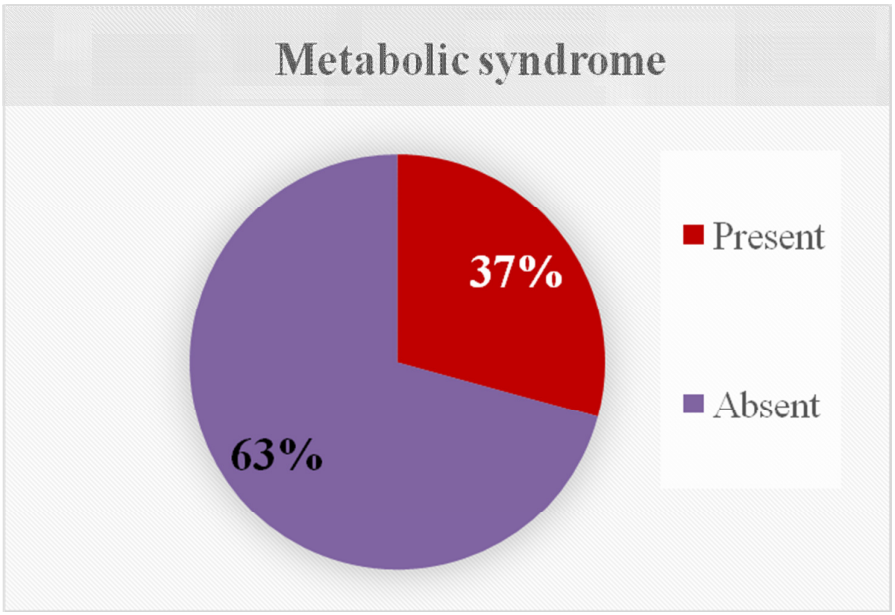

Figure 16: Bar chart showing Number of Components of Metabolic Syndrome among study population (n=130)

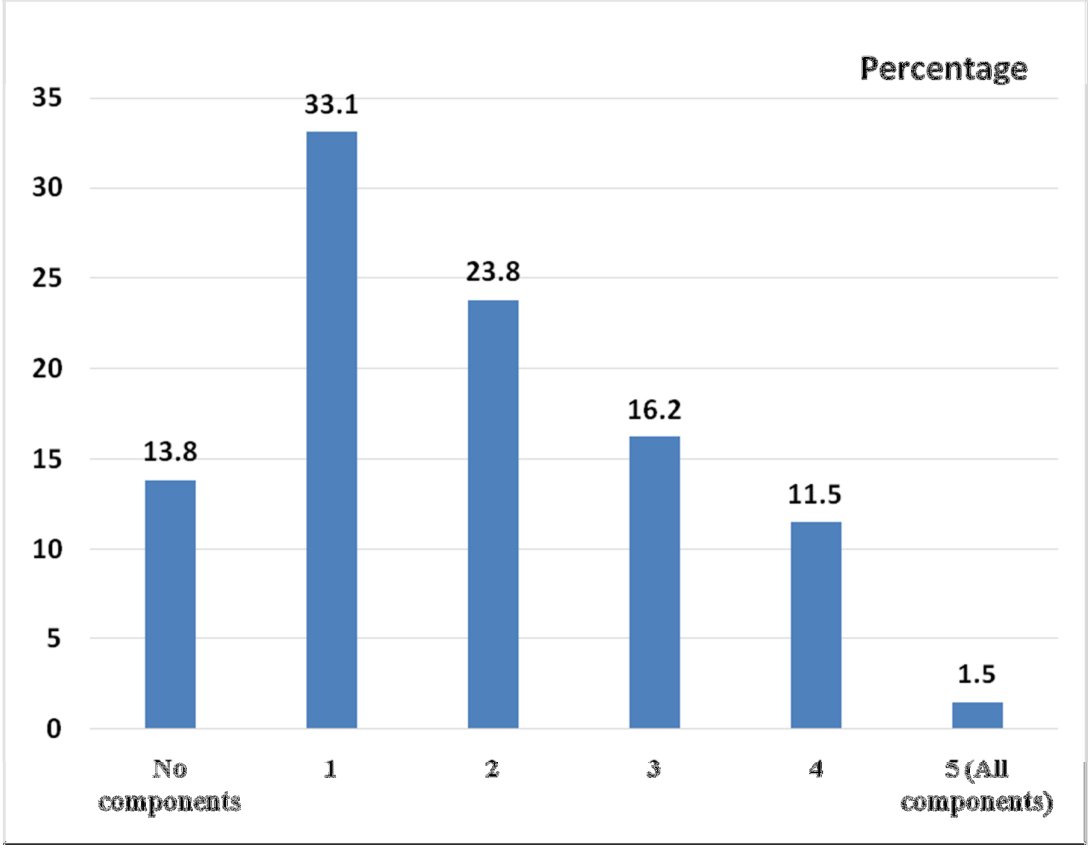

Table 22: Comparison of age among cases with and without metabolic syndrome (n=100)

The "t" test

Metabolic Syndrome	Average Age	SD	Mean difference	Value of p	95% confidence interval
Present (37)	52.41	10.76	10.34	<0.001*	5.35 to 15.32
Absent (63)	42.06	12.86			

* **Significant at 0.05 level**

Comments:

The Metabolic Syndrome subjects had higher mean age of 52 years when compared to subjects without metabolic syndrome (42 years) and this mean difference was statistically significant. Hence, age of the patient which is an important factor in the occurrence of metabolic syndrome viz as age increases, risk of metabolic syndrome also increases.

Table 23: Comparison of duration of psoriasis among cases with and without metabolic syndrome (n=100)

Student "T" test

Metabolic Syndrome	Mean duration of psoriasis	Std. Deviation	Mean difference	p value	95% confidence interval
Present (37)	20.49	12.74	7.025	0.002*	2.61 to 11.43
Absent (63)	13.46	9.36			

*** Significant at 0.05 level**

Comments:

The Metabolic Syndrome Subjects had higher duration of psoriasis in comparison to subjects without metabolic syndrome and this mean difference was statistically significant. Hence, duration of psoriasis was also very important factor in the occurrence of metabolic syndrome viz as duration of psoriasis increases, risk of metabolic syndrome also increases among patients.

Table 24: Distribution of the metabolic syndrome among cases based on type of diet (n=100)

Diet	Metabolic syndrome	NO Metabolic syndrome	Total N (%)
Non-Vegetarian N (%)	33 (39.8)	50 (60.2)	83 (100)
Vegetarian N (%)	4 (23.5)	13 (76.5)	17 (100)
Total	37 (37)	63 (63)	100 (100)

Chi square value: 1.594 p value: 0.207

Comments: Metabolic syndrome was common among subjects taking non-vegetarian diet (40%) than subjects taking vegetarian diet (23.5%) but this difference was not statistically significant.

Table 25: Distribution of the metabolic syndrome among cases based on type of residence (n=100)

Residence	Metabolic syndrome	NO Metabolic syndrome	Total N (%)
Urban N (%)	21 (36.8)	36 (63.2)	57 (100)
Rural N (%)	16 (37.2)	27 (62.8)	43 (100)
Total	37 (37)	63 (63)	100 (100)

Chi square value: 0.001 p value: 0.970

Comments: The occurrence of metabolic syndrome was almost same among all patients with psoriasis irrespective of their type of residence whether urban or rural.

Table 26: Distribution of the metabolic syndrome among cases based on type of psoriasis (n=100)

Psoriasis	Metabolic syndrome	NO Metabolic syndrome	Total N (%)
Erythrodermic N (%)	0 (0)	1 (100)	1 (100)
Plaque N (%)	37 (37.4)	62 (62.6)	99 (100)
Total	37 (37)	63 (63)	100 (100)

Comments: Metabolic syndrome and its association with type of psoriasis cannot be commented as only 1 patient had erythrodermic type of psoriasis and all the others had plaque type.

Table 27: Distribution of the metabolic syndrome among cases based on severity of psoriasis according to BSA score (n=100)

BSA score	Metabolic syndrome	NO Metabolic syndrome	Total N (%)
MILD-3%	18 (36)	32 (64)	50 (100)
MODERATE-3-10%	15 (36.6)	26 (63.4)	41 (100)
SEVERE >10%	4 (44.4)	5 (55.6)	9 (100)
Total	37 (37)	63 (63)	100 (100)

Chi square value: 0.238 p value: 0.888

Comments: The occurrence of metabolic syndrome was almost same among all patients with psoriasis irrespective of severity of psoriasis according to BSA score. This indicates that the occurrence of metabolic syndrome is an 'all or none' phenomenon because of the underlying pathogenesis and is not associated with severity of disease.

Table 28: Distribution of the metabolic syndrome among cases with and without arthritis (n=100)

Arthritis	Metabolic syndrome	NO Metabolic syndrome	Total N (%)
Absent N (%)	35 (36.8)	60 (63.2)	95 (100)
Present N (%)	2 (40)	3 (60)	5 (100)
Total	37 (37)	63 (63)	100 (100)

Chi square value: 0.020 p value: 0.887

Comments: The occurrence of metabolic syndrome was almost same among all patients with psoriasis irrespective of occurrence of arthritis.

Table 29: Distribution of the metabolic syndrome among cases with and without nail changes (n=100)

Nail changes	Metabolic syndrome	NO Metabolic syndrome	Total N (%)
Absent N (%)	25 (34.2)	48 (65.8)	73 (100)
Present N (%)	12 (44.4)	15 (55.6)	27 (100)
Total	37 (37)	63 (63)	100 (100)

Chi square value: 0.879 p value: 0.348

Comments: The occurrence of metabolic syndrome was almost same among all patients with psoriasis irrespective of occurrence of nail changes.

Table 30: Distribution of thyroid profile parameters between cases and controls (n = 130)

	Group	N	Mean	Mean difference	Student "t" test p value
FT3 (pg/ml)	Cases	100	2.117	0.0803	0.53
	Controls	30	2.037		
FT4 (ng/dl)	Cases	100	1.284	-0.0193	0.809
	Controls	30	1.303		
TSH (mIU/ml)	Cases	100	3.580	1.303	0.001
	Controls	30	2.277		

Comments:

1. Cases had higher mean TSH levels than controls (3.5 vs 2.2) and this mean difference was statistically significant

2. Serum T3 and T4 values did not differ much between cases and controls and the minor difference was not statistically significant.

DISCUSSION

DISCUSSION

Psoriasis patients are at risk of developing MS, the proper mechanism of action involved is not known exactly. Gerald Reaven, an endocrinologist from Stanford University, first described the MS in 1988. MS and psoriasis share certain common immunological mechanisms. The abdominal (Intra) fat acts as an endocrine organ capable of secreting adipocytokines that accelerate inflammation, affect glucose metabolism and vascular endothelial biology [17].

Proinflammatory cytokines are found on plaques of psoriasis and these involved in the features of insulin resistance, dyslipidemia and hypertension.

Leptin, another hormone secreted by the adipocytes, has a role in acute inflammation and chronic inflammation via the regulation of cytokine expression that modulates the type 1 and type 2 T-helper cells. Hyperleptinemia has been associated with the development of MS. Elevated leptin have also been observed in psoriasis. However, the exact effect in psoriasis is yet to be explored [64].

Many studies have identified that psoriasis is associated with metabolic syndrome. Zindancy et al [25] after studying plaque type of 115 psoriatic patients and 140 healthy controls found the Insulin resistance syndrome in cases (53%) than controls (39%), (p value was <0.001). Gisondi et al [20], studied 338 plaque psoriatic patients as well as controls and they found MS which statistically significant in psoriatic subjects.

(30.1%) controls (20.6%) , (p= 0.005) using (NCEP) ATP III criteria.

Sristi Lakshmi et al [29] south Indian study have shown that prevalence of metabolic syndrome in cases (32.5%) when compared to controls (30%) as per NCEP ATP III criteria, but the difference was not statistically significant.

Our study observed that the prevalence of Metabolic Syndrome in cases out of 100 psoriatic patients (37%), compared to controls out of skin disease patients other than psoriasis (3.3%), ($p < 0.001$).

According to Misra and Khurana [61], South Asian population is, in general inherently predisposed to an increased risk of metabolic syndrome and associated cardiovascular risk factor compared to Caucasians.

MS in patients was associated with higher age in our study. Metabolic Syndrome subjects had higher mean age of (52 years) when compared to subjects without metabolic syndrome (42 years) and , standard deviation (10.76), mean difference (10.34) this mean difference was statistically significant, ($p < 0.01$). Hence, age of the patient is very important factor in the occurrence of metabolic syndrome as age increases; risk of metabolic syndrome also increases.

Gisondi et al [20]. found that MS was common after 40 years of age. Zindancy et al [25] et al., found that MS was common in the age group of 40-50 years of age.

Our study found that the occurrence of metabolic syndrome was almost same among all patients with psoriasis irrespective of severity of psoriasis according to BSA score. This indicates that the occurrence of metabolic syndrome is an 'all or none' phenomenon because of the underlying pathogenesis and is not associated with severity of disease. Zindancy et al [25] et al found that prevalence of MS was

independent of severity of psoriasis. Kim et al [62] found that MS was associated with severe forms of psoriasis (p=0.048).

On analyzing the individual components of MS in psoriatic patients, Central obesity was almost equal in both cases and controls (14% versus sixteen. Seven%) but which value not statistically significant (p value >0.05).Elevated Fasting Blood glucose was common in Cases (38%) than in controls (3.3%) , (p value < 0.001) and also elevated serum triglyceride level was more in cases (55%) than controls (6.7%) and differences were statistically significant (p<0.05).

Reduced serum HDL level was more in cases (71%) than controls (63.3%) and It's difference not significant statistically (p>0.05).Elevated blood pressure was common in Cases (32%) than in controls (3.3%) and it was statistically highly significant (p<0.05).

In Cases, the components in descending order of prevalence are Reduced HDL > Elevated level of Triglycerides > Raised FPG > Raised BP> Central obesity. In controls, the components in descending order of prevalence are Reduced HDL > Obesity > Elevated Triglycerides > Elevated blood pressure & Elevated fasting glucose. Increased blood sugar level was an important factor contributing to increased prevalence of MS in psoriatic patients. The possible explanation is that psoriasis and diabetes share common genetic loci. CDKLI gene has been associated with both psoriasis and type2 diabetes mellitus.

About 14% of the study populations were free from any MS component by using NCEP Ault Treatment Panel III criteria. About 86% had one or more components of

metabolic syndrome, while 1.5% was suffering from all 5 Metabolic Syndrome components based on NCEP ATP III criteria.

Sristi Lakshmi et al [29] South Indian study found that statistically significant level of fasting blood sugar among those with MS (P<0.001), but they could not found a significantly higher prevalence of other components of MS such as obesity, hypertension and dyslipidemia among psoriatic patients with MS.

Several studies have demonstrated that higher lipid levels in psoriasis. Shapiro et al [26] found that psoriasis was associated hyperlipidemia.

Several studies have found that IL-2 , IL-8 and Tumour Necrosis Factor-Alpha levels, correlate with psoriasis. These cytokines also play a role in the development of psoriasis [60]. An increased level of these cytokines is a possible explanation of triglyceridemia in psoriasis. In our study duration of psoriasis higher when associated to MS than without MS and this mean difference was statistically significant. Mean duration of psoriasis (20.49), Std. Deviation (12.74), Mean difference (7.025), p value (0.002*).Hence, duration of psoriasis was also most important factor in the occurrence of metabolic syndrome as duration of psoriasis increases, risk of metabolic syndrome also increases among patients.

Metabolic syndrome was common among subjects taking non-vegetarian diet (40%) than subjects taking vegetarian diet (23.5%) and this difference was not statistically significant (p value: 0.207).

In this study the occurrence of metabolic syndrome was almost same among all patients with psoriasis irrespective of their type of residence whether urban or rural (p value: 0.970).

In our study the occurrence of metabolic syndrome was almost same among all patients with psoriasis irrespective of occurrence of arthritis (p value: 0.887). The occurrence of metabolic syndrome was almost same among all patients with psoriasis irrespective of occurrence of nail changes (p value: 0.348).

T3 and T4 thyroid hormones can play a role in psoriasis due to the mechanism of increasing the level of epidermal growth factor which may involved in the participation of keratinocyte proliferation. Many immunological mechanisms and biochemical processes, lead to enhanced action in the inflammation of the dermis and proliferation of epidermal cells .

In our study level of FT3 (pg/ml) in cases Mean (2.117), in Controls Mean (2.037), Mean difference (0.0803), p value (0.53) FT4 (pg/ml) in cases Mean (1.284), in Controls Mean (1.303), Mean difference (-0.0193), p value (0.809). TSH (mIU/ml) in cases Mean (3.580), in Controls Mean (2.277), Mean difference (1.303), p value (0.001).

Cases had higher mean TSH levels than controls (3.5 vs 2.2) and this mean difference was statistically significant. Serum T3 and T4 values did not differ much between cases and controls and the minor difference was not statistically significant.

CONCLUSION

THE CONCLUSION OF STUDY

One the most common skin disorder in India is Psoriasis, both the epidemiological characteristics and its prevalence similar as in west. results suggest that there is correlation between psoriasis and metabolic syndrome.

Psoriasis may manifest as a multisystem disease not restricted to the skin and its appendages. The association of psoriasis with several comorbidities may occur due to various factors, such as the chronic inflammatory nature of the disease, genetic susceptibility, environmental factors and related to the patient's quality of life and even adverse effects o drugs used for systemic therapy.

Comorbidities that are associated with psoriasis greatly increase the morbidity and mortality of the disease. Many studies suggested that psoriasis may take a role as a risk factor for diseases such as ischemic heart disease. Therefore screening for metabolic syndrome which is essential to prevent the further complications.

One of the important tissues for thyroid hormones is skin and this leads to rising of endothelial growth factor and therefore accelerates epidermal proliferation. Thyroid function test which can be useful in patients with relapsing psoriasis as well as in an uncontrolled Psoriasis. Thyroid hormones may play a role in the Psoriatic etiology so must be investigated in wider clinical ,comprehensive and laboratory studies.

ANNEXURE

MASTER CHART - CONTROLS

AGE	SEX	DIAGNOSIS	HT (CM)	WT (Kg)	HC (cm)	WC (cm)	BMI	BP (mmhg)	FBS (mg/dl)	T.C (mg/dl)	TGL (mg/dl)	VLDL (mg/dl)	LDL (mg/dl)	HDL (mg/dl)	FT3 (pg/ml)	FT4 (ng/dl)	TSH (Miu/ml)
55	F	DERMATITIS	158	60	82	80	24	130/70	88	170	110	22	108	40	2.1	1.2	3.4
50	F	INSECT BITE ALLERGY	155	53	78	76	22	110/80	97	165	90	18	102	45	2.6	1.3	2.3
37	F	ACNE VULGARIS	165	54	80	78	19	130/80	80	174	130	26	105	43	2.8	1.4	2.7
55	F	FURUNCULOSIS	154	56	76	74	23	120/80	87	199	67	13	149	37	1.9	1	2.7
29	M	TINEA VERSICULAR	148	51	72	70	23	120/80	99	156	89	17	94	45	2.4	1.2	1.5
31	M	URTICARIA	152	60	78	76	25	100/70	93	140	98	19	75	46	2.4	1.4	1.1
55	F	WARTS	157	52	76	74	21	130/80	92	164	115	23	97	44	0.6	0.3	3.2
35	F	VITILIGO	158	54	75	73	21	100/80	87	186	94	18	126	42	1.2	1	2.4
25	F	HERPES SIMPLEX	156	59	76	74	24	110/60	81	145	86	17	80	48	2.2	1.2	1.4
55	F	DERMATITIS	160	55	80	78	21	120/80	86	168	112	22	104	42	1.9	2.4	2.6
46	M	ECZEMA	154	64	81	79	26	130/80	79	166	155	31	103	32	2.2	1.6	1.2
45	M	CALLOSITY	149	56	74	72	25	120/70	87	150	76	15	88	47	2	1.2	0.6
40	F	CORN FOOT	162	54	78	76	20	130/70	95	180	94	18	118	44	2.1	0.8	1.6
55	M	CORN FOOT	157	69	80	78	27	140/80	84	204	112	22	139	43	2	1	0.7
43	M	ALOPECIA AREATA	159	60	82	80	23	120/70	97	156	86	17	94	45	2.4	1.6	0.6
50	F	CONTACT DERMATITIS	146	50	72	70	23	110/70	80	169	120	24	100	45	2.4	1	2.6
25	M	APTHOUS ULCER	150	54	78	76	22	120/70	83	154	85	17	87	50	2.2	1.6	1.8
72	M	CORN FOOT	169	65	86	84	22	130/80	92	198	148	29	131	38	3.2	1.4	1.8
55	F	ECZEMA	158	55	76	74	22	120/80	81	182	153	30	110	42	1.9	1.7	2.2
24	M	DRUG ERUPTION	154	59	78	76	24	110/80	98	130	85	17	67	46	2.2	1.2	1.4

AGE	SEX	DIAGNOSIS	HT (CM)	WT (Kg)	HC (cm)	WC (cm)	BMI	BP (mmhg)	FBS (mg/dl)	T.C (mg/dl)	TGL (mg/dl)	VLDL (mg/dl)	LDL (mg/dl)	HDL (mg/dl)	FT3 (pg/ml)	FT4 (ng/dl)	TSH (Miu/ml)
48	F	FURUNCULOSIS	158	69	90	88	27	130/80	86	136	128	25	73	38 **CONTROLS**	2.2	1.9	2.8
44	F	KELOID	156	60	82	80	24	120/80	94	145	84	16	85	44	1.8	1.7	1.8
51	F	INSECT BITE ALLERGY	155	54	83	81	22	130/70	84	162	90	18	103	41	2.6	1.6	0.7
27	M	ACNE VULGARIS	168	56	82	81	19	100/70	80	142	80	16	80	46	1.5	1.5	1.4
55	M	DERMATITIS	156	59	80	78	24	120/80	78	178	90	18	118	42	1.5	1.4	1.9
46	M	TINEA CORPORIS	159	56	78	76	22	130/70	90	186	125	25	120	41	2.6	1.1	5.9
60	F	PEDICULOSIS	158	60	80	78	24	120/70	80	138	134	26	73	39	2.4	1.5	2.6
46	M	APTHOUS ULCER	149	50	74	72	22	110/70	76	164	126	25	91	48	2.5	1.7	1.6
60	F	HERPES SIMPLEX	157	54	72	70	21	110/80	81	192	122	24	125	43	1.4	1	2.4
52	F	KELOID	154	70	92	90	29	130/70	120	134	86	17	73	44	1.9	1.3	1.8

STER CHART - CASES

AGE	SEX	DURATION OF PSORIASIS	MARITAL HISTORY (M/UM)	VEGETARIAN/ NONVEGETARIAN	RURAL/ URBAN	FAMILY HISTORY	TYPE OF PSORIASIS	PSORIATIC ARTHRITIS /NAIL CHANGES	BSA SCORE	BLOOD GROUP	SOCIOECONOMIC STATUS	SMOKER	ALCOHOLIC	HT(cm)	WT (Kg)	CASES (PSORIASIS)	WC(cm)	BMI	BP(mmhg)	FBG(mg/dl)	T.C(mg/dl)	TGL (mg/dl)	VLDL (mg/dl)	LDL(mg/dl)	HDL(mg/dl)	FT3(pg/ml)	FT4(ng/dl)	TSH (mIU/ml)
60	M	10 YEARS	MARRIED(NCM)	NON VEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD - 3%**	A+VE	LOW	**YES**	**YES**	160	65	82	80	25	100/70	86	199	67	13	149	37	2.1	1.2	4.3
27	F	7 MONTHS	MARRIED(NCM)	VEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD - 3%**	O+VE	MIDDLE	NO	NO	150	58	78	76	25	110/70	80	136	128	25	73	38	3.4	1.3	2.3
37	F	20 YEARS	MARRIED (NCM)	NON VEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD - 3%**	A+VE	LOW	NO	NO	162	58	86	84	22	100/70	98	158	93	18	90	50	2.6	0.9	2.7
43	M	23 YEARS	MARRIED(NCM)	NON VEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD - 3%**	B+VE	MIDDLE	NO	NO	166	60	84	82	21	140/90	156	236	199	39	163	34	1.9	1	2.7
75	M	35 YEARS	UNMARRIED	NON VEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD - 3%**	A-VE	LOW	NO	NO	168	55	76	74	19	140/90	98	206	120	24	144	38	2.4	1.2	1.5
46	F	15 YEARS	MARRIED (IIIdegree CM)	NON VEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD - 3%**	B+VE	MIDDLE	NO	NO	156	69	82	80	28	140/90	146	250	252	50	175	25	2	1.6	3.6

44	F	10 YEARS	MARRIED(NCM)	VEGETARIAN	RURAL	NO	PLAQUE	**YES/NO**	**MILD-3%**	O+VE	LOW	NO	NO	164	60	80	78	22	110/70	78	191	110	22	112	57	2.9	1.3	6
70	F	50 YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/YES	**M0DERATE3-10%**	A+VE	LOW	NO	NO	156	66	76	74	27	140/80	184	220	279	55	128	37	2.6	1.1	6
45	M	15 YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	**NO/YES**	**MODERATE-3-10%**	A-VE	LOW	NO	NO	170	51	76	74	17	130/80	165	188	108	21	127	40	2.2	1.2	1.4
32	F	10 YEARS	MARRIED(NCM)	VEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	AB+VE	LOW	NO	NO	160	68	80	78	26	120/70	80	175	170	34	91	50	1.8	1.6	3.2
68	M	40 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	**NO/YES**	**MODERATE-3-10%**	O+VE	LOW	NO	NO	158	66	78	76	26	140/90	112	206	312	62	104	40	2.8	1.7	0.6
27	F	10 YEARS	UNMARRIED	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	LOW	NO	NO	148	56	76	74	25	130/80	80	174	108	21	103	50	2.2	1.4	4
45	M	25 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MODERATE-3-10%**	B+VE	LOW	**YES**	NO	164	55	78	76	20	110/70	80	256	304	60	158	38	2.7	0.8	1.6
25	F	15 YEARS	UNMARRIED	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	AB-VE	LOW	NO	NO	154	54	78	76	22	110/70	78	212	329	65	107	40	2	1	0.7
20	F	10 YEARS	UNMARRIED	VEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MODERATE-3-10%**	A-VE	MIDDLE	NO	NO	164	59	76	74	21	100/70	76	186	98	19	132	35	2.4	1.6	0.6

60	M	30 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	**NO/YES**	**MODERATE-3-10%**	A+VE	LOW	**YES**	**YES**	158	60	80	78	24	140/90	146	256	314	62	169	25	2.1	1	1
51	M	20 YEARS	UNMARRIED	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MODERATE-3-10%**	B+VE	LOW	NO	**YES**	162	52	76	86	19	140/100	142	170	134	26	94	50	2.8	1.6	4
34	M	6 YEARS	UNMARRIED	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	O-VE	LOW	**YES**	**YES**	160	62	80	78	24	120/70	138	220	240	48	142	30	1.9	1.7	2.2
24	M	14 YEARS	UNMARRIED	VEGETARIAN	URBAN	NO	PLAQUE	**NO/YES**	**MODERATE-3-10%**	B+VE	LOW	**YES**	**YES**	158	50	76	74	20	110/70	89	154	114	22	90	42	2.2	1.2	1.4
48	M	20 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	LOW	**YES**	**YES**	160	72	86	84	28	140/100	145	202	266	53	97	52	2	1.4	2.6
60	F	10 YEARS	MARRIED(III degree CM)	NONVEGETARIAN	URBAN	NO	PLAQUE	**NO/YES**	**MODERATE-3-10%**	A+VE	LOW	NO	NO	158	56	78	76	22	140/90	276	220	280	56	134	30	1.8	1.7	1.8
35	F	20 YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MODERATE-3-10%**	O+VE	LOW	NO	NO	148	50	90	90	22	120/70	158	162	278	55	74	33	2.6	1.6	0.7
28	F	5 YEARS	MARRIED(III degree CM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	A+VE	LOW	NO	NO	150	60	78	76	26	120/70	94	172	86	17	116	39	1.5	1.5	1.4
42	M	20 YEAR	MARRIED(NCM)	NONVEGETARIA	URBAN	NO	PLAQUE	**NO/YES**	**MODERATE-3-**	B+VE	MIDDLE	**YES**	NO	160	65	84	82	25	140/90	165	227	201	40	154	33	1.5	1.4	1.9

		S		N					**10 %**																		
46	M	25 YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MODERATE-3-10 %**	B+VE	LOW	**YES**	NO	160	68	84	82	26	130/80	97	108	92	18	67	23	1.4	0.9	5
21	F	10 YEARS	UNMARRIED	VEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	A+VE	MIDDLE	NO	NO	154	48	68	66	20	120/70	90	150	95	19	81	50	2.5	1.5	3
63	M	43 YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	**NO/YES**	**MODERATE-3-10 %**	AB+VE	MIDDLE	**YES**	**YES**	158	52	66	64	20	140/90	165	220	279	55	128	7	2.5	1.7	1.7
59	M	40 YEARS	MARRIED(NCM)	VEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MODERATE-3-10 %**	A+VE	LOW	NO	NO	165	78	88	86	28	150/90	134	250	217	43	160	47	1.3	0.9	4.4
35	M	20 YEARS	UNMARRIED	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	LOW	**YES**	NO	158	64	70	68	25	130/70	181	220	203	40	150	30	1.3	1.6	1.4
33	F	10 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	O+VE	MIDDLE	NO	NO	152	50	66	64	21	130/70	97	207	173	34	126	47	2.4	1.1	3.7
54	M	30 YEARS	MARRIED(NCM)	VEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	LOW	NO	NO	158	70	80	78	28	140/90	158	253	215	43	170	40	3.6	1.8	0.3
60	F	10 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	**NO/YES**	**MODERATE-3-10 %**	B+VE	LOW	NO	NO	154	70	78	76	29	140/90	150	268	198	39	194	35	1.6	1	2.2

31	M	10 YEARS	UNMARRIED	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	O+VE	LOW	**YES**	**YES**	150	56	70	68	24	110/80	96	153	242	48	80	25	2.5	2	2.4
40	M	20 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	A+VE	LOW	**YES**	NO	160	81	90	88	31	140/90	156	265	159	31	194	40	2.5	1.2	3.2
40	M	3 YEARS	MARRIED(NCM)	VEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	B-VE	MIDDLE	**YES**	NO	160	65	80	78	25	140/80	98	171	283	56	73	42	2	1.2	1.4
42	M	5 YEARS	MARRIED	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	MIDDLE	NO	NO	160	58	68	66	22	110/70	96	206	202	40	131	35	2.1	0.6	2.6
34	M	10 YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	A+VE	MIDDLE	**YES**	**YES**	164	80	88	86	29	140/90	130	216	335	67	119	30	1.8	2	2.9
65	M	40 YEARS	MARRIED(NCM)	VEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	B-VE	LOW	**YES**	NO	158	76	86	82	30	140/90	126	251	312	62	147	42	1.6	1	2.2
25	M	10 YEARS	UNMARRIED	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	LOW	NO	NO	155	45	58	56	18	110/70	74	179	153	30	100	49	2.3	1.3	5
54	M	4 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MODERATE-3-10%**	B+VE	LOW	**NO**	NO	164	88	92	90	32	120/70	116	282	332	66	186	30	0.4	0.2	8
48	M	20 YEARS	UNMARRIED	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	B-VE	LOW	**YES**	**YES**	156	45	62	60	18	120/70	76	206	175	35	131	40	2.2	1.6	3.8
49	M	10 Y	MARRIED(	NONVEG	URB	NO	PLAQUE	NO/	**MODER**	O+V	LOW	NO	NO	162	60	72	70	22	110	98	276	326	65	187	24	1.2	1.8	3

		EARS	NCM)	ETARIAN	AN			NO	**ATE-3-10%**	E								/70										
35	F	15YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	LOW	NO	NO	158	60	70	68	24	120/70	76	179	235	47	88	44	2.5	1	1.2
60	M	25YEARS	MARRIED	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MODERATE-3-10%**	A+VE	LOW	**NO**	NO	155	76	88	86	31	140/100	98	225	331	66	125	34	1.5	1.4	1.9
43	M	5YEARS	MARRIED	NONVEGETARIAN	URBAN	NO	PLAQUE	**NO/YES**	**MODERATE-3-10%**	B-VE	LOW	**YES**	**YES**	165	70	88	86	25	140/70	151	256	314	62	145	49	1.4	1.2	7.5
39	M	5YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	**ERYTHRODERMIC**	NO/NO	**SEVERE>10%**	B+VE	MIDDLE	NO	NO	156	60	70	68	24	130/80	82	254	368	73	126	55	2.3	1.4	3.7
35	M	2YEARS	UNMARRIED	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MODERATE-3-10%**	A+VE	LOW	NO	NO	162	70	88	86	26	120/70	86	149	233	46	64	39	1.8	1.8	2.3
60	M	20YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	B-VE	LOW	NO	NO	156	68	78	76	27	140/100	91	177	324	64	81	32	2.1	1.6	6
63	F	2YEARS	UNMARRIED	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MODERATE-3-10%**	O-VE	LOW	NO	NO	154	64	76	74	26	110/70	115	207	163	32	128	47	1.6	1.4	3.3
22	F	2YEARS	UNMARRIED	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	O+VE	MIDDLE	NO	NO	154	65	76	74	27	100/70	76	156	143	28	83	45	1.1	0.6	6

60	M	25 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	LOW	**YES**	NO	158	54	76	74	21	130/90	77	206	111	22	141	43	1.9	1.6	2.4
60	M	30 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	**NO/YES**	**MODERATE-3-10%**	A-VE	MIDDLE	**YES**	**YES**	160	68	80	78	26	140/90	79	169	89	17	124	28	3.3	1.4	7
28	F	8 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	MIDDLE	NO	NO	156	50	76	74	20	110/70	79	177	116	23	127	27	3.5	1.4	3
41	M	10 YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MODERATE-3-10%**	A+VE	MIDDLE	NO	NO	168	69	84	82	24	130/80	72	188	112	22	123	43	3.3	1.5	2.1
65	M	40 YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	**NO/YES**	**SEVERE >10%**	A+VE	LOW	**YES**	**YES**	158	60	80	78	24	110/70	135	225	300	60	130	35	2.1	1.4	5
50	M	5 YEARS	UNMARRIED	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MODERATE-3-10%**	O+VE	LOW	**YES**	**YES**	162	70	80	78	26	140/90	87	152	135	27	100	25	2.7	1.5	3.2
27	F	3 YEARS	UNMARRIED	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MODERATE-3-10%**	B+VE	LOW	NO	NO	158	56	78	76	22	120/70	88	195	154	30	134	31	3.2	1.3	7.6
45	F	10 YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	LOW	NO	NO	156	55	78	76	22	110/70	96	180	152	30	110	40	3.3	1.5	3.6
62	M	25 YEARS	UNMARRIED	NONVEGETARIAN	URBAN	NO	PLAQUE	**NO/YES**	**MODERATE-3-10**	A+VE	LOW	NO	NO	160	62	82	80	24	100/70	86	175	160	32	103	40	2.7	1.4	4.6

									%																		
71	M	20 YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/YES	**MODERATE-3-10 %**	B+VE	LOW	NO	NO	156	50	76	74	20	110//70	90	249	110	22	181	46	2.5	0.5	5.2
57	F	7 YEARS	MARRIED	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	LOW	NO	NO	145	60	86	84	28	150/90	129	255	62	12	196	47	1.8	1.3	2.3
55	M	25 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**SEVERE >10 %**	A-VE	LOW	YES	NO	162	60	86	84	22	130/70	86	150	130	26	89	35	2.6	1.4	3
28	F	1 YEAR	UNMARRIED	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	O+VE	LOW	NO	NO	158	60	80	78	24	110/70	90	154	110	22	92	40	2.4	1.4	3
40	M	10 YEARS	MARRIED(NCM)	VEGETARIAN	URBAN	NO	PLAQUE	**YES/YES**	**SEVERE >10 %**	B+VE	MIDDLE	NO	NO	160	62	88	86	24	110/70	101	146	200	40	66	40	2.3	0.9	5
58	F	20 YEARS	MARRIED(III degree CM)	NONVEGETARIAN	URBAN	NO	PLAQUE	**NO/YES**	**MODERATE-3-10 %**	B+VE	LOW	NO	NO	152	58	72	70	25	110/80	83	201	105	21	140	40	1	0.6	7.2
44	M	4 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MODERATE-3-10 %**	A+VE	MIDDLE	NO	NO	156	50	68	66	20	110/70	108	213	182	36	137	40	1.2	0.7	6.7
55	M	5 YEARS	UNMARRIED	VEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	LOW	NO	NO	168	60	86	84	21	120/70	118	260	180	36	186	38	2	1.6	3.8
35	M	2 YEAR	UNMARRIED	VEGETARIAN	RURAL	NO	PLAQUE	**NO/YE**	**MODERATE-**	O-VE	LOW	NO	NO	157	60	86	84	24	130/9	86	205	150	30	135	40	2.2	1.2	6.5

		S						**S**	**3-10 %**									0									
52	M	4 YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	**NO/YES**	**SEVERE >10 %**	A+VE	MIDDLE	NO	NO	164	60	86	84	22	120/70	120	237	131	26	173	38	2	1.5	5.5
52	M	1 YEAR	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	O+VE	LOW	NO	NO	162	56	70	68	21	130/80	89	180	119	23	112	45	1.3	1	3.2
59	F	6 MONTHS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MODERATE-3-10 %**	A+VE	LOW	NO	NO	160	61	78	76	23	120/70	128	162	114	22	108	32	0.9	1.2	4
46	F	10 YEARS	MARRIED(IIIdegree CM)	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	LOW	NO	NO	171	65	88	86	22	120/70	136	212	130	26	151	35	1	0.4	7
25	F	5 YEARS	UNMARRIED	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	A-VE	LOW	NO	NO	152	45	74	72	19	110/60	98	209	135	27	136	46	2	1.4	3.6
65	F	25 YEARS	UNMARRIED	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	A+VE	LOW	NO	NO	160	62	86	84	24	120/70	90	230	170	34	156	40	2.4	1.8	3.5
24	F	5 YEARS	UNMARRIED	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MODERATE-3-10 %**	A+VE	LOW	NO	NO	162	48	76	74	18	130/70	86	170	130	26	99	45	3	1.2	3.5
33	F	10 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	O+VE	LOW	NO	NO	155	53	78	76	22	110/70	90	180	140	28	112	40	2	1.4	2.6
45	F	20 YEA	MARRIED(IIdegree	NONVEGETA	URBAN	NO	PLAQUE	NO/NO	**MODERATE-**	A+VE	LOW	NO	NO	158	42	70	68	16	100/7	98	168	130	26	102	40	2.2	1.8	4

		RS	CM)	RIAN					**3-10 %**									0									
56	F	30YEARS	UNMARRIED	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	LOW	NO	NO	150	60	84	82	26	140/90	130	256	165	33	188	35	2	1	6.7
34	F	8YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MILD-3%**	A+VE	LOW	NO	NO	162	55	82	80	20	110/70	96	158	135	27	86	45	1.8	1	7.4
37	F	7YEARS	MARRIED(IIIdegreeCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	A+VE	LOW	NO	NO	156	50	80	78	20	110/70	98	180	120	24	111	45	2.6	1.8	4
63	F	10YEARS	MARRIED(IIIdegreeCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	B-VE	LOW	NO	NO	152	54	78	76	23	130/70	136	270	160	32	198	40	1.8	1.4	4.3
40	F	20YEARS	MARRIED(NCM)	VEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MODERATE-3-10 %**	B+VE	MIDDLE	NO	NO	156	60	80	78	24	140/90	87	225	130	26	157	42	1.8	1.2	7
45	F	20YEARS	UNMARRIED	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	A+VE	MIDDLE	NO	NO	160	56	78	76	21	110/70	92	190	152	30	118	42	2	1.8	3.6
45	F	20YEARS	MARRIED(IIIdegreeCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	**NO/YES**	**MILD-3%**	A+VE	LOW	NO	NO	156	50	80	78	20	110/70	90	162	125	25	97	40	1.6	0.8	7.2
43	M	20YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	B+VE	LOW	NO	NO	160	56	86	84	21	110/80	96	180	140	28	112	40	1.6	1	3.8
41	F	20YEAR	MARRIED(NCM)	NONVEGETARIA	URBAN	NO	PLAQUE	NO/NO	**MILD-3%**	O-VE	LOW	NO	NO	158	60	86	84	24	110/70	92	170	126	25	105	40	1.6	1.2	7.2

		S		N																							
30	F	13 YEARS	MARRIED(IIIdegree CM)	VEGETARIAN	URBAN	NO	PLAQUE	**YES/YES**	**SEVERE >10 %**	O+VE	MIDDLE	NO	NO	156	54	76	74	22	120/70	98	158	110	22	91	45	2	0.6	5.8
45	F	20 YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MODERATE-3-10 %**	B+VE	LOW	NO	NO	160	55	76	74	21	110/70	96	165	120	24	96	45	1.6	0.8	3.2
57	F	30 YEARS	MARRIED(IIIdegree CM)	NONVEGETARIAN	URBAN	NO	PLAQUE	**NO/YES**	**MODERATE-3-10 %**	A-VE	LOW	NO	NO	158	65	88	86	26	130/70	116	200	150	30	130	40	0.8	0.6	7.6
60	F	40 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	**NO/YES**	**MILD-3%**	B+VE	LOW	NO	NO	158	55	76	74	22	110/70	108	175	130	26	109	40	1.6	1.2	2.8
45	M	25 YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	**NO/YES**	**MODERATE-3-10 %**	A+VE	LOW	**YES**	**YES**	160	58	78	76	22	110/80	90	141	203	40	66	35	2.4	1.4	1.1
55	F	20 YEARS	MARRIED(II degree CM)	NONVEGETARIAN	RURAL	NO	PLAQUE	**YES/YES**	**SEVERE >10 %**	O+VE	MIDDLE	NO	NO	163	68	82	80	25	130/90	98	173	86	17	117	39	2.9	1.6	2.6
48	F	10 YEARS	MARRIED(NCM)	NONVEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MODERATE-3-10 %**	A+VE	MIDDLE	NO	NO	158	55	78	76	22	120/70	86	154	114	22	90	42	1.3	1.6	2.6
50	M	30 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**SEVERE >10 %**	B+VE	LOW	**YES**	**YES**	164	90	106	104	24	150/90	153	186	98	19	136	34	2.8	1.6	3

52	F	25 YEARS	MARRIED(NCM)	VEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MODERATE-3-10 %**	O+VE	MIDDLE	NO	NO	155	48	76	74	19	110/70	90	175	145	29	101	45	2.5	1.4	2.8
56	F	10 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/NO	**MODERATE-3-10 %**	B+VE	MIDDLE	NO	NO	158	56	78	76	22	140/90	170	206	162	32	140	34	2.2	1.4	1.3
50	F	24 YEARS	UNMARRIED	NONVEGETARIAN	RURAL	NO	PLAQUE	**NO/YES**	**MILD-3%**	O-VE	LOW	NO	NO	162	65	86	84	24	120/80	97	178	108	21	107	50	2.1	0.6	2.6
56	M	4 YEARS	MARRIED(NCM)	VEGETARIAN	URBAN	NO	PLAQUE	NO/NO	**MODERATE-3-10 %**	A+VE	MIDDLE	NO	NO	160	58	84	82	22	110/70	78	188	201	40	113	35	2.8	1.6	2.6
54	F	30 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	NO/YES	**MILD-3%**	B+VE	MIDDLE	NO	NO	162	54	80	78	20	100/70	80	198	126	25	127	46	2.4	1.3	2.2
43	M	10 YEARS	MARRIED(NCM)	NONVEGETARIAN	RURAL	NO	PLAQUE	**YES/YES**	**SEVERE >10 %**	A+VE	LOW	YES	YES	156	52	72	70	21	140/100	145	154	165	33	91	30	2.8	1.4	2.2

CASE REPORT FORM

TITLE: " A study of metabolic syndrome in psoriatic patients"- proforma:

Name : **Date:**

Age/Sex : **OP/IP NO:**

Education :

Occupation :

Place :

Socioeconomic status : **Low/Middle/High**

Chief complaints :

Menstrual History:

Cycle Duration -

Last Menstrual Periods **-**

Marital History:

Duration **-**

Consanguinity -

Obstetric History:

Number of children:

Past History:

DM/HT/Treatment/Drugs

Personal History:

Family History:

Concurrent illness:

General examination:

Ht:	Wt:	BMI:
PR:	BP:	Thyroid:
Hip circumference:		Waist circumference:

Study specific physical examination :

Skin:

Morphology of the psoriatic lesion:

Plaque

Pustule

Guttate

Erythrodermic

Distribution of the lesion :

Scalp

Face

Trunk

Extremities

Palms

Soles

Flexural

BSA Score (Body Surface Area Score)

Mild	**3%**
Moderate	**3-10%**
Severe	**>10%**

Nail Changes : (+)/ (-)

Psoriatic Arthritis : (+)/ (-)

Systemic examination : CVS **:**

RS :

ABDOMEN :

CNS :

Investigations done :

Hb%

TC

DC

Platelets

Blood urea

Plasma glucose

Serum creatinine

Liver function test:

S. Protein A/G :

S. Bilirubin :

GGT :

AST :

ALT :

ALP :

Special investigations to be done :

Fasting blood glucose:

Lipid profile:

DATE	**VALUES**
S. T. CHOLESTEROL	
S. TGL	
S. VLDL	
S. LDL	
S. HDL	

Thyroid profile :

DATE	**VALUES**

FT3

FT4

TSH

Skin biopsy:

Investigator comment:

Signature of the principle investigator:

Chennai Medical College Hospital & Research Centre

Irungalur,Trichy – 621 105.

Consent Form

You are requested to participate in a study conducted in the Department of Biochemistry, Chennai Medical College Hospital & Research Centre, Irungalur, Trichy, Tamilnadu, titled (A study of Metabolic Syndrome in Psoriatic patients) "Estimation of blood gluose, serum lipid profile, .serum thyroid profile.Your participation in the study is voluntary.

- There will be no cost for participating in the study
- Your participation is not a compulsion
- You have the right to withdraw from the study at any time.

Nature of Study:

- If any abnormalities are identified, you will be informed for further consultation.
- The results of this study will be kept confidential.

We believe that the results of this study will be beneficial for advancements in medicine & Science. We assure you that we will not use these result for any other purpose.

Consent

I Mr. / Mrs. / Ms. _____ residing at _____
_____ on this day _____ after having read the consent form carrying information for the above mentioned study and I hereby give my consent to take 5 ml of my blood sample for the purpose of doing, "Estimation of blood gluose, serum lipid profile, .serum thyroid profile.

I was explained about the procedure in detail and give my consent for participating in the study and for using the results for Medical & Scientific purposes.

Signature of the participant **Signature of the Investigator**

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BIBILIOGRAPHY

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