METABOLIC SYNDROME AMONGST POLYCYSTIC OVARIAN DISEASE

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CHENNAI

BONAFIDE CERTIFICATE

This is to certify that the study entitled **"METABOLIC SYNDROME AMONGST POLYCYSTIC OVARIAN DISEASE is the bonafide work done by Dr. M.UMAMAHESWARI,** at the Institute of Obstetrics and Gynecology, in Government Hospital for Women's and Children attached to Madras Medical College, Chennai, from 2004-2007 under the guidance of **Prof. Dr. P.G.Sundaraman, M.D., DM (Endo).**

This dissertation submitted to **Dr. M.G.R. Medical University** is in partial fulfillment of the University by rules and regulations for the award of M.D. Degree in Obstetrics and Gynecology.

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Prof: Dr. Mohanambal, **M.D., D.G.O.,** **Institute of Obstetric and Gynecology** **Madras Medical College** **Chennai**	**Dean** **Madras Medical College** **Chennai — 600 003**

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INTRODUCTION

The most common form of anovulatory infertility is polycystic ovary syndrome[1]. The most typical form, the association of hyperandrogenism and chronic anovulation without specific underlying diseases of the adrenal or pituitary glands.

The principle underlying disorder is insulin resistance resulting in hyper insulinemia and hyperandrogenism[2,3] in later on life results in metabolic Syndrome which predisposes to cardiovascular disease and NIDDM in later life[4].

Metabolic syndrome is a combination of several factors which may share a common etiology and each of which is a risk factors for cardiovascular disease.

The clustering of Insulin resistance, dysglycemia, dyslipidemia and hypertension was originally defined as syndrome X in 1988[5]

Metabolic syndrome includes general obesity (as reflected by BMI defined as wt in kg/ht in m^2 central obesity as reflected by waist circumference or waist hip ratio) dyslipidemia (as reflected by Low HDL and High TGL levels) hyperglycemia, high blood pressure and resistance to action of insulin.

The risk of diabetes and cardiovascular disease with polycystic ovarian syndrome necessitates the awareness to detect it at an earlier stage. The identification of these insults in the preclinical state will lead on to delay in clinical manifestation and the postponement of metabolic morbidity.

The current study focuses this problem to identify early markers and to set guidelines for defining the metabolic syndrome among PCOS patients.

AIM OF THE STUDY

To study the significance of:

1. Anthropometric Measurements
2. Insulin Resistance (Fasting Glucose)
3. Lipid Profile (TGL/HDL Ratio)

4. Hypertension (Blood Pressure) in Polycystic ovarian disease.

MATERIALS AND METHODS

Patients who attended the Endocrinology OPD, Institute of Obstetric and Gynecology with history and clinical examination suggestive of polycystic ovaries were analyzed and those who fit in inclusion criteria were taken for study numbering one hundred and nine.

A control group consisting of hundred women were also studied. The group had spontaneous, regular cylical menstrual flow and with out hyperandrogenic features. All were euthyroid and euprolactinemic.

Study and Control group were in the age group 16-35.

Period of study was between Oct 2005 to Sep 2006.

Inclusion Criteria:

1. Less than 40 years
2. USG findings of Polycystic ovaries
3. Hyperandrogenic features
4. Menstrual disorders.

Exclusion Criteria:

1. Prior known endocrine or medial disorder
2. Patients with chronic infection
3. Patients on hormone therapy.

A detailed menstrual history was taken. History regarding obesity, acne, hirsutism and infertility was taken.

Family History of Diabetes Mellitus / Hypertension / Polycystic ovarian disease, history suggestive of obesity, irregular cycles, infertility was enquired. History of PIH / GDM/Recurrent pregnancy loss) secondary infertility enquired.

Physical Examination:

A thorough clinical examination including cardiovascular, Respiratory and Central nervous system were done.

Measurement of height was done with barefoot and medial malleoli touching.

Weight was measured by a standard weighing scale.

Body mass index was calculated

Body mass index: $\frac{\text{Weight in kg}}{\text{Height in } M^2}$

Waist and hip measurements in cms were taken. Waist is measured at the narrowest point between highest point between iliac crest. Hips are measured at the maximal points of buttocks in cms. Waist / Hip ratio of 0.85 and above were taken as clinically significant.

Waist circumference is taken and more of 88cms is predictive of abnormality.

Blood pressure was recorded using standard sphygmomanometer in right upper limb in sitting posture.

Detailed physical examination for evidence of hyperandrogenism was done.

Features of hyperandrogenism included:

1. Hirsutism
2. Acne

3. Temporal Recession

4. Clitoromegaly

5. Masculinization

Hirsutism was assessed by Ferriman-Gallway Scoring F and G score of 9 and above were diagnostic.

Breasts were examined for evidence of galactorrhoea.

Ultra sound evidence of pelvic visera was done for diagnosis of polycystic ovarian disease using L and T ultrasound scanner.

Tran abdominal scanning with full bladder using linear transducer was done.

Uterine measurements was taken and endometrial thickness was accurately measured.

All regularly menstruating women were scanned during the follicular phase and those with irregular menstruation and secondary amenorrhea were scanned at random.

Criteria used for diagnosing polycystic ovaries are .

ADAMS CRITERIA : More than eight peripherally oriented cysts or less than 10mm surrounded by increased stromal mass and ovarian volume of more than nine cc.

Ovaries were scanned in transverse as well as linear phase to as to obtain length width and thickness of each ovary. Ovarian volume was calculated using the formula 0.523 x Length x Width x Thickness.

Biochemical and Hormonal Profile:

Blood for lipid profile, fasting glucose, hormonal status were taken in the morning with subjects in about 12 hrs fasting state. Venous blood was obtained and sent for biochemical and hormonal studies.

Lipid profile and blood glucose measurement:

Fasting blood glucose and lipid profile were done in Department of Biochemistry MMC using commercially available kits.

Total Cholestrol, HDL, and triglycerids were estimated by enzymatic methods.

Hormonal Assay: TSH, Prolactin were done in the department of endocrinology using radioimmuno assay technique. Of the 109 study group eight patients had elevated TSH level (more than 5 IU/l) and 1 had elevated

prolactin (more than 30 mg/ml) and hence 9 were excluded from further study and analysis.

THE ROTTER-DAM REVISED DIAGNOSTIC CRITERIA OF POLYCYSTIC OVARY SYNDROME:

1990 Criteria (both 1 and 2)

1. Chronic anovulation and

2. Clinical and/or biochemical signs of hyperandrogenism and exclusion of other etiologies

Revised 2003 criteria (2 out of 3)

1. Oligo – or anovulation

2. Clinical and / or biochemical signs of hyperandrogenism

3. Polycystic ovaries and exclusion of other etiologies (congenital adrenal hyperplasia, androgen-secreting tumors, Cushing's syndrome).

   The revised criteria having sufficient sensitivity and specificity to define PCO are "presence of 12 or more follicles in each ovary measuring 2-9 mm in diameter and increased ovarian volume >10 ml

REVIEW OF LITERATURE

In 1935, Irving F.Stein and Michael L. Leventhal first described a symptom complex associated with anovulation[6]. It was later called as STEIN-LEVENTHAL SYNDROME.

The PCOS is the commonest endocrine disturbance affecting women. The Syndrome is denoted by combination of hyperandrogenism and ovulatory dysfunction, with enlarged polycystic ovaries by ultrasound, in the absence of non-classical adrenal hyperplasia, Cushing's syndrome and androgen producing tumors.

The Characteristic polycystic ovary emerges when a state of anovulation persists for any length of time.. The polycystic ovary is the result of a functional derangement, not a specific central or local defect (Leon Speroff, 1999.[7]

HORMONAL MILIEU IN PCO IS AS FOLLOWS:

In patients with persistent anovulation, the average daily production of estrogen and androgen is both increasesd and dependent upon LH stimulation. This is reflected in higher circulating levels of Testosterone, Androstenedione, Dehydroepiandrosterone, DHEAS, 17 (OH) Progesterone and Estrone (De Van GW, 1975). The Testosterone, Androstenedione and

DHEA are secreted directly by the ovary while DHEAS is almost exclusively an adrenal contribution [8].

In PCOS, the ovaries donot secrete enough Estradiol, the level of which is equivalent to early follicular phase concentrations. The increased Estrogen is due to peripheral conversion of increased amounts of Androstenedione to Estrone. The consequence of raised Estrogen levels results in alteration in H-P-O axis.

Clinical Consequences of Persistent anovulation[9]

1. Reproductive Morbidities associated with PCOS are

- Infertility
- Oligoamenorrhoea
- Increased Pregnancy loss
- Increased PIH, Diabetes mellitus during pregnancy
- Endometrial carcinoma

2. Hyperandrogenic features

- Acne
- Oily skin
- Hirsutism

3. Other metabolic morbidity in later life

- Diabetes mellitus

- Hypertension
- Dyslipidemia
- Coronary artery disease, 7 fold increased risk for infarction.

Insulin resistance is defined as decreased glucose response to a given amount of insulin. Resistance to insulin stimulated glucose uptake is a relatively common phenomenon but is only one component of a condition called metabolic syndrome[10]

CRITERIA FOR METABOLIC SYNDROME WITH POLYCYSTIC OVARIAN DISEASE

(Three out of Five qualify for the syndrome (NCEP)[11]

1. Hypertension: B.P. more than 130/85 mm/Hg or higher
2. Abdominal obesity (waist hip circumference more than 88cm / waist hip ratio morethan 0.85)
3. Serum TG morethan 150mg/dl
4. Serum HDL lessthan 50mg/dl
5. Serum fasting glucose – 110 mg/dl or more.

Summary of 2003 polycystic ovary syndrome (PCOS) consensus regarding screening for metabolic disorders[12]. (Rotterdam)

Summary of Consensus:

1. No test of insulin resistance are necessary to make the diagnosis of PCOS, nor are they needed to select treatments.

2. Obese women with PCOS should be screened for the metabolic tests.

3. Further studies are necessary in nonobese women with PCOS to determine the utility of these tests, although they may be considered if additional risk factors of insulin resistance such as a family history of diabetes, are present.

   A large number of epidemiological studies have shown there is a relationship between low birth weight and subsequent risk of development of type 2 diabetes mellitus[13] . insulin resistance[14] and other features of metabolic syndrome such as hypertension[15].

   Fetal and Neonatal life are critical periods for development and growth of systems involved in the pathology of metabolic syndrome.

   Striking evidence show there is as much as 18 fold increase in the risk of developing the metabolic syndrome on comparing the lowest birth weight individuals with the highest birth weight individuals[16].

   DEVELOPMENTAL ORIGINS

   THRIFTY PHENOTYPE HYPOTHESIS:

This hypothesis status that in response to poor fetal nutrition the compromised fetus will adopt a number of strategies to maximize its channel of survival postnatally.

Firstly growth of brain is spared at the expense of other tissue such as muscle, kidneys and endocrine pancreas (Rudolph 1984) in addition metabolic programming is proposed to occur in the manner beneficial to survival under the conditions of poor postnatal nutrition. Foetus is programmed to store nutritions such as fat. Problems are proposed to occur if malnourished Foetus is born in conditions of adequate or over nutritions. This conflicts the earlier programming and obesity, type 2 diabetes and other features of metabolic syndrome.

Foetal Insulin Hypothesis:

An alternative hypothesis to explain the link between low birthweight and type 2diabetes was suggested by Hattersley and Tooke in 1999[17]. In this hypothesis, a genetic susceptibility to insulin resistance or beta cell dysfunction leads to retard growth in utero due to impaired insulin-mediated foetal growth and thus to reduced birthweight. This insulin-resistant or deficient phenotype would then show metabolic impairments in adulthood.

Rare mutations have been identified in the glucokinase gene that are associated with a low birthweight and the development of a rare monogenic form of diabetes; maturity onset diabetes of the young[18].

Recent studies have identified interesting gene-birth size interaction in the case of peroxisome proliferators-activated receptor gamma 2(PPARgama2) and polymorphisms[19].

Genes known to alter the risks of metabolic syndrome diseases are:

1. **CALPAIN-10** calpain-10 is a ubiquitously expressed cysteine protease important in the modifications of processing of proteins in the cell .calpan 10 is likely to be important in Beta cell apoptosis.

   Bairer[20] have show that calpain 10 polymorphisms are associated with insulin resistance. Lynn[21] have shown a correlation between Calpain 10 genotype and response to an oral glucose load.

   THE PPAR GAMA GENE

2. PPAR gama gene lodes for a protein that is a key controller of gene transcription in adipocytes. Severe mutations of the PPAR gama gene cause a disorder with many features of the metabolic syndrome22.

3. **The Kir 6.2 and SUR genes**

In pancreatic beta cells, ATP Sensitive Potassium channels (KATP) are critical components for the control of appropriate insulin secretion

4. The Hepatocyte Nuclear Facator 1 Alpha

HNF 1 Alpha is a transcription factor primarily expressed in liver but also in beta cell. Rare, severe mutations cause MODY. A common variant of the HNF 1Alpha gene predisposes to type 2 diabetes in the Oji-Cree population of Canada. This isolated native Indian population had highest prevalence of type 2 diabetes in the world (Hegele)[23].

5. The Hepatocyte Nuclear Factor 4 Gene

The chromosome (20q12-13.1) contains the HNF 4alpha gene (McCarthy and Gloyn et al 2003). P2 promoter controls the activation version of HNF 4 Alpha gene that it is a critical point of transcription factor that controls the differentiation and maintenance of beta cell phenotype[24] (Boj and Thomas).

6. The GCK GENE

A common variant in the promoter of the glucokinase (GCK) gene alters fasting glucose. Glucokinase is the glucose sensing enzyme of the beta cell (Matschinsky 1990). GCK mutation cause a relatively mild and stable increase fasting glucose concentration. (Stride and Hattersley 2002)[25].

OBESITY

Obesity can be classified as

- Gynoid type &
- Android type

Gynoid obesity refers to distribution in lower body – (Femoral and gluteal region)

Android obesity refers to central body distribution.

Whereas Gynoid fat is more resistant to catecholamines and sensitive to insulin.

Android fat is more sensitive to catecholamines and less sensitive to insulin and more active metabolically.

Android distribution is associated with,

- Hyperinsulinemia
- Impaired Glucose tolerance
- Diabetes mellitus
- Increase in androgen production rates

- Decrease in SHBG and
- Increase in free Testosterone and Estrogen

Android – Central body-obesity is associated with cardiovascular risk factors, including Hypertension and adverse cholesterol lipoprotein profiles ([26]).

Waist: Hip ratio had been used as a measure of estimating the degree of upper to lower body obesity, as the ratio accurately predicts the intraabdominal fat which is greater in android obesity [27].

However, studies have demonstrated that the more easily determined circumference of the waist is better predictor of android abdominal fat[28]

At any given level of BMI, an increase in android fat increases the cardio vascular risk.

A waist circumference greater than 90cm (35”) now 88cm in women in predictive of abnormal endocrinologic and metabolic function circumference>88 cm is most and is associated with an increased risk of cardiovascular morbidity as mentioned[29] .Waist hip ratio is strongly associated with metabolic syndrome. The WORLD HEALTH ORGANISATION clinical criteria for metabolic syndrome used BMI >30 or WHR interchangeably (WHO-1999)

BMI is calculated by $\frac{\text{Wt in kg}}{\text{Ht in M}^2}$

BMI $\geq$ 27 warrants treatment [30].

OLIGOMENORRHOEA:

Oligomenorrhoea is defined as infrequent and irregularly timed episodes of bleeding usually occurring at intervals of more than 35 days and upto 6 months.

HIRSUTISM:

The increased androgen leads on to Hirsutism and Acne. Hirsutism should be regarded as an increase in sexually stimulated terminal hair. Among the ovarian causes, PCOS, is the most common cause of androgen excess seconded by ovarian neoplasms.

Several scoring system are available to assess Hirsutism, the most clinically useful being that of Ferriman and Gallway (1961) was uses a score of 0-4 for each area of the body.[31] Score of >9 taken as significant.

HIRSUTISM – FERRIMAN GALLWAY SCORING SYSTEM

1. **Upper lip**

   1 – A few hairs at anterior margin

   2 – small moustache at anterior margin

   3 – Moustache extending halfway from outer margin

   4 – Moustache extending to midline

2. Chin

1 – A few scattered hair

2 – Small concentration of scattered hairs

3 – Light complete cover

4 – Heavy complete cover

3. Chest

1 – Circum areolar hairs

2 – Additional midline hairs

3 – Fusion of these areas with three quarter cover

4 – Complete cover

4. Upper Back

1 – Few scattered hairs

2 – Rather more, still scattered

3 – Light complete cover

4 – Heavy complete cover

5. Lower Back

1 – A Sacral tuft of hair

2 – With some lateral extension

3 – Three Quarter cover

4 – Complete cover

6. Upper Abdomen

1 – A few midline hairs

2 – Rather more still midline

3 – Half cover

4 – Full cover

7. Lower Abdomen

1 – A few midline hairs

2 – Midline steak of hair

3 – Midline band of hair

4 – Inverted ‘V’ shaped growth

8. Upper arm

1 – Sparse growth affecting not more that a quarter of limb surfaces

2 – More cover still incomplete

3 – Light complete cover

4 – Heavy complete cover

9. Thigh

1 – Sparse growth affecting not more than a quarter of limb surfaces

2 – More cover still incomplete

3 – Light complete cover

4 – Heavy complete cover

INSULIN RESISTANCE:

Insulin Resistance is defined as a reduced glucose response to a given amount of insulin.

A part from the association of insulin resistance with PCOS, Where defect is in post binding signaling pathways, rare clinical models with insulin resistance has been described. In those rare clinical models defects is in insulin receptor either due to point mutation (Type A syndrome) or due to autoantibodies against insulin receptor associated with autoimmune disease.

PCOS is associated with peripheral insulin resistance and hyperinsulinemia and the degree of both abnormalities is amplified by the presence of obesity. Studies had showed that women with PCOS have

peripheral insulin resistance similar in magnitude to that seen in patients with non-insulin dependent, diabetes mellitus (NIDDM)[32].

Hyperinsulinemia consequent to peripheral insulin resistance represents a mechanism to overcome the impaired insulin-mediated glucose use in women with PCOS. Obesity, commonly associated with PCOS (50% cases) has an additive negative effect on insulin resistance as indicated by the reduced hepatic insulin sensitivity in obese PCOS women beyond that associated with obesity alone [33].

Serine instead of Tyrosine phosphorylation is an “off” mechanism for glucose transport, but an ‘on” mechanism for P450 C17 enzyme activity [34]

Combination of increased androgen secretion and insulin resistance has been reported in both obese and non obese anovultory women[35]

Hyperandrogenism and hyperinsulinemia are not explained solely by obesity; However the presence of obesity adds the insulin resistance and hyperinsulinemia associated with obesity to that which is specifically unique to the anovulatory polycystic ovary state[36].

Fasting plasma glucose concentration depends primarily on the rate of hepatic glucose release which in turn is regulated by insulin concentration.

In addition, hyperandrogenism and insulin resistance are often associated with Acanthosis nigricans[37.]

Acanthosis nigricans is a grey-brown velvety sometimes verrucous, discoloration of the skin usually at the neck, groin, axillae and under the breast which is a marker for insulin resistance. It is most highly correlated with the magnitude of peripheral insulin resistance.

The state of chronic hyperinsulinemia represents a compensatory response to the target tissue problem. If the insulin levels necessary to suppress free fatty acid levels cannot be achieved, then the increase in free fatty acid leads to increased in hepatic glucose production and hyperglycemia.

Hyperinsulinemia leads to hypertension and an increased risk of Coronary heart disease[38]. Resistance to insulin is further associated with increased triglycerides, small dense lipoproteins and decreased HDL-Cholesterol levels, a potent bad combination that promote coronary artery disease[39].

Hyperinsulinemia act in genetically predisposed women as a “Second Hit" to unmask latent abnormalities in steroidogenesis 1997.

a). Ovary

b). Adrenal cortex and

c). Other organs

Paradoxically function as if responding to hyperinsulinemic state inspite of resistance to effects of insulin on glucose metabolism.

INSULIN RESISTANCE AT PUBERTY

Insulin resistance normally develops during the process of puberty in association with the rise in secretion of insulin, IGF-1, GH and gonadotropin.

PUBERTY

PHYSIOLOGICAL EVENTS

↑ Growth Hormone

↑ **Insulin Resistance**

↑ **Insulin Secretion**

↑IGF -1

↑IGFBP -1

↑SHBG

Hyperinsulinemia

↑ Bioavailability

↑ IGFS & Steroids

These changes may synergistically enhance gonadotropin action on the ovary. It contributes to the transition of hyperandrogenism from the adrenal to the ovary.

Insulin resistance and hyperinsulinemia appear to extend beyond puberty in women who have inherent cellular defects of insulin resistance and are thus destined to develop PCOS.

Although the adverse effect is present in anovulatory women with polycystic ovaries, hypertension is not encountered in these women until later in life, not in their reproductive years[40].

Hyperinsulinemia and polycystic ovaries are also associated with an increased production of Plasminogen Activator Inhibitory type (PAI – 1). Increased PAI – 1 levels are correlated with an increased risk of coronary heart disease, impaired fibrinolysis may effect vascular tissue changes associated with vascular disease.

LIPID PROFILE IN PCOS

Lipid profile in androgenized women with polycystic ovaries (who are also exposed to reltively low estrogen levels over time) in similar to the male pattern with higher levels of cholesterol, triglycerides and LDL Cholesterol and lower levels of HDL – Cholesterol, and this abnormal pattern is independent of body weight[41]

An adverse lipid profile is a distinguishing feature of these patients even when body mass index, insulin and age are controlled in case control studies[42].

Lipids and Lipoproteins:

The lipoprotein profile in obese women with PCOS is generally characterized by elevated plasma triglyceride and reduced high density lipoprotein (HDL) Cholesterol concentrations, wild 2002.[43]

A contributing factor to the abnormal lipid pattern in many of these patients is hyperinsulinemia.

Insulin resistance may be a more significant factor than androgens in determining the abnormal lipoprotein profile in overweight, anovulatory women.

The metabolic syndrome is accompanied by a 2 fold increase in risk of CVD and as fold increase in the risk of type 2 DM[44].

The aetiological mechanisms underspinning insulin resistance and reproductive abnormalities in women with PCOs are :

- Genetic Contributions to both reproductive and metabolic features[45]
- Defects in adipose tissue Lipolytic cascades [46]
- Inflammation Mediators leading to insulin resistance[47]

- Fetal programming effect[48]
- Primary ovarian hypersensitivity to insulin, leading firsty to altered hormonal milieu and then to alterations in body fat distribution (central > peripheral) with possible feedback towards greater insulin resistance.

Insulin resistance was seen at higher BMI level and was largely determined by increased Truncal-abdominal fat mass in PCOs 49 women with PCOs with 42 BMI matched control study of HOLTE (1994) [49]

In a study by Escobar- Morreale[50] 2004, has shown greater waist circumference or waist hip ratio in women with PCOs independent of BMI.

Recent study by Yildrim, Sabir and Kaleli[51] 2003 intraabdominal fat preperitoneal fat and subcutaneous fat thickness by USG found 2-fold higher visceral and preperitoneal fat thickness in women with PCOS despite near identical BMI and clinical subcutaneous fat content.

Women with PCOs accure more metabolic abnormalities with increasing BMI compared with women without PCOs which is generally in keeping with greater increments in insulin resistance with increasing BMI (Talbott[52] 2004. Aprindonindze)[53] 2004.

In a study by Dokras[54] 2005 found serum triglycerides / high density lipoprotein cholesterol (TG / HDL-C) ratio correlated with insulin resistance.

Serum TG /HDL-C > 3.2 had a high sensitivity and specificity for detection of metabolic syndrome in women with PCOS. The TG/HDL-C ratio may serve as a screening tool and needs to be prospectively validated.

DISCUSSION

Insulin resistance is linked to a Spectrum of metabolic abnormalities that can predate the onset of diabetes and cardiovascular disease by many years. Such abnormalities include lipid perturbance, hemostatic alteration, lowgrade chronic inflammation, high blood pressure endothelial dysfunction, body fat redistribution and glucose intolerance.

The Lipoprotein profile in PCOS women may result in Lipid alteration leading to metabolic disturbances, cardiovascular disease, endothelial dysfunction and hyperinsulinemia. In contrast to the caucasion population, Asian women have increased body fat content in spite of Low BMI. visceral adiposity serves as an important marker for future misery relating it to PCOS.

Our study highlights the importance of evaluating the markers of metabolic syndrome in PCOD women. BMI and visceral adiposity directly correlate with lipoprotein abnormalities. Insulin resistance, a close associate of metabolic syndrome is present in significant population amongst the study group. Hypertension has significant correlation.

In my study, prevalence of metabolic syndrome was higher in women with PCOS (66%) C.I 1.98,9.02 is comparable to the study done by ANUJA DOKRAS AND MELINDA(ACOG JULY/2005) where age adjusted prevalence of metabolic syndrome was 47.3%.

Elevated fasting glucose was detected least frequently among patients with PCOS in the study by Dokras and Bradley was 12.6 ± 4.0% and these were not significantly different compared with their control group.

In my study, Fasting blood sugar was found to be elevated in 6% of PCOD compared to control group (2%)

- In most common abnormality in PCOS group in my study was increased BMI and WHR> .85 was 74%, Low HDL 39% and high triglycerides 63% which is quite comparable to similar study done by Dokras & Melinda. BMI –(72.3%) Low HDL-C (63.7%) and high Triglycerides 46.8%.
- The high incidene of metabolic syndome could be explained to be due to variation in the ethnicity, Racial, genetic and environmental factors which is found to be more in South Asian when compared to Europeans.

CONCLUSION

PCOS, a clinical entity is a complex association of metabolic disturbances expressing in different ways both clinically and chemically. The

focus of our study is to unreveil its association. Compared to the western literature, PCOS, has profoundly present in our Asian population.

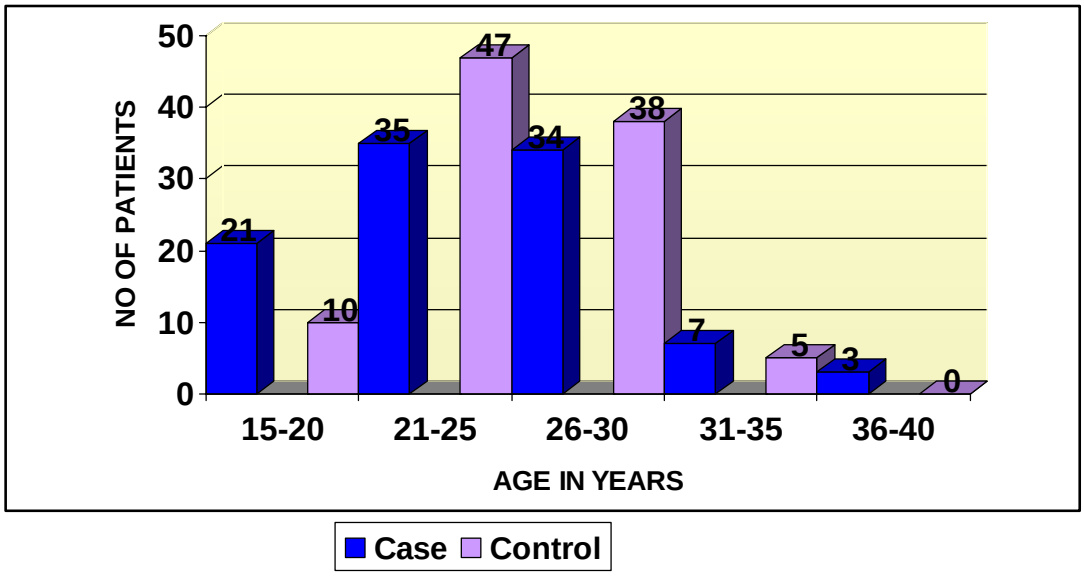
Body weight has direct correlation with the presence of PCOS as weight, BMI, waist hip ratio increasing which tells us the importance of documenting anthropometic measurements. Clinical scenerio inclusive of Hypertention, fasting lipid abnormality goes hand in hand amongst PCOS Patients.

The Significane of elevated fasting glycemic status was not appreciably present which precludes the importance of documenting post glucose challenge which will unmask the impairment of glucose tolerance.

The role of treating physician is expanded in cases of polycystic orarian syndrome after realising its close association whith chemical abberations resulting in the Misery of Systemic abnormalities. Till we understand the real etiological factor which result in PCOS, the responsibility to detect and treat the tentacles of morbid associations as highlighted in our study.

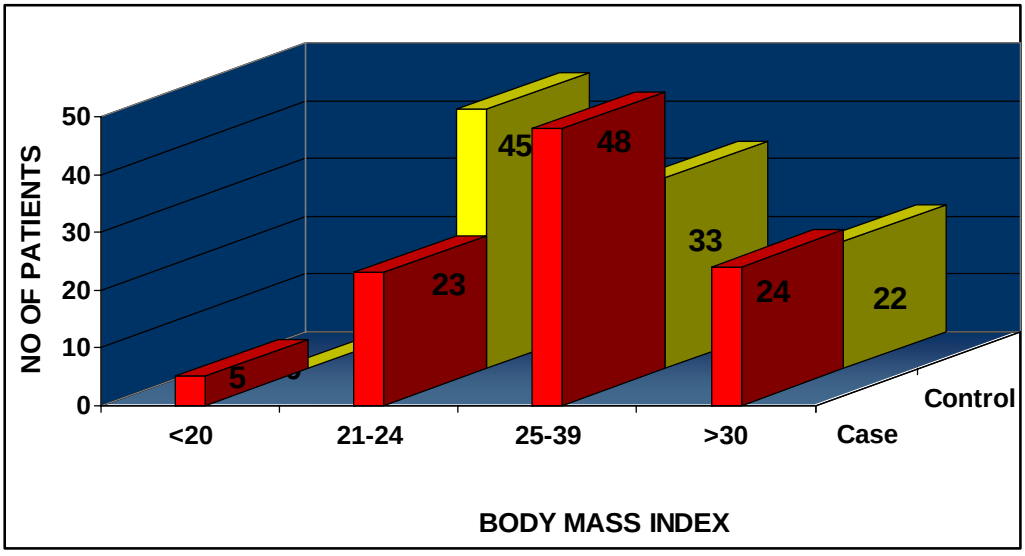
1.AGE DISTRIBUTION

OBSERVATION AND ANALYSIS

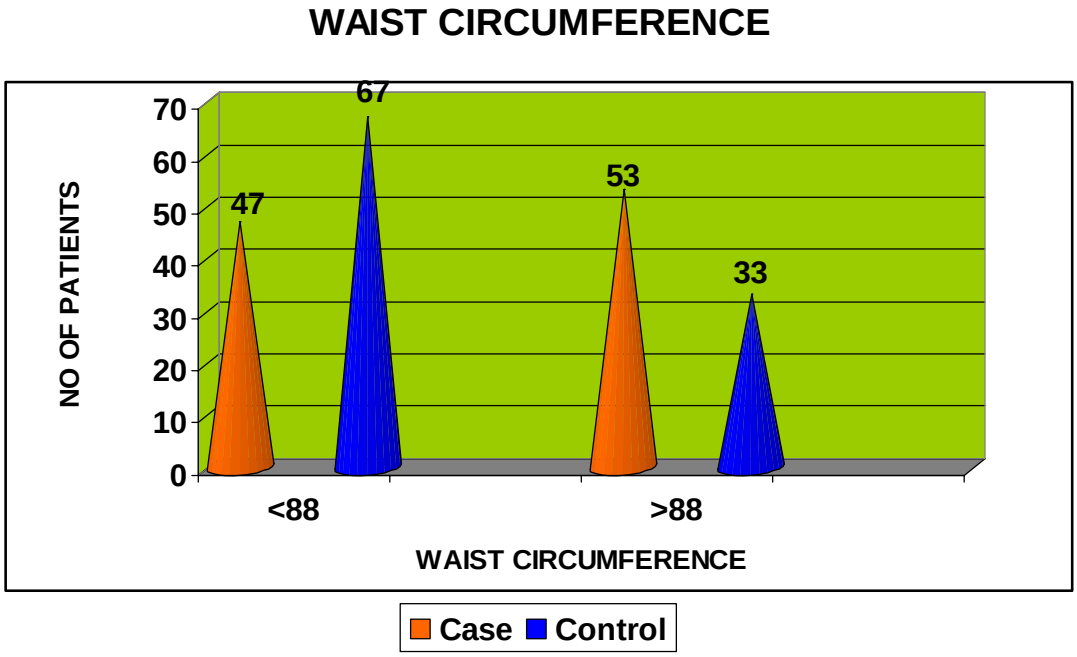


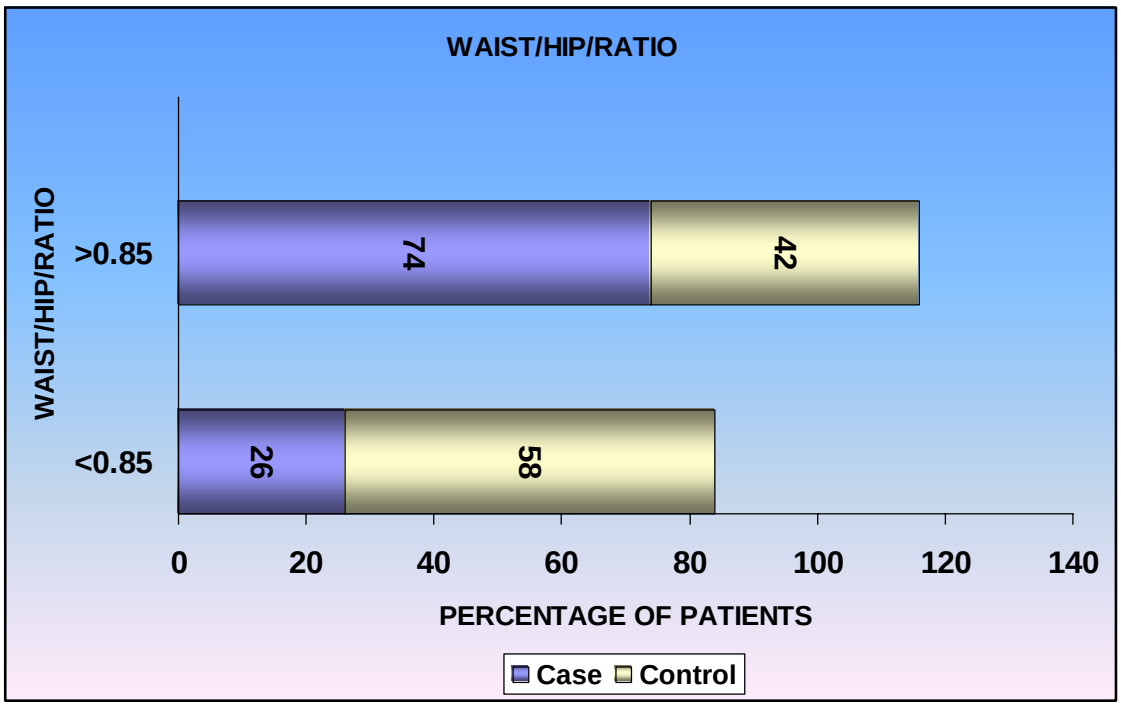
2. BMI DISTRIBUTION

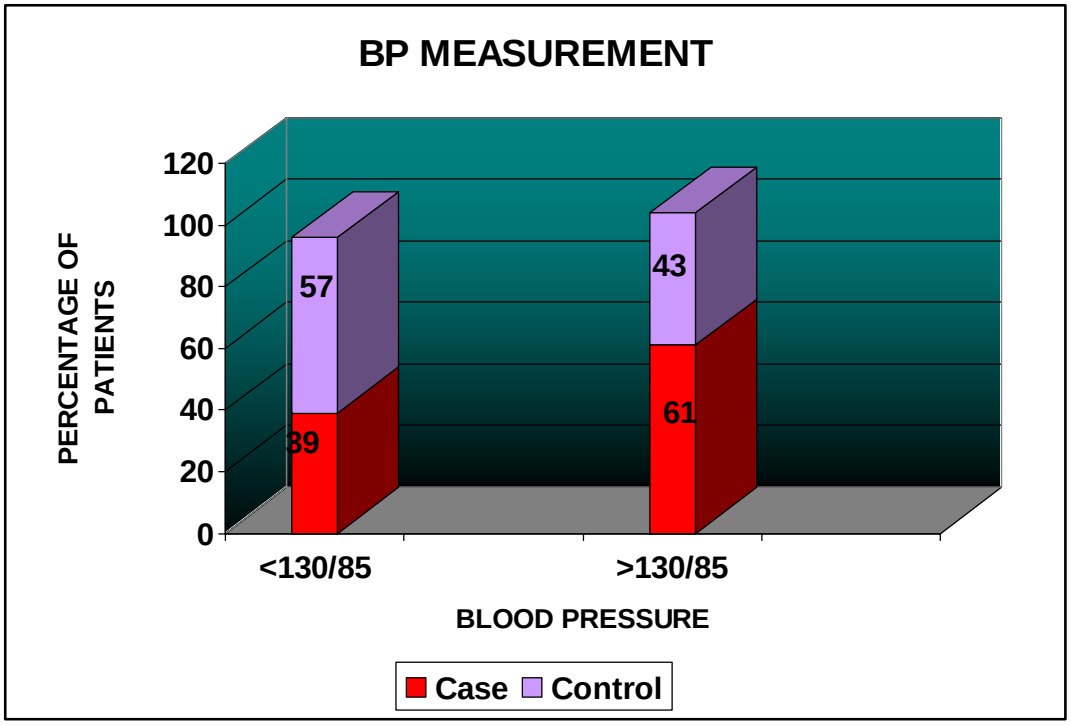
BODY MASS INDEX DISTRIBUTION



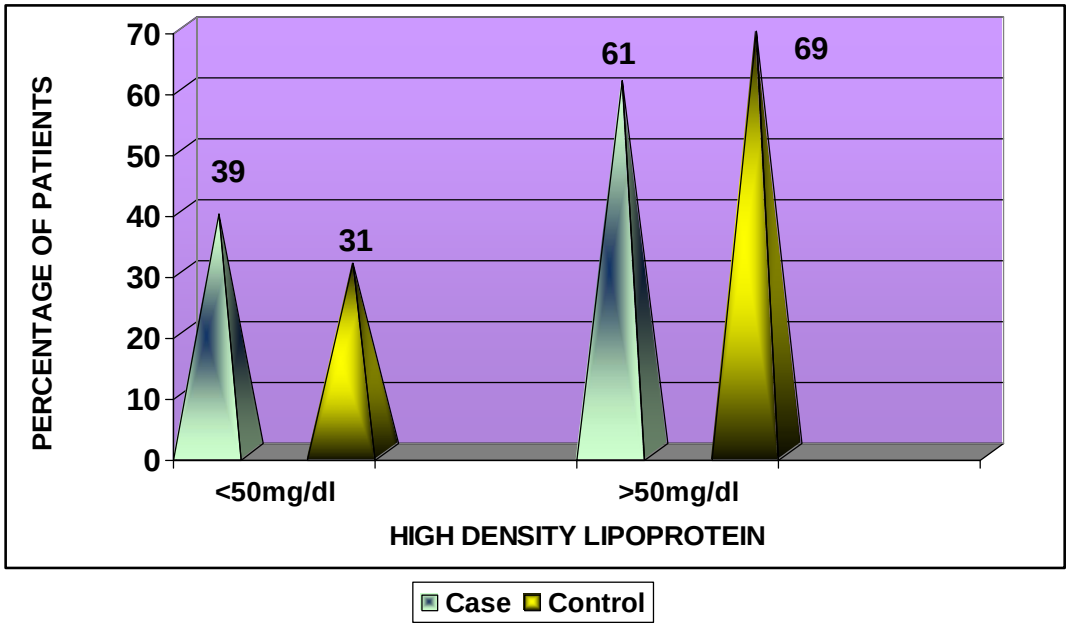
3. WAIST CIRCUMFERENCE

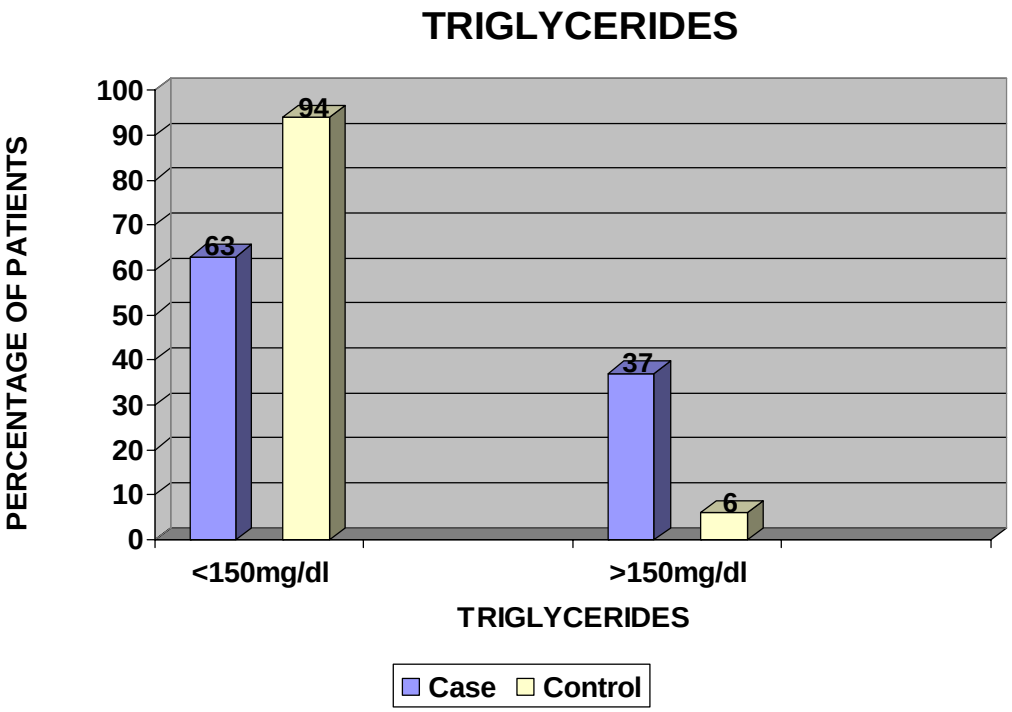


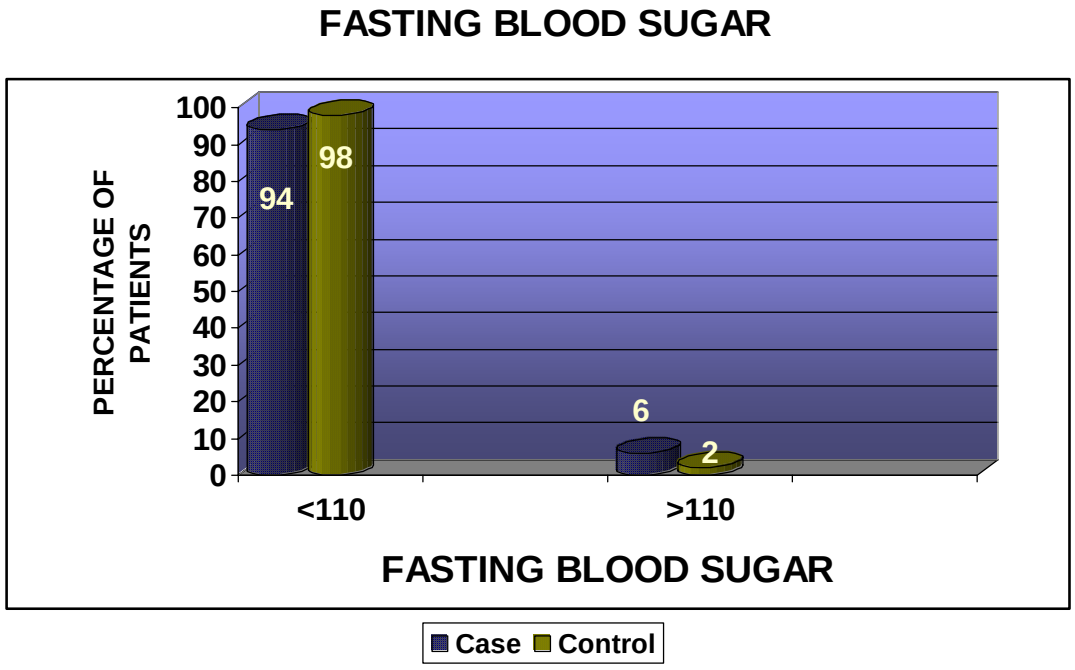




HIGH DENSITY LIPOPROTEIN




TRIGLYCERIDES
PERCENTAGE OF PATIENTS
100
90
80
70
60
50
40
30
20
10
0
63
94
37
6
<150mg/dl
>150mg/dl
TRIGLYCERIDES
Case
Control


FASTING BLOOD SUGAR
PERCENTAGE OF PATIENTS
100
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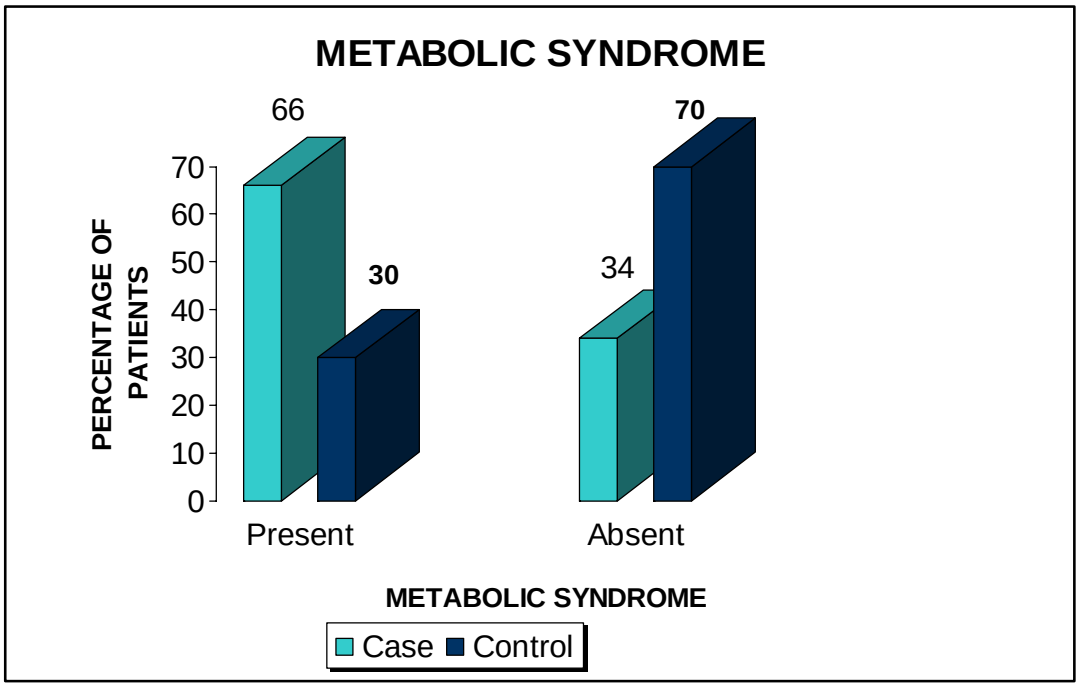
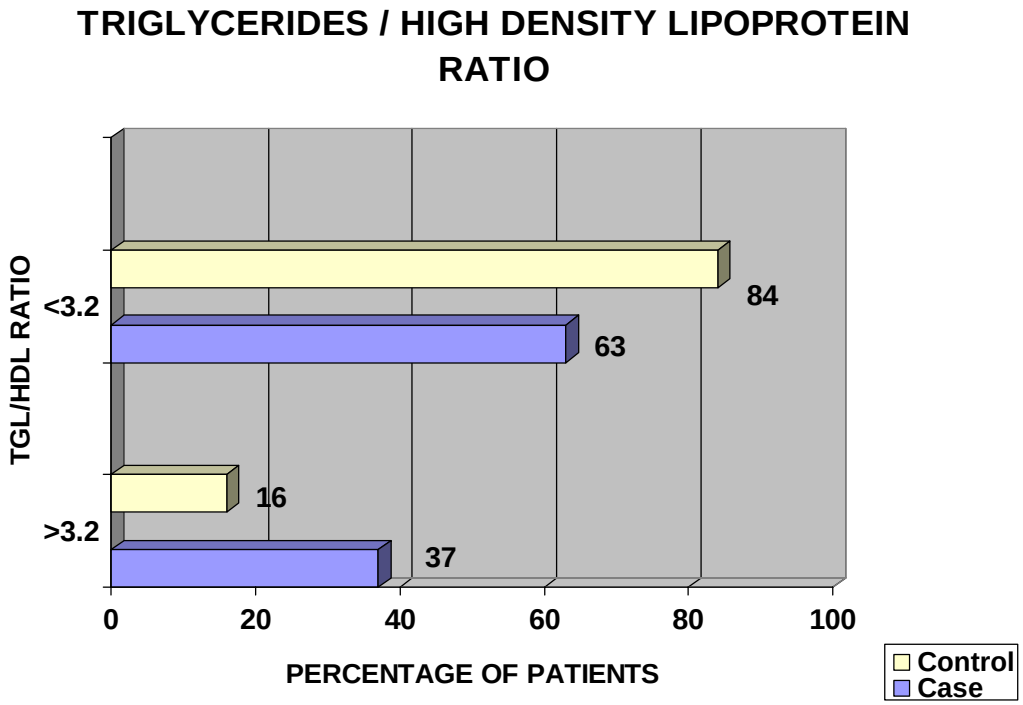
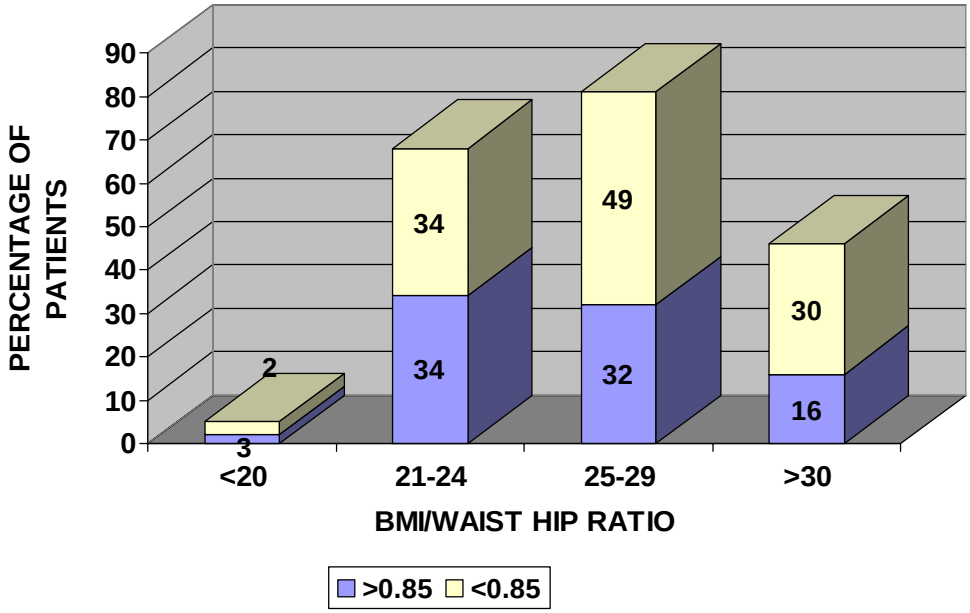
2
<110
>110
FASTING BLOOD SUGAR
Case
Control

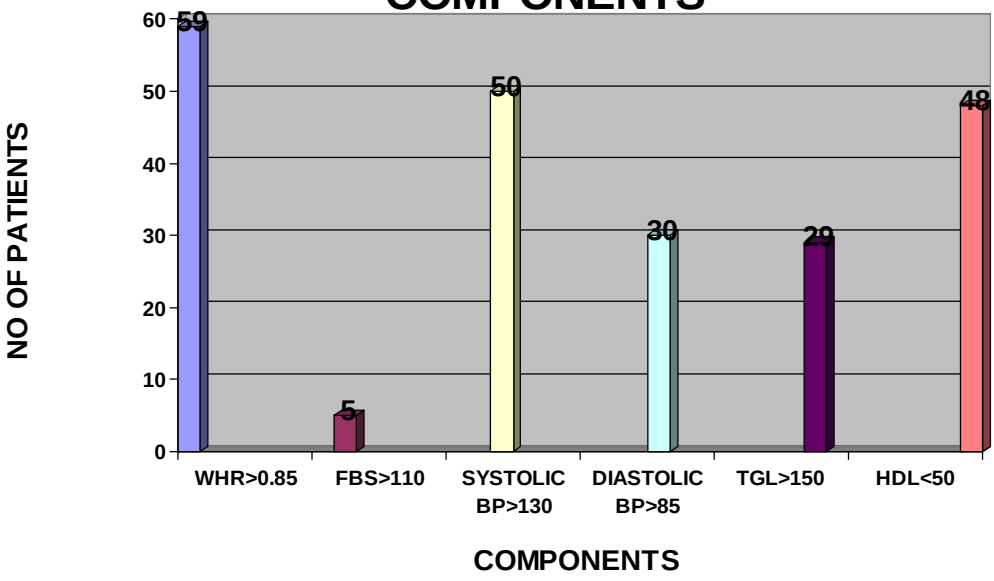

METABOLIC SYNDROME
66
70
30
34
70
60
50
40
30
20
10
0
PERCENTAGE OF PATIENTS
Present
Absent
METABOLIC SYNDROME
Case
Control


TRIGLYCERIDES / HIGH DENSITY LIPOPROTEIN RATIO
TGL/HDL RATIO
<3.2
>3.2
84
63
16
37
0
20
40
60
80
100
PERCENTAGE OF PATIENTS
Control
Case

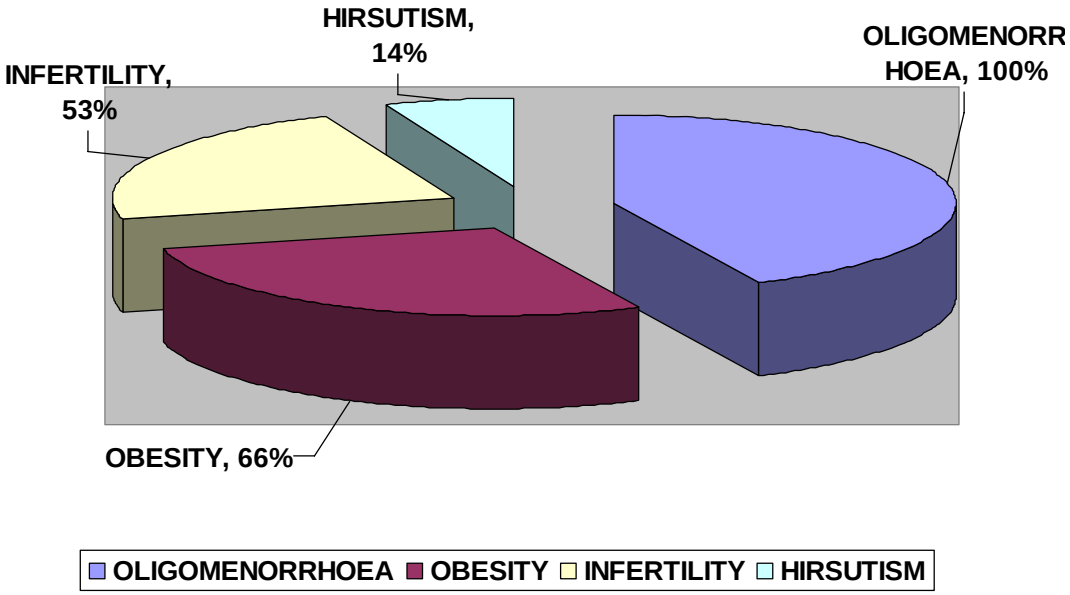
BMI/WAIST HIP RATIO



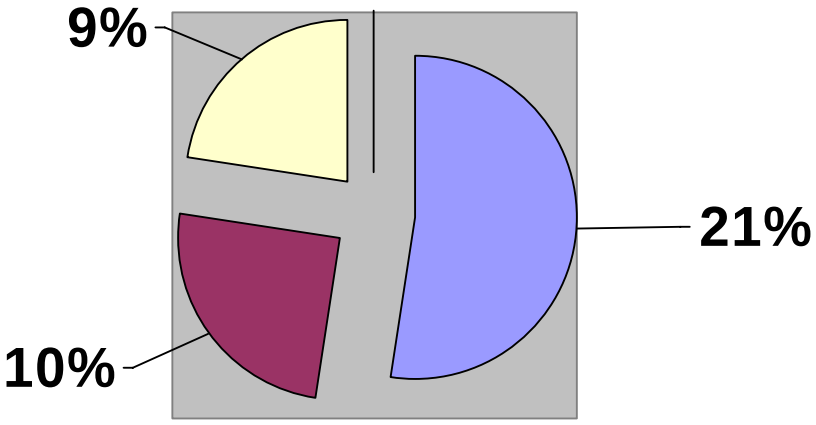
METABOLIC SYNDROME WITH COMPONENTS

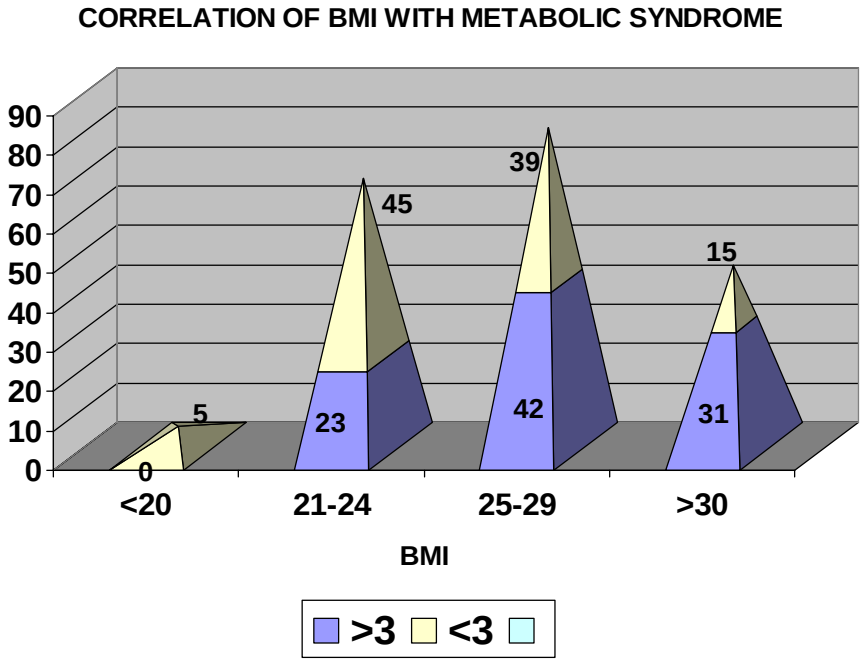


PRESENTING COMPLAINTS

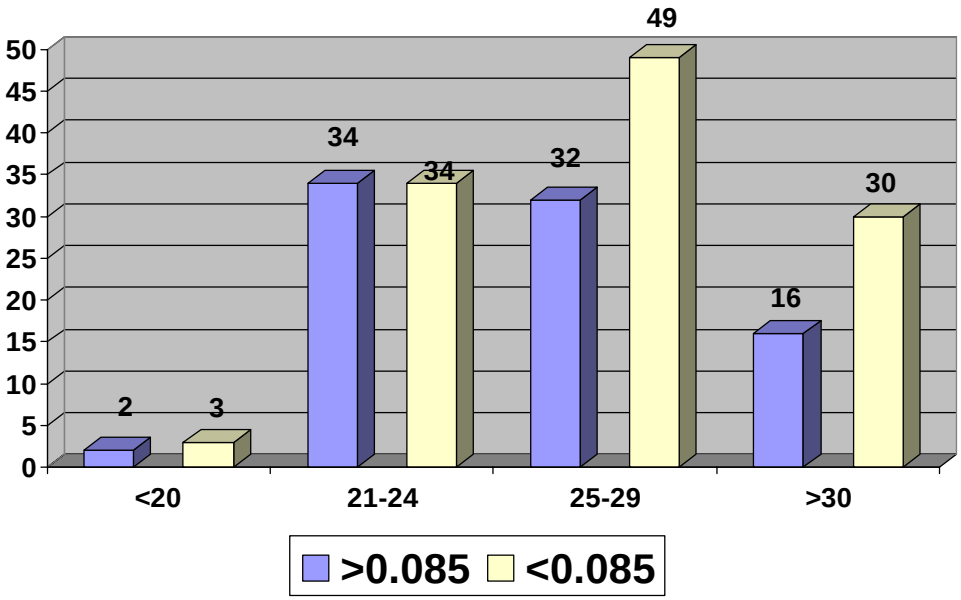


FAMILY HISTORY IN STUDY GROUP





CORRLEATION OF BMI WITH WAIST/ HIP RATIO



BIBLIOGRAPHY

1. **Greer. I.A. Zoe EC Hopkinson, Naveed Saltar,** Polycystic Orarian Syndrome; the metabolic syndrome comes to gynaecology BMJ 317 : 329-332, 1998

2. **Barbieri RC, smith S. Ryan KJ,** The role of hyperinsulinemia in the pathogenesis of orarian hyperandrogenism Fertil Steril, 50 : 197-212, 1998

3. **Orchard RE, Becker, DJ, Bates M, Kuller LH, Drash A,** Plasma Insulin and Lipoprotein concentration; an atherogenic association ? Am. J. epidemic 118 : 326 – 37, 1983.

4. **Conway GS, Agrawl R, Betteridge DJ. Jacobs HS,** Risk factor for coronary artery diesease in lean and obese women with the polycystic orary syndrome 1992.

5. **Reaven GM** 1988 banting lecture Role of insulin resistane in human diseas. Diabetics 37 (12), 1595 – 1607, 1988.

6. **Stein — IF.** The Stein-Leventhal syndrome, New England Jmed., 259.420, 1958

7. **Leon Speroff, Robert H.Glass, Nathan G. Kase** clinical gynecologic endocrinology and infertility, sixth edition, 493, 494, 497, 499; 1999.

8. **Kirschner M.A.,Samojlik E,Drejda M.Szmal E,Schneider G,Ertel N,** Androgen-estrogen metabolism in women with upper body versus lower body obesity,J. clin. Endocrino.met 70:473, 1990.

9. **D. Keith Edmonds,** Dewhurst Text book of obstetrics and Gynaecology for post graduate sixth edition, 46, 1999.

10. **Reaven GM** 1988 banting lecture Role of insulin resistane in human diseas. Diabetics 37 (12), 1595 – 1607, 1988.

11. **NCEP National Institute of Health.** Third Report of the NCEP expert panel on Detection, Evaluation, Treatment of high blood cholesterol in adults (Adult treatment panel III NIH publication 91-3670;2001)

12. **The Rotter-dam ESHRE / ASRM,** Sponsored PCOS consensus Workshop group Revised 2003 consensus on diagnotic criteria. Fertil Steril 81:19.25, 2004.

13. **Hales C.N. Barker D.J.P. Clark P.M.S. Cox, L.J. Full, C. Osmond C,** Winter P.D. 1991, Fetal and infant growth and impaired glucose tolerance at age 64 years BMJ. 303, 1019-1022.

14. **Philips D.I., Barker, D.J. Hales, C.N. Hirst, S, Osmond C,** Thinness at birth and insulin resistance in adult life. Diabetolgia, 37, 150-154. 1994

15. **Barker D.J, Osmond C,** low birth weight and hyertension Br. Med. J. 297, 134-135,1988

16. **Barker D.J.P, Hales, C.N. Fall, C.H.D., Osmond .C., Philipe. K; Clark P.M.S** Type 2 (NIDM) HT, hyperhpidemia syndrome X relation to reduced fetal growth, Diabetoliga 36, 62-67, 1993.

17. **Hatterseley A.T, Tooke J.E.** Fetal Insulin hypothesis alternative explanation for Low birth weight with DM & HT LANCET 353, 1789-1792, 1999.

18. **Hatterseley A.T., Beards, F, Ballantyne**, Mutatations in glucokinase gene of the fetus results in decrease birth weight. Nat.genet 19; 268-270, 1998.

19. **Eriksson J.G, Lindi V forsen T.J.M. Osmond Barker**. The effects of the pro 12 Ala polymorphism on insulin sensitivity and insulin metabolism 51; 2321-2324, 2002.

20. **Bairer, L.J. Permana, P.A. Yang. Pratley,**A calpain — 10 gene Polymorphrim is associated with decrease muscle MRNA and insulin resistance; J. Clin. Invest.106 R69-73, 2000.

21. **Lynn.S.Evans.JC, White, Bell, Walker** Variation in calpain-lO gene affects blood glucose level diabetes 51 ;247-250, 2002.

22. **Barroso, I Gurnell, M.Crowky,** Dominant negative Mutation in human PPAR gamma associated with IR, DM, Hypertension nature 402, 880-883, 1999.

23. **Hegele, R.A.Cao H.Harris, Hanley**. The hepatic nuclear factor – 1 alpha G3 193 S variant is associated with early onset type2 DM J. Clin. Endocrino met 84, 1077-1082, 1999.

24. **Boj SF. Parrizas Maestro, Ferrer J.** A transcription factor regulatory circuit in differenciated pancreatic cells Proc nat Acad. Sci USA 98; 14481-86, 2001.

25. **Stride, A Hattersley.A T.** Different genes, different diabetes; Lesson from maturity onset diabetes of the young. Ann Med. 34; 207-216, 2002.

26. **Lapidus L.Bengtsson, Larsson B, Pennert K.** Distribution of adipose tissue and risk of CVD death Br. Med. J 289; 1257, 1984.

27. **Ashwell M. Chin S, Stalley, S, Garron JS,** female fat distribution a simple classification based on two circumference measurement Int. J.Obesity 6; 143, 1982.

28. **Lean MEJ Han TS, Duren berg P,** Predicting body composition by densitometry from simple anthropometric measurement AM. J. Clin nut. 63; 4, 1996.

29. **Pouliot MC, Despress JP, Lemineox, Mooranji S,** Waist circumference and abdominal saggital diameter visceral adipose tissue accumulation and related CV risk in men & women. AMJ Cardin 73; 960, 1994.

30. **Van Itallic TB,** Health implication of overweight and obesity in U.S. Ann. Int. med. 103; 983, 1985.

31. **Ferriman-D, Gallway JD.** Clinical assessment of body hair in women J Clinical Endo. Met 21; 1440-1447, 1961

32. **Dunaif A, Segal KR, Eutterweit W et al.** Profound peripheral insulin resistance, independent of obesity in polycystic ovary syndrom. Diabetes 38: 1165, 1989.

33. **Morales AJ, Laughlin GA, Butzow T, Maheswari H, Banumann G, Yen SSC,** Insulin, Somatotrophic and Luternising hormone axis in lean and obese women with polycystic ovary syndrom : common and distinct features, J clin Endocrinl Metab 81 : 2854. 1996.

34. **Zhang L, Rodriguez H, Ohno S, Miller WL,** Serine phosphorylation of human p.450 C.17 increases 17, 20 lyase activity; implications for adrenarche and the polycystic ovary syndrome, Proc Nat Acad Sci USA, 92 : 10619, 1995.

35. **Peiris AN Sothmann MS, Aiman EJ, Kissebah AH**, relationship of Insulin to SHBG role of adiposity fertility sterility 52; 69, 1989.

36. **Campbell PJ, Gerich JE,** Impact of obesity an insulin action in volunteers with normal glucose tolerance; demonstration of a threshold for the adverse effect of obesity, J Clin Endocrinol Metab 70 : 1114, 1990.

37. **Legro RS, Kunselman AR, Dunaif A.** Prevalence Predictors of dyslipidemia in women with polycystic ovary syndrome. AM J:med 607-13, 2001

38. **Reaven GM, Lithell, H, landsberg L,** Hypertension and associated metabolic abnormalities – the role of insulin resistance and the sympathoadrenal system, New Engl J. Med 334 : 374, 1996

39. **Legro RS, Dunaif A,** Prevalence and predictors of risk for type 2 diabetes mellitus and impaired glucose tolerance in polycystic ovary syndrome J clin Endocrinology meta 84:165-9, 1999

40. **Dahlgren E Janson Po, Johansson S,** Polycystic ovarian syndrome and risk for Myocardial infarction— Act.aObstet Gynecol Scand 71, 599.604, 1992

41. **Wild R.A. Grubb B.Hartz** Clinical signs of androgen excess as risk factors for coronary artery disease fertility sterility 54, 255-259, 1990. **Wild RA, Painter PC, Coulson, PB, Carruth K,** Lipoprotein concentrations and cardiovascular risk in women with PCOS J. Clin. Endo. Met. 61; 947, 1995.

42. **Talbott, E.O. Zborowski, J.V. Rager, J.R. Boudreaux, M.Y. Edmundowicz D.A.** a. evidence for an association between metabolic cardiovascular syndrome and coronary and aortic calcification among women with polycystic ovary syndrome J. Clin. End. Met. 89. 5454-5461, 2004.

43. **Wi1d R.A.** Polycystic ovary syndrome a risk for coronary artery disease AMJ Obs.-Gyn. 186; 35-43, 2002.

44. **Grundy SM.AM.J Clin.** Nutrition June 83 (6) 1248.51.2006

45. **Dunaif A.Thomas A. Thomas A.** Current concepts in polycystic ovary syndrome. Annual. Rev. Med 52; 401 -409, 2001.**EK, IARNER, P. Ryden, M.Holm Wabrenberg H.** A unique defect in the regulation of visceral fat cell hpolysis in PCOS as an early link to insulin resistance. diabetes, 51; 489-492, 2002.

46. **Kelly C.C. Lyall, H.Petrie, Satlar N.** Low grade chronic inflammation in women with PCOS J. Clin endocrine met 86; 2453-2455, 2001.

47. **Cresswal, J.L, Barker, D.J.Osmond, Fraser. R.B.** fetal growth, length of gestation and polycystic ovaries in adult life Lancet 350, 1131-1135, 1997.

48. **Holte, J. Bergh, T. Berne, C, Berglund, L. Lithell, H,** enhanced early insulin response to glucose in relation to insulin resistance in women with Polycystic ovary syndrome and normal glucose tolerance J. Clin endo met 78. 1052 – 1058, 1994.

49. **Escobar-Morreak H.F,** Serum interleukin - 18 relationship to insulin resistane and to obesity J.Clin Endromology med 89, 806-811, 2004.

50. **Yildrim B. Sabir N. Kaleli B,** Relation of intra abdominal fat distribution to metabolic disorders in nonobese patinets with polycystic ovary syndrome fertility Sterility, 79 1358 – 1364, 2003.

51. **Talbott E, Guzick DS, Clerici A, Berga S, Detre K, Weimer K, Kuller L,** Coronary artery disease risk factors in women with polycystic ovary syndrome. Arterioscler Thromb Vas Biol 57 : 314, 1992.

52. **Aprindonidze T., Essah, P.A., Luorno. M.J. Nestler, J.E.,** Prevalence and characteristics of the metabolic syndrome in women with polycystic orary syndrome J. Clin. End. Met., 90, 1929 – 1935, 2005.

53. **Anuja Dokras, Melinda, Elizabeth,** Screening women with polycystic ovary syndrome for metabolic syndrome vol. 106, 1 July 2005.

54.

55.

56.

57. **Hales C.N. Barker D.J.P.** Type 2 (NIDM) Thirfty phenotype hypothesis. Diabetolgia 35; 596-661, 1992.

58.

59. **ESHRE (Rotterdam ESHRE / ASRM)** Sponsored PCO conscensus workshop longterm health risk related to PCOS 19; 41-47, 2004.

60. **Franks.S**, Polycystic syndrome N.Eng J Med 333; 853-86, 1995.

61. **Hopkinson, Z.E, Sattar, N.Fleming.Greer I.A.** Polycystic ovarian syndrome comes to gynecology BMJ, 317; 329-332, 1998.

62.

63. Despress JP, lemineros, Moorjani S, waist circumference and abdominal saggital diameter visceral adpose tissue accumulation and related CV risk in men & women. AMJ cardio 73; 460,1994.

CONTROL STUDY

LIPID PROFILE

SI.No	Name	IP. No	AGE	BMI	WC	WH	SBP	DBP	HIRSUTISM	TSH	PROLACTIM	FASTBSUGAR	TCHO	TGL	HDL	RATTIO	OLIGO MENOR	OBESITY	INFERTILITY	HIRSUTISM	PCO	DM	HYP	GROUP	WCCODE	AGECODE	BMICODE	WHRATCOD	BP	BP1	HDLCODE	TGLCODE	RATIOCOD	FASTCODE	METSYN	METSYNCO
1	Santha		26	23.5	84	0.82	120	80	0	1.67	4.07	75	104	119	39.6	2.9							104	2	0	3	2	0	2	0	1	0	2	0	1	2
2	Revathy		26	24	73	0.79	110	70	0	2.32	7.5	82	161	169	32.5	3.3							161	2	0	3	2	0	2	0	1	1	1	0	2	2
3	Asha		25	23	94	0.89	130	86	0	1.002	13.4	83	162	138	31.7	4.2							162	2	1	2	2	1	1	1	1	0	1	0	4	1
4	Devaki		25	24	77	0.84	110	70	0	2.119	19.26	102	117	158	52.8	3.03							117	2	0	2	2	0	2	0	0	1	2	0	1	2
5	Ambika		25	31	90	0.9	116	70	0	1.06	7.8	70	191	147	48.2	3.06							191	2	1	2	4	1	2	0	1	0	2	0	3	1
6	Indu		32	29	87	0.85	100	60	0	3.21	12.2	74	172	91	43	2.11							173	2	0	4	3	0	2	0	1	0	2	0	1	2
7	Radha		26	35	101	1.2	130	90	0	2.78	14.9	93	194	70	58.6	1.2							194	2	1	3	4	1	1	1	0	0	2	0	3	1
8	Vijayalakshmi		27	29	74	0.81	140	80	0	2.41	8.74	81	200	76	45.2	1.68							200	2	0	3	3	0	2	1	1	0	2	0	2	2
9	Aarthy		23	24	70	0.76	130	70	0	4.01	1.62	75	140	78	43	1.81							140	2	0	2	2	0	2	1	1	0	2	0	2	2
10	Shoba		21	31	89	0.82	120	80	0	3.167	10.4	92	159	138	40.7	3.45							159	2	1	2	4	0	2	0	1	0	1	0	2	2
11	Radhika		22	34	74	0.76	130	80	0	2.21	8.72	80	180	132	65	2.03							180	2	0	2	4	0	2	1	0	0	2	0	1	2
12	Shenbagum		23	24.2	89	0.9	110	80	0	3.34	12.6	82	140	72	43	1.67							140	2	1	2	3	1	2	0	1	0	2	0	3	1
13	Roshani		20	23.9	87	0.88	110	70	0	2.101	7	75	157	118	57.6	2.07							157	2	0	1	2	1	2	0	0	0	2	0	1	2
14	Devi		23	29	84	0.8	130	80	0	2.041	9.4	92	160	109	32.5	3.3							160	2	0	2	3	0	2	1	1	0	1	0	2	2
15	Vasundara		22	22.6	73	0.85	110	80	0	1.76	2.36	80	190	135	54.3	2.5							190	2	0	2	2	0	2	0	0	0	2	0	0	2
16	Muniammal		24	30	85	0.9	110	60	0	2.34	5.71	75	149	167	43.7	2.5							149	2	0	2	3	1	2	0	1	1	2	0	3	1
17	Sudha		26	34	84	0.92	100	70	0	1.07	4.02	76	159	90	36.4	2.6							159	2	0	3	4	1	2	0	1	0	2	0	2	2
18	Seetha		28	24.7	80	0.81	120	80	0	3.01	1.01	80	223	70	58.1	1.2							223	2	0	3	3	0	2	0	0	0	2	0	0	2
19	Manju		25	23	73	0.8	130	70	0	1.12	6.2	91	194	61	38.6	1.605							194	2	0	2	2	0	2	1	1	0	2	0	2	2
20	alamelu		27	23	76	0.9	110	74	0	1.002	4.07	80	156	92	62.6	1.48							156	2	0	3	2	1	2	0	0	0	2	0	1	2
21	Hema		28	22	90	0.91	130	86	0	5.11	7.826	81	162	66	43.3	1.53							162	2	1	3	2	1	1	1	1	0	2	0	4	1
22	Sunitha		24	24	94	0.9	130	70	0	1.69	5.314	85	168	135	51.5	2.64							168	2	1	2	2	1	2	1	0	0	2	0	3	1
23	Sumathi		26	30	81	0.8	110	60	0	2.804	8.72	80	121	70	54.3	1.29							121	2	0	3	3	0	2	0	0	0	2	0	0	2
24	Aruna		23	30.6	75	0.75	130	80	0	4.11	6.101	79	190	90	43.7	2.09							190	2	0	2	4	0	2	1	1	0	2	0	2	2
25	Rekha		22	23.2	86	0.84	140	90	0	2.04	8.443	75	159	132	38.1	3.42							159	2	0	2	2	0	1	1	1	0	1	0	2	2
26	Selvi		21	24	75	0.88	116	80	0	4.169	7	83	180	72	65	1.107							180	2	0	2	2	1	2	0	0	0	2	0	1	2
27	Saradha		25	24	74	0.81	116	76	0	2.54	5.601	85	168	107	43	7							168	2	0	2	2	0	2	0	1	0	1	0	1	2
28	Sublakshmi		27	30	88	0.86	130	70	0	1.76	16.26	90	154	86	46.4	1.91							184	2	1	3	3	1	2	1	1	0	2	0	4	1
29	Anajli		25	30.6	90	0.91	120	80	0	3.32	4.8	89	161	135	47	2.87							161	2	1	2	4	1	2	0	1	0	2	0	3	1
30	Swarana		24	30.3	81	0.83	140	80	0	2.41	7.5	87	143	115	35.2	3.28							143	2	0	2	4	0	2	1	1	0	1	0	2	2
31	Janaki		26	33	112	1.2	140	90	0	1.12	6.29	87	143	115	35.2	3.28							143	2	1	3	4	1	1	1	1	0	1	0	4	1
32	Jothi		31	30	88	0.8	110	70	0	1	6.79	70	194	61	38	1.6							194	2	1	4	3	0	2	0	1	0	2	0	2	2
33	Yeshoda		21	22	96	0.9	100	70	0	2.1	7	75	168	66	48.3	1.53							168	2	1	2	2	1	2	0	1	0	2	0	3	1
34	Radha		22	29	73	0.74	130	80	0	2.78	11.2	85	121	70	50	1.2							121	2	0	2	3	0	2	1	1	0	2	0	2	2
35	Vasanthi		26	23	81	0.8	110	70	0	3.2	11.4	92	110	72	65	1.107							110	2	0	3	2	0	2	0	0	0	2	0	0	2
36	Sujatha		30	22	85	0.86	120	80	0	3.17	11.6	80	180	135	51.6	2.64							180	2	0	3	2	1	2	0	0	0	2	0	1	2
37	Rajeswari		26	33.1	64	0.72	130	80	0	2.117	8.06	73	223	107	44	2.4							223	2	0	3	4	0	2	1	1	0	2	0	2	2
38	Sumathi		20	31	90	0.91	130	70	0	3.21	7.03	74	170	66	43.3	1.5							170	2	1	1	4	1	2	1	1	0	2	0	4	1
39	Surya		28	29	94	0.9	150	90	0	4.07	5.06	86	159	90	36.4	2.6							159	2	1	3	3	1	1	1	1	0	2	0	4	1
40	Reshma		20	24.2	80	0.8	140	80	0	0.06	6.71	88	194	61	38	1.605							194	2	0	1	3	0	2	1	1	0	2	0	2	2
41	Kamali		21	26	82	0.82	110	70	0	1.002	4.02	78	103	90	36.4	2.6							103	2	0	2	3	0	2	0	1	0	2	0	1	2
42	Karthika		25	30	79	0.75	120	80	0	2.016	4.06	74	172	61	38	1.6							172	2	0	2	3	0	2	0	1	0	2	0	1	2
43	Renuka		28	22	79	0.78	110	70	0	2.74	7.73	92	186	136	51	1.31							186	2	0	3	2	0	2	0	0	0	2	0	0	2
44	Chitra		20	23	79	0.77	120	70	0	2.61	9.06	89	164	107	43	2.42							164	2	0	1	2	0	2	0	1	0	2	0	1	2
45	Kalpana		19	26	92	0.9	118	74	0	1.84	5.74	90	144	61	38	1.607							144	2	1	1	3	1	2	0	1	0	2	0	3	1
46	Vasundara		22	22.6	73	0.85	110	80	0	1.76	2.36	80	190	135	54.3	2.5							190	2	0	2	2	0	2	0	0	0	2	0	0	2

47	Muniammal		24	30	85	0.9	110	60	0	2.34	5.71	75	149	167	43.7	2.5							149	2	0	2	3	1	2	0	1	1	2	0	3	1
48	Sudha		26	34	84	0.92	100	70	0	1.07	4.02	76	159	90	36.4	2.6							159	2	0	3	4	1	2	0	1	0	2	0	2	2
49	Seetha		28	24.7	80	0.81	120	80	0	3.01	1.01	80	223	70	58.1	1.2							223	2	0	3	3	0	2	0	0	0	2	0	0	2
50	Latha		25	23	73	0.8	130	70	0	1.12	6.2	91	194	61	38.6	1.605							194	2	0	2	2	0	2	1	1	0	2	0	2	2
51	Janaki		27	23	76	0.9	110	74	0	1.002	4.07	80	156	92	62.6	1.48							156	2	0	3	2	1	2	0	0	0	2	0	1	2
52	Hemalatha		28	22	90	0.91	130	86	0	5.11	7.826	81	162	66	43.3	1.53							162	2	1	3	2	1	1	1	1	0	2	0	4	1
53	Kumari		24	24	94	0.9	130	70	0	1.69	5.314	85	168	135	51.5	2.64							168	2	1	2	2	1	2	1	0	0	2	0	3	1
54	Prema		26	33	112	1.2	140	90	0	1.12	6.29	87	143	115	35.2	3.28							143	2	1	3	4	1	1	1	1	0	1	0	4	1
55	Meena		31	30	88	0.8	110	70	0	1	6.79	70	194	61	38	1.6							194	2	1	4	3	0	2	0	1	0	2	0	2	2
56	Jayalakshmi		21	22	96	0.9	100	70	0	2.1	7	75	168	66	48.3	1.53							168	2	1	2	2	1	2	0	1	0	2	0	3	1
57	Jeeva		22	29	73	0.74	130	80	0	2.78	11.2	85	121	70	50	1.2							121	2	0	2	3	0	2	1	1	0	2	0	2	2
58	Rekha		26	23	81	0.8	110	70	0	3.2	11.4	92	110	72	65	1.107							110	2	0	3	2	0	2	0	0	0	2	0	0	2
59	Indra		30	22	85	0.86	120	80	0	3.17	11.6	80	180	135	51.6	2.64							180	2	0	3	2	1	2	0	0	0	2	0	1	2
60	Rajathi		26	33.1	64	0.72	130	80	0	2.117	8.06	73	223	107	44	2.4							223	2	0	3	4	0	2	1	1	0	2	0	2	2
61	Komala		20	31	90	0.91	130	70	0	3.21	7.03	74	170	66	43.3	1.5							170	2	1	1	4	1	2	1	1	0	2	0	4	1
62	Kavitha		28	29	94	0.9	150	90	0	4.07	5.06	86	159	90	36.4	2.6							159	2	1	3	3	1	1	1	1	0	2	0	4	1
63	Jenny		20	24.2	80	0.8	140	80	0	0.06	6.71	88	194	61	38	1.605							194	2	0	1	3	0	2	1	1	0	2	0	2	2
64	Ushanandhini		21	26	82	0.82	110	70	0	1.002	4.02	78	103	90	36.4	2.6							103	2	0	2	3	0	2	0	1	0	2	0	1	2
65	leela		26	23.5	84	0.82	120	80	0	1.67	4.07	75	104	119	39.6	2.9							104	2	0	3	2	0	2	0	1	0	2	0	1	2
66	sumathi		26	24	73	0.79	110	70	0	2.32	7.5	82	161	169	32.5	3.3							161	2	0	3	2	0	2	0	1	1	1	0	2	2
67	Sujatha		25	23	94	0.89	130	86	0	1.002	13.4	83	162	138	31.7	4.2							162	2	1	2	2	1	1	1	1	0	1	0	4	1
68	Sreedevi		25	24	77	0.84	110	70	0	2.119	19.26	102	117	158	52.8	3.03							117	2	0	2	2	0	2	0	0	1	2	0	1	2
69	yamuna		25	31	90	0.9	116	70	0	1.06	7.8	70	191	147	48.2	3.06							191	2	1	2	4	1	2	0	1	0	2	0	3	1
70	Reena		32	29	87	0.85	100	60	0	3.21	12.2	74	172	91	43	2.11							173	2	0	4	3	0	2	0	1	0	2	0	1	2
71	Fathima		26	35	101	1.2	130	90	0	2.78	14.9	93	194	70	58.6	1.2							194	2	1	3	4	1	1	1	0	0	2	0	3	1
72	Logeshwari		27	29	74	0.81	140	80	0	2.41	8.74	81	200	76	45.2	1.68							200	2	0	3	3	0	2	1	1	0	2	0	2	2
73	Bindu		23	24	70	0.76	130	70	0	4.01	1.62	75	140	78	43	1.81							140	2	0	2	2	0	2	1	1	0	2	0	2	2
74	Umamaheswari		21	31	89	0.82	120	80	0	3.167	10.4	92	159	138	40.7	3.45							159	2	1	2	4	0	2	0	1	0	1	0	2	2
75	Aruna		22	34	74	0.76	130	80	0	2.21	8.72	80	180	132	65	2.03							180	2	0	2	4	0	2	1	0	0	2	0	1	2
76	Prema		23	24.2	89	0.9	110	80	0	3.34	12.6	82	140	72	43	1.67							140	2	1	2	3	1	2	0	1	0	2	0	3	1
77	Thangam		20	23.9	87	0.88	110	70	0	2.101	7	75	157	118	57.6	2.07							157	2	0	1	2	1	2	0	0	0	2	0	1	2
78	Diya		23	29	84	0.8	130	80	0	2.041	9.4	92	160	109	32.5	3.3							160	2	0	2	3	0	2	1	1	0	1	0	2	2
79	Jenitha		22	22.6	73	0.85	110	80	0	1.76	2.36	80	190	135	54.3	2.5							190	2	0	2	2	0	2	0	0	0	2	0	0	2
80	Leelavathy		26	30	81	0.8	110	60	0	2.804	8.72	80	121	70	54.3	1.29							121	2	0	3	3	0	2	0	0	0	2	0	0	2
81	Indra		23	30.6	75	0.75	130	80	0	4.11	6.101	79	190	90	43.7	2.09							190	2	0	2	4	0	2	1	1	0	2	0	2	2
82	Geetha		22	23.2	86	0.84	140	90	0	2.04	8.443	75	159	132	38.1	3.42							159	2	0	2	2	0	1	1	1	0	1	0	2	2
83	Komala		21	24	75	0.88	116	80	0	4.169	7	83	180	72	65	1.107							180	2	0	2	2	1	2	0	0	0	2	0	1	2
84	Vidya		25	24	74	0.81	116	76	0	2.54	5.601	85	168	107	43	7							168	2	0	2	2	0	2	0	1	0	1	0	1	2
85	Lalithapriya		28	29	94	0.9	150	90	0	4.07	5.06	86	159	90	36.4	2.6							159	2	1	3	3	1	1	1	1	0	2	0	4	1
86	Sandhiya		20	24.2	80	0.8	140	80	0	0.06	6.71	88	194	61	38	1.605							194	2	0	1	3	0	2	1	1	0	2	0	2	2
87	Thamilselvi		21	26	82	0.82	110	70	0	1.002	4.02	78	103	90	36.4	2.6							103	2	0	2	3	0	2	0	1	0	2	0	1	2
88	Vinatha		25	30	79	0.75	120	80	0	2.016	4.06	74	172	61	38	1.6							172	2	0	2	3	0	2	0	1	0	2	0	1	2
89	Vishalakshi		28	22	79	0.78	110	70	0	2.74	7.73	92	186	136	51	1.31							186	2	0	3	2	0	2	0	0	0	2	0	0	2
90	Banumathy		20	23	79	0.77	120	70	0	2.61	9.06	89	164	107	43	2.42							164	2	0	1	2	0	2	0	1	0	2	0	1	2
91	vijaya		21	24	84	0.82	110	70	0	1.67	4.07	75	104	119	39.6	2.9																				
92	Edema		28	21	73	0.79	130	80	0	2.78	8.72	88	161	76	45.2	1.68																				
93	Latha		31	23.5	74	0.81	136	86	0	4.01	7.08	92	191	132	64	2.06																				
94	Yuvarani		20	22	80	0.88	140	84	0	1.021	8.7	64	172	66	48.3	1.53																				
95	Kamachi		32	23	73	0.74	110	76	0	3.016	1.212	80	167	61	38	1.6																				
96	Kanchana		27	22	88	0.91	120	70	0	3.6	0.24	69	191	169	32.5	3.3																				
97	Janakilakshmi		29	30	82	0.82	124	82	0	1.87	6.87	78	143	115	35.2	3.2																				
98	haritha		22	24	77	0.85	116	80	0	2.16	9.2	84	160	109	32	3.3																				
99	jeevatha		26	20	80	0.8	128	74	0	1.213	7.36	96	166	132	65	2.03																				
100	Deepa		25	19	84	0.9	100	84	0	4.1	8.17	88	206	72	43	1.67																				