

Original Research Article

Effect of Age and Body Mass Index on Various Clinical and Anthropometric Parameters of Type 2 Diabetic Patients: A Case-Control Study

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ABSTRACT

Background: Though much research has been carried out, still the pathological pathway of Type 2 Diabetes Mellitus (T2DM) is unclear. Prevalence of the disease is increasing with increment in comorbidities in younger to older Indian population.

Aim: The aim of this case-control study was to investigate the association of age intervals and Body Mass Index (BMI) with various clinical and anthropometric parameters in T2DM and age-matched healthy controls (AMHCs).

Methods: Total 300 subjects (150 T2DM and 150 AMHCs) were enrolled from urban and rural Northern India. Clinical and anthropometric parameters [BMI, Waist Circumference (WC) and Waist-Hip Ratio (WHR)] were analyzed in all subjects and associated with age groups (25-35 years, 36-45 years, 46-55 years, 56-65 years and 66-75 years) and BMI groups (underweight, normal weight, overweight and obese).

Statistical analysis: P values were calculated by ANOVA unpaired t-test. $p < 0.05$ was considered statistically significant.

Results: In our study the maximum load of T2DM was found in age groups of 36-45 years and 46-55 years (30% and 26.7%, respectively). About 49.3% of T2DM and 48% of AMHCs were found obese. FBS, PPBS, HbA1c and SCr, SBP and DBP were observed significantly higher in T2DM as compared to AMHCs in all age groups ($p < 0.001$) as well as in all BMI groups ($p < 0.001$). Lipid profile abnormalities and poor glycemic controls were significantly observed mainly in the age group of 25-35 years and 36-45 years. In T2DM cases, BMI had significantly positive correlation with WC & WHR ($r = +0.65$, $r = +0.65$, respectively, $p < 0.001$).

Conclusion: WC and WHR is an important marker for obesity than BMI in our study. WHR was the only significant predictor of BMI. Mainly, younger (25-35 years) and middle (36-45 years) age group have lipid profile abnormalities and poor glycemic controls in this study.

Keywords: Obesity, Type 2 Diabetes Mellitus, Body Mass Index, Glycated Haemoglobin, Waist Circumference, Waist-Hip Ratio.

INTRODUCTION

Diabetes mellitus (DM) is a growing public health problem worldwide with high

levels of morbidity and mortality. [1]

Although the exact pathogenesis of T2DM is unclear, it is generally accepted that

T2DM is a multifactorial disorder resulting from genetic polymorphisms and several environmental factors. [2] India, after China is the largest contributor to regional mortality, with one million deaths attributable to diabetes. India is one of the seven countries of the International Diabetes Federation, South East Asia (IDF, SEA) region. Worldwide 415 million people have diabetes of which 78.3 million people are in the SEA region and by 2040 this will rise to 140.2 million. In 2015, India had 69.2 million people with diabetes (20-79 years) which is expected to rise to 123.5 million by 2040. India is second only to China which is home to 109.6 million diabetics, but topped by having 36.5 million impaired glucose tolerance people in 2015 and by 2040 this will rise to 63.6 million. [3] The prevalence of diabetes was high in urban India than in rural (14.2 versus 8.3%) whereas prediabetes prevalence was found to be equal (urban 14.5%; rural 14.7%). [4]

“Asian Indian Phenotype” refers to certain unique clinical and biochemical abnormalities in Indians which include increased insulin resistance, greater abdominal adiposity i.e., higher waist circumference despite lower body mass index. This phenotype makes Asian Indians more prone to diabetes and premature coronary artery diseases (CAD). The prevalence of microvascular complications of diabetes like retinopathy and nephropathy are comparatively lower in Indians whereas the prevalence of premature CAD higher in Indians compared to other ethnic groups. [5]

A follow up study of Chennai Urban Population based Study (CUPS) cohort showed that the overall mortality rate was nearly three times higher in people with diabetes compared to non diabetic subjects (18.9 versus 5.3 per 1000 person-years, $p=0.004$). [6,7] The hazard ratio (HR) for all cause mortality for diabetes was found to be 3.6 compared to non diabetic subjects. The study also showed that mortality due to cardiovascular disease (52.9% vs. 24.2%, $p=0.042$) and renal disease (23.5% vs. 6.1%, $p=0.072$) causes was higher among diabetic

subjects than non-diabetic subjects. Indians are known to have a relatively unfavourable risk profiles for type 2 diabetes and cardiovascular disease. [8]

Obesity is a complex disorder associated with variety of diseases such as cardiovascular disease (CVD), stroke, cancer, hypertension, diabetes, osteoarthritis and early death. [9] According to the World Health Organization (WHO), obesity is one of the common and most neglected public health problems in both developed and developing countries. [10] Globally one in six adults is obese and nearly 2.8 million individuals die each year due to overweight or obesity. [11] Due to this, the risk of morbidity and mortality has significantly increased.

Epidemiological survey used body mass index (BMI) as a measure of general obesity, and waist circumference (WC) and waist hip ratio (WHR) as measures of central/abdominal obesity. These have been associated with a number of metabolic abnormalities including prediabetes, type 2 diabetes (T2DM), hypertension (HTN), and cardiovascular diseases (CVDs). [12,13] There is prevalence of obesity and hypertension with type 2 diabetes. [14] A study by Mandal (2014) states that T2DM and hypertension increases with increasing weight of individuals. [15]

The 2010 American Diabetes Association Standards of Medical Care in Diabetes added glycated haemoglobin (HbA1c) as important criterion for the diagnosis of diabetes ($\geq 6.5\%$). [16] In diabetes mellitus, higher amounts of HbA1c, indicating poorer control of blood glucose levels, have been associated with macrovascular and microvascular complications such as CAD, nephropathy, retinopathy and neuropathy. [17,18] The blood HbA1c reading provides a good picture of average plasma glucose for the previous two to three months. [19] Further, the studies demonstrated that improving HbA1c by 1% for people with type 1 diabetes or type 2 diabetes cuts the risk of microvascular complications by 25%. [20]

A recent finding by Iglay et al. (2016) shows HTN, overweight/obesity, hyperlipidemia, CKD, and CVD as the most common conditions associated with T2DM in this study group (82.1%; 78.2%; 77.2%; 24.1%; and 21.6%, respectively). Their result further demonstrated that the highest co-prevalence for the combination of HTN and hyperlipidemia (67.5%), followed by overweight/obesity and HTN (66.0%), overweight/obesity and hyperlipidemia (62.5%), HTN and CKD (22.4%), hyperlipidemia and CKD (21.1%), HTN and CVD (20.2%), hyperlipidemia and CVD (20.1%), overweight/obesity and CKD (19.1%) and overweight/obesity and CVD (17.0%).^[21]

Due to these multiple comorbidities and pathophysiology associating with T2DM, we measured various clinical parameters (Blood pressure, blood sugar fasting, 2-hour post prandial blood sugar, glycated haemoglobin, lipid profile and serum creatinine) and anthropometric parameters (BMI, WC and WHR) for better assessment, diagnosis, management and treatment of T2DM patients and also correlate these parameters with AMHCs to have closure view to address these comorbidities in T2DM patients and healthy controls with comparing age and BMI.

MATERIALS AND METHODS

Subject Selection: This case-control study was approved by the institutional ethical committee of the University. Subjects with T2DM and AMHCs were enrolled from the outpatients attending the Diabetes Clinic of University. Written informed consent was taken from each subject and all procedures performed in studies involving human participants were in accordance with the ethical standards of this university and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Screening and management of patients was done as per American Diabetes Association guidelines.^[22] This study was conducted on 300 subjects (150 T2DM and

150 AMHCs), the recruited subjects aged between (25-75 years). T2DM was defined according to the criteria provided by The **American Diabetes Association:**^[23] (1) glycated haemoglobin (HbA1c) $\geq 6.5\%$, or (2) fasting blood sugar $\geq 126\text{mg/dl}$, or 2 hour post-prandial blood sugar $\geq 200\text{mg/dl}$. For the assessment of blood sugar levels of known diabetic subjects on strict dietary control and AMHCs fasting (8-10 hour overnight fasting) and 2 hours post prandial blood sugar was recorded. Glycated haemoglobin (HbA1c) was also measured as one the diagnostic parameter for the confirmation of diabetes. Subjects with ischemic heart disease, angina, MI, electrocardiogram abnormalities, those with other concurrent sickness like chronic liver disease, hypothyroidism or those on drugs like diuretics, pregnant women and women using oral contraceptives were excluded from the study.

Study design: All subjects were grouped based on age wise distribution in five groups (25-35 years, 36-45 years, 46-55 years, 56-65 years and 66-75 years) for determining the impact of age on biochemical variables of T2DM and AMHCs. Secondly, all biochemical variables were grouped as per BMI in four groups i.e. underweight (BMI $< 18.5 \text{ Kg/m}^2$), normal weight (BMI $18.5-22.9 \text{ Kg/m}^2$), overweight (BMI: $23-24.9 \text{ kg/m}^2$) and obese (BMI $\geq 25 \text{ kg/m}^2$) for determining the impact of BMI and obesity on biochemical variables of T2DM and AMHCs.

Metabolic syndrome was diagnosed according to the modified International Diabetes Federation (IDF) definition i.e. (1) Abdominal obesity with waist circumference of more than 90cm, (2) Elevated serum triglycerides (TG) $\geq 150\text{mg/dl}$, (3) Low high density lipoprotein (HDL) cholesterol (Men $< 40\text{mg/dl}$, Women $< 50\text{mg/dl}$), (4) Elevated blood pressure $\geq 130\text{mmHg}/ \geq 85\text{mmHg}$, and (5) Elevated Fasting blood sugar $\geq 110\text{mg/dl}$.^[24]

Laboratory Investigations: 5ml blood sample was taken in EDTA and plain vials after overnight fasting for biochemical estimations. Investigations performed included blood sugar fasting (F) and 2-hours post-prandial (PP), lipid profile including total cholesterol (TC), low density lipoprotein (LDL)-cholesterol, high density lipoprotein (HDL)-cholesterol, very low density lipoprotein (VLDL)-cholesterol, triglyceride (TG) (mg/dl) and serum creatinine. The estimations were done with commercially available ortho-clinical diagnostics kits (Johnson & Johnson) using Vitros 250 system chemistry auto-analyzer. HbA1c was measured by high-performance liquid chromatography (HPLC) based method using the D10 HbA1c analyzer (Bio-Rad).

Anthropometric measurements: Weight, height, hip circumference (at the level of the greater trochanters in a standing position) and waist circumference (at the level of the umbilicus in a standing position) were measured. The measurements were repeated by two different trained staff using the same device and mean value was recorded.

After resting for 10 minutes in a sitting position, systolic and diastolic blood pressures were measured twice by the same two investigators using the same manual sphygmomanometer for all subjects. Mean blood pressures of two measurements were recorded. Hypertension was defined as use of antihypertensive medication or means systolic blood pressure above 130 mmHg and means diastolic blood pressure more than 85 mmHg without use of any anti-hypertensive medication.

BMI Estimation:-Weight was recorded to the nearest kilogram (kg) with the subject standing on the weighing machine without shoes and using minimum of clothing. The same weighing machine was used for all the study subjects and the machine was tested with a known set of weights for any error.^[25] Height was recorded with the subject erect; bare footed; feet together; back and heels against the upright bar of height scale; head upright in Frankfurt horizontal plane

‘look straight ahead’. The height measuring equipment consisted of a vertical bar with a steel tape attached. Attached perpendicularly to the vertical bar was a horizontal bar which was brought down snugly on the examinee’s head.^[26] Body Mass index was calculated from the formula; $[BMI = \text{Weight in Kilogram} / (\text{Height in meters})^2]$

Study subjects were grouped in four categories viz. underweight, normal weight, overweight and obese. Because Asian Indians tend to develop diabetes at a significantly lower BMI and WC than white Europeans, lower thresholds of BMI to define overweight (BMI: 23-24.9 kg/m²) and obesity (BMI \geq 25 kg/m²) were proposed by IDF and NICE.^[27,28] Similarly the upper limit for waist circumference of men and women was defined as 90 cm and 80 cm respectively. The average Waist-Hip ratio of men and women was defined as 0.90-0.95 and 0.80-0.86 respectively and at risk of men and women was defined as \geq 0.95 and \geq 0.86 respectively. A detailed clinical history was taken and physical examination performed.

Statistical analysis

Statistical analysis was applied to all data using SPSS software (v20.0). Mean \pm SD (Standard Deviation) of all clinical parameters were calculated in each age group as well as in BMI groups. *P values* were calculated by ANOVA unpaired t-test. All p values were two sided and differences were considered statistically significant for $p < 0.05$; all significant data suggest the strength of association with clinical parameters.

RESULTS

A total of 300 subjects were enrolled for this case control study (150 T2DM and 150 AMHCs). Of these, 61.3% were males and 38.7% were females. The male: female ratio in cases and controls were 1.83:1 and 1.38:1 respectively. The anthropometric and biochemical profile of both the groups are given in Table 1. Anthropometric parameters i.e. WC, WHR,

SBP and DBP were significantly higher in T2DM cases. Similarly, biochemical parameters such as blood sugar, HbA1c, SCr were also significantly raised in T2DM

cases as compared to AMHCs (p<0.001). Additionally, higher triglyceride and VLDL were also observed in T2DM cases than in AMHCs (p=0.002, p=0.003, respectively).

Table 1: Baseline characteristics of Case (T2DM) and Control Groups

Parameters	Case (n=150)	Control (n=150)	P-value
Age (years)	48.31±10.88	48.03±11.83	0.83
Gender (M/F)	97/53	87/63	0.29
BMI (kg/m ²)	24.96±4.68	24.73±4.74	0.67
Waist circumference (cm)	97.93±6.54	95.76±7.31	0.007*
Waist-Hip Ratio (WHR)	0.98±0.07	0.96±0.07	0.01*
SBP (mmHg)	140.75±26.65	114.45±7.52	<0.001*
DBP (mmHg)	82.39±15.67	70.21±8.69	<0.001*
FBS (mg/dl)	167.01±73.41	93.83±11.47	<0.001*
PPBS (mg/dl)	260.59±112.52	127.29±24.41	<0.001*
HbA1c (%)	8.01±2.09	5.30±0.72	<0.001*
Total Cholesterol (mg/dl)	167.81±53.94	157.73±46.27	0.08*
Triglyceride (mg/dl)	182.27±112.76	148.35±68.36	0.002*
HDL (mg/dl)	38.31±10.81	39.08±11.81	0.56
LDL (mg/dl)	92.59±47.59	91.15±47.11	0.79
VLDL (mg/dl)	36.14±22.57	29.67±13.72	0.003*
Serum Creatinine (mg/dl)	2.44±2.11	0.91±0.25	<0.001*

Values are expressed as Mean ± Standard Deviation, *Significant considered as P<0.05.

M: Male, F: Female, FBS: Fasting Blood Sugar, PPBS: Post-Prandial Blood Sugar, HbA1c: Glycated Haemoglobin, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Sugar, BMI: Body Mass Index, WC: Waist Circumference, TC: Total Cholesterol, TG: Triglyceride, HDL: High-Density Lipoprotein, LDL: Low-Density Lipoprotein, VLDL: Very Low-Density Lipoprotein

Table 2: Age-wise Distribution of Anthropometric and Clinical Profiling of Type 2 Diabetic Patients with Age Matched Healthy Controls

Age Group (years) → Variables ↓	25-35			36-45			46-55			56-65			66-75		
	Case (n=25)	Control (n=25)	P-value	Case (n=45)	Control (n=45)	P-value	Case (n=40)	Control (n=40)	P-value	Case (n=29)	Control (n=29)	P-value	Case (n=11)	Control (n=11)	P-value
Gender (M/F)	16/9	15/10	1.00	24/21	25/20	1.00	25/15	20/20	0.37	23/6	17/12	0.16	9/2	10/1	1.00
BMI (kg/m ²)	23.35 ±5.35	24.31 ±4.12	0.48	25.62 ±4.80	25.18 ±4.65	0.65	25.58 ±4.60	25.02 ±4.87	0.59	24.81 ±3.44	24.12 ±4.38	0.51	24.02 ±4.55	24.39 ±6.32	0.88
Waist circumference (cm)	96.94 ±7.68	96.72 ±5.65	0.91	97.16 ±6.88	96.56 ±7.45	0.69	98.63 ±5.94	95.22 ±8.35	0.04	99.10 ±5.47	94.28 ±7.33	0.006	97.73 ±6.15	96.18 ±4.59	0.51
Waist-Hip Ratio (WHR)	0.97 ±0.08	0.97 ±0.06	>0.99	0.97 ±0.07	0.97 ±0.07	>0.99	0.99 ±0.06	0.95 ±0.08	0.01	0.99 ±0.06	0.94 ±0.07	0.005	0.98 ±0.06	0.96 ±0.05	0.41
SBP (mmHg)	135.96 ±17.31	116.96 ±5.00	<0.001	137.04 ±25.17	113.33 ±7.32	<0.001	143.58 ±29.73	114.90 ±7.36	<0.001	146.93 ±30.60	114.21 ±7.51	<0.001	140.18 ±22.21	112.36 ±11.18	0.001*
DBP (mmHg)	82.42 ±14.47	73.64 ±6.67	0.008	79.78 ±15.44	69.89 ±8.86	0.0003	84.48 ±16.15	69.90 ±8.31	<0.001	84.31 ±16.15	68.86 ±6.99	<0.001	80.36 ±12.98	68.36 ±13.88	0.049*
FBS (mg/ml)	191.32 ±101.01	91.84 ±11.80	<0.001	169.91±82.52	92.64 ±12.16	<0.001	151.99±53.55	93.05 ±10.72	<0.001	173.63±49.18	95.66 ±10.38	<0.001	137.00±49.33	101.27±9.52	0.03*
PPBS (mg/ml)	292.46 ±164.49	119.40 ±22.43	<0.001	271.02 ±120.98	127.44 ±24.08	<0.001	243.46 ±91.47	127.55 ±26.71	<0.001	263.56±56.81	129.48±22.06	<0.001	199.91±69.39	137.91±21.58	0.01*
HbA1c (%)	8.74 ±2.84	5.35±0.64	<0.001	7.92 ±2.22	5.13 ±0.71	<0.001	7.90 ±1.71	5.26 ±0.79	<0.001	8.07 ±1.31	5.52 ±0.58	<0.001	6.96 ±1.85	5.52 ±0.73	0.03*
Total Cholesterol (mg/dl)	159.38 ±41.01	133.62 ±37.96	0.02	178.65 ±63.95	165.23 ±45.01	0.25	165.04 ±61.59	164.73 ±46.00	0.98	166.39 ±32.46	154.47 ±41.78	0.23	156.44 ±41.74	164.98 ±59.33	0.70
Triglyceride (mg/dl)	164.08 ±70.56	126.80 ±42.61	0.03	200.97 ±108.27	158.53 ±85.70	0.04	198.26 ±149.17	149.53 ±67.99	0.06	163.61 ±95.46	149.90 ±59.87	0.52	138.17 ±56.47	147.35 ±42.35	0.67
HDL (mg/dl)	34.07 ±9.68	38.23 ±11.95	0.18	37.85 ±11.32	38.71 ±11.80	0.73	39.69 ±9.27	40.63 ±11.17	0.68	37.86 ±11.93	40.40 ±11.02	0.40	35.80 ±10.51	43.63 ±13.52	0.15
LDL (mg/dl)	88.71 ±38.22	74.18 ±34.15	0.16	95.23 ±47.53	95.35 ±46.93	0.99	93.01 ±63.03	97.82 ±47.98	0.70	94.20 ±30.75	83.76 ±37.79	0.25	84.80 ±35.34	107.78 ±71.79	0.35
VLDL (mg/dl)	32.74 ±14.09	25.38 ±8.54	0.03	40.14 ±21.60	31.63 ±17.14	0.04	38.96 ±30.16	29.90 ±13.71	0.09	32.20 ±18.62	30.08 ±12.09	0.61	27.63 ±11.42	29.49 ±8.55	0.61
Serum Creatinine (mg/dl)	3.07 ±3.85	1.05 ±0.20	0.01	2.24 ±1.58	0.86 ±0.26	<0.001	2.41 ±1.56	0.86 ±0.18	<0.001	2.44 ±1.44	0.87 ±0.23	<0.001	1.95 ±1.05	1.01 ±0.37	0.01*

Values are expressed as Mean±SD (Standard Deviation), *P-value < 0.05 is significant.

FBS, Fasting Blood Sugar; PPBS, Post-Prandial Blood Sugar; HbA1c, glycated Haemoglobin; BMI, Body Mass Index; HDL, High-Density Lipoprotein; LDL, Low-Density Lipoprotein; VLDL, Very Low-Density Lipoprotein.

Subjects enrolled in study were aged between 25-75 years. All the 150 T2DM cases were divided in 5 groups with the interval of 10 years. For matching the age group, similar numbers of healthy controls

were enrolled under each age group. We found no significant association of BMI with T2DM cases and AMHCs as a whole as well as in each age group. Waist circumference was higher in T2DM cases

but only in the age group of 56-65 years. Waist-hip ratio was significantly higher in T2DM cases as compared to AMHCs in age group of 46-55 years and 56-65 years

($p < 0.05$). However, HDL & LDL were not found significant in T2DM cases as compared to AMHCs in either of age group (Table 2).

Table 3: Distribution of BMI in Type 2 Diabetic Patients with age-matched Healthy Control

BMI (kg/m ²) → Variables ↓	Underweight (<18.5 kg/m ²)			Normal weight (18.5-22.9 kg/m ²)			Overweight (23-24.9 kg/m ²)			Obese (≥25 kg/m ²)			P-value
	Case (n=09)	Control (n=12)	P-value	Case (n=46)	Control (n=40)	P-value	Case (n=21)	Control (n=26)	P-value	Case (n=74)	Control (n=72)	P-value	
Gender (M/F)	4/5	8/4	0.39	25/21	23/17	0.83	15/5	18/8	0.75	52/22	38/34	0.04	0.29
Age (years)	44.67 ±13.57	53.83 ±10.08	0.09	47.63 ±10.83	46.73 ±12.19	0.72	54.05 ±9.33	48.46 ±10.29	0.06	47.55 ±10.60	47.64 ±12.12	0.96	0.15
Waist circumference (cm)	86.67 ±7.38	85.42 ±6.09	0.68	94.79 ±5.10	91.43 ±5.92	0.006	97.24 ±4.09	95.51 ±3.61	0.13	101.45 ±5.01	99.99 ±5.89	0.11	<0.001*
Waist-Hip Ratio (WHR)	0.87 ±0.07	0.85 ±0.06	0.49	0.95 ±0.05	0.91 ±0.06	0.001	0.97 ±4.09	0.96 ±0.04	0.39	1.01 ±0.05	1.00 ±0.06	0.28	<0.001*
SBP (mmHg)	127.44 ±18.34	114.50 ±5.72	0.03	148.07 ±33.13	115.75 ±5.90	<0.001	144.86 ±19.08	114.08 ±6.04	<0.001	136.65 ±23.17	113.86 ±8.86	<0.001	<0.001*
DBP (mmHg)	79.33 ±12.00	70.00 ±5.03	0.02	87.28 ±18.60	71.88 ±7.55	<0.001	78.95 ±18.60	71.00 ±7.04	0.05	80.69 ±12.01	69.03 ±10.02	<0.001	<0.001*
FBS (mg/ml)	155.78 ±74.81	88.50 ±10.47	0.006	173.98 ±95.02	92.45 ±9.34	<0.001	174.29 ±64.91	92.42 ±11.68	<0.001	161.97 ±57.89	96.00 ±12.12	<0.001	<0.001*
PPBS (mg/ml)	215.11 ±88.80	115.83 ±28.42	0.002	275.79 ±130.04	120.13 ±24.37	<0.001	259.29 ±100.67	128.69 ±25.29	<0.001	257.04 ±104.44	132.68 ±21.57	<0.001	<0.001*
HbA1c (%)	8.49 ±2.80	5.22 ±0.80	0.001	8.33 ±2.22	5.31 ±0.62	<0.001	7.66 ±1.82	4.98 ±0.72	<0.001	7.85 ±1.93	5.43 ±0.71	<0.001	<0.001*
Total Cholesterol (mg/dl)	143.53 ±33.6	127.87 ±16.82	0.18	166.62 ±41.88	158.63 ±48.83	0.42	158.80 ±59.43	154.25 ±41.91	0.76	175.96 ±59.04	160.85 ±47.58	0.09	0.05
Triglyceride (mg/dl)	156.29 ±64.23	129.19 ±49.06	0.29	192.18 ±141.57	131.33 ±49.87	0.01	161.93 ±64.51	144.35 ±66.75	0.37	188.34 ±106.21	157.94 ±76.07	0.048	0.02*
HDL (mg/dl)	35.93 ±8.08	41.70 ±9.41	0.16	40.57 ±10.38	42.83 ±10.62	0.32	33.71 ±11.29	38.36 ±8.32	0.11	37.43 ±11.96	37.96 ±12.11	0.79	0.06
LDL (mg/dl)	76.28 ±27.20	59.93 ±11.78	0.08	95.23 ±50.7	85.21 ±33.60	0.29	96.55 ±66.11	90.43 ±51.00	0.72	100.02 ±49.07	91.62 ±45.67	0.29	0.19
VLDL (mg/dl)	25.82 ±9.78	31.32 ±12.82	0.29	38.45 ±28.26	26.11 ±10.01	0.01	31.46 ±12.06	28.88 ±13.30	0.49	37.2 ±21.43	31.66 ±15.29	0.07	0.02*
Serum Creatinine (mg/dl)	1.60 ±0.85	0.94 ±0.21	0.02	2.45 ±1.47	0.93 ±0.22	<0.001	2.44 ±1.59	0.93 ±0.26	<0.001	2.31 ±1.62	0.88 ±0.26	<0.001	<0.001*

Values are expressed as Mean±SD (Standard Deviation). *P-value < 0.05 is significant.

WC – waist circumference, WHR – waist hip ratio, SBP – systolic blood pressure, DBP – diastolic blood pressure, FBS - Fasting Blood Sugar; Post - Prandial Blood Sugar; HbA1c - glycated haemoglobin; TC – total cholesterol, TG – triglyceride, HDL - High-Density Lipoprotein; LDL - Low-Density Lipoprotein; VLDL - Very Low-Density Lipoprotein, SCr – serum creatinine, BMI - Body Mass Index

Table 4: Correlation between variables in T2DM cases

	Age	WC	WHR	SBP	DBP	FBS	PPBS	HbA1c	TC	TG	HDL	LDL	VLDL	SCr	BMI
Age	1	0.09	0.09	0.16*	0.08	-0.11	-0.15	-0.14	-0.05	-0.11	0.09	0.02	-0.12	-0.07	0.02
WC	-	1	1.00**	-0.02	0.00	0.13	0.07	0.04	0.08	-0.02	-0.06	0.05	-0.02	0.10	0.65**
WHR	-	-	1	-0.02	0.00	0.13	0.07	0.04	0.08	-0.02	-0.06	0.05	-0.02	0.10	0.65**
SBP	-	-	-	1	0.69**	-0.06	-0.00	-0.12	0.02	-0.06	0.12	0.04	-0.05	0.16	-0.10
DBP	-	-	-	-	1	-0.01	0.04	-0.01	0.05	-0.12	0.13	0.10	-0.11	0.12	-0.10
FBS	-	-	-	-	-	1	0.84**	0.59**	0.01	-0.06	-0.01	0.05	-0.06	0.02	-0.00
PPBS	-	-	-	-	-	-	1	0.56**	0.05	0.03	-0.02	0.08	0.02	-0.01	-0.00
HbA1c	-	-	-	-	-	-	-	1	-0.01	0.00	0.03	-0.04	-0.01	-0.13	-0.09
TC	-	-	-	-	-	-	-	-	1	0.37**	0.34**	0.74**	0.39**	-0.03	0.18*
TG	-	-	-	-	-	-	-	-	-	1	-0.05	-0.01	0.99*	0.02	0.07
HDL	-	-	-	-	-	-	-	-	-	-	1	0.16	-0.05	-0.09	-0.13
LDL	-	-	-	-	-	-	-	-	-	-	-	1	-0.01	-0.01	0.20*
VLDL	-	-	-	-	-	-	-	-	-	-	-	-	1	0.03	0.07
SCr	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-0.05
BMI	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1

** correlation is significant at the 0.01 level (2-tailed) * correlation is significant at the 0.05 level (2-tailed)

WC – waist circumference, WHR – waist hip ratio, SBP – systolic blood pressure, DBP – diastolic blood pressure, FBS - Fasting Blood Sugar; Post - Prandial Blood Sugar; HbA1c - glycated haemoglobin; TC – total cholesterol, TG – triglyceride, HDL - High-Density Lipoprotein; LDL - Low-Density Lipoprotein; VLDL - Very Low-Density Lipoprotein, SCr – serum creatinine, BMI - Body Mass Index

Table 3 shows the distribution of BMI in T2DM with AMHCs. All subjects were categorized into four groups as underweight, normal weight, overweight and obese based on BMI value. Among T2DM cases, maximum number of cases

were found under obese category (49.3%, BMI ≥25kg/m²) and followed by cases with normal weight (30.7%, BMI between 18.5-22.9 kg/m²), overweight (14%, BMI between 23-24.9 kg/m²) and underweight (6%, BMI <18.5 kg/m²). Nearly half of the

individuals (48%) of the control group belonged to obese category.

Significantly higher mean FBS, PPBS and HbA1c were observed in T2DM as compared to AMHCs ($p < 0.001$, $p < 0.001$ and $p < 0.001$, respectively) in all groups of BMI. No significant mean WC and WHR were observed in three BMI groups; underweight ($p = 0.66$ and $p = 0.48$), overweight ($p = 0.13$ and $p = 0.39$) and obese ($p = 0.11$ and $p = 0.28$) of T2DM as compared to AMHCs. However, significant mean WC and WHR were observed in T2DM as compared to AMHCs in normal weight ($p = 0.006$ and $p = 0.001$ respectively). SBP and DBP were found statistically significant in T2DM as compared to AMHCs in all BMI groups. Additionally, TG (normal weight, $p = 0.01$; obese, $p = 0.04$), and VLDL (normal weight, $p = 0.01$) were found significant in T2DM as compared to AMHCs. However TC, HDL and LDL were

not found significant in T2DM when compared with AMHCs in all BMI groups. Significantly higher mean SCr was observed in T2DM as compared to AMHCs in all BMI groups.

We found significant high positive correlation of BMI in T2DM cases with WC ($r = +0.65$, $p < 0.001$) and WHR ($r = +0.65$, $p < 0.001$). Further, BMI had positive correlation with HbA1c ($r = 0.18$, $p < 0.05$) as well as LDL also ($r = 0.20$, $p < 0.05$) (Table 4).

A multiple linear regression analysis was performed to find the predictor of BMI based on anthropometric & biochemical variables (Table 5). A significant regression equation was found ($F(10, 139) = 14.05$, $p < 0.000$), with an R^2 of 0.503. The mean BMI in T2DM cases increased 46.5 kg/m^2 for each unit of WHR. Only WHR was a significant predictor of BMI but HDL as well as SCr was the weak predictor of BMI.

Table 5: Multiple linear regression analysis to show the dependence of BMI on study parameters in T2DM cases (N=150)

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95% Confidence Interval for B	
	B	Std. Error	Beta			Lower Bound	Upper Bound
(Constant)	-15.650	4.760		-3.288	0.001	-25.061	-6.238
SBP	-0.004	0.015	-0.021	-0.250	0.803	-0.033	0.026
DBP	-0.022	0.025	-0.074	-0.883	0.379	-0.072	0.027
FBS	-0.006	0.008	-0.100	-0.850	0.397	-0.021	0.008
PPBS	0.004	0.005	0.088	0.770	0.443	-0.006	0.013
HbA1c	-0.258	0.173	-0.115	-1.494	0.137	-0.601	0.084
TC	0.005	0.008	0.056	0.591	0.556	-0.011	0.021
HDL	-0.050	0.026	-0.126	-1.906	0.059	-0.101	0.002
LDL	0.014	0.009	0.144	1.589	0.114	-0.003	0.032
SCr	-0.266	0.138	-0.120	-1.930	0.056	-0.538	0.006
WHR	46.555	4.381	0.652	10.627	0.000	37.893	55.217

Dependent variable: BMI, $p < 0.05$ is considered significant at 95% confidence interval

SBP – systolic blood pressure, DBP – diastolic blood pressure, FBS - Fasting Blood Sugar; Post - Prandial Blood Sugar; HbA1c - glycated haemoglobin; TC – total cholesterol, HDL - High-Density Lipoprotein; LDL - Low-Density Lipoprotein; VLDL - Very Low-Density Lipoprotein, SCr – serum creatinine, WHR – waist hip ratio

DISCUSSION

In present study, 300 subjects (150 T2DM with 150 AMHCs) were enrolled from urban as well as rural population of Uttar Pradesh in north India. The maximum load of T2DM was found in the age groups of 36-45 years and 46-55 years (30% and 26.7%, respectively). The mean of BMI was higher in T2DM patients and AMHCs subjects grouped age of 36-45 years and 46-55 years. Result showed that most of the individuals belonged to obese category (49.3% subjects of T2DM patients and 48% subjects of AMHCs). Present study

indicates that the major risk factor for developing diabetes and its complications are increase in prevalence of overweight and obesity in all age group which is almost similar to the previous observation of ~30-65% Indians being overweight or obese. [29] A report from CDC states that most adults with diagnosed diabetes were overweight or obese. The survey reported from 1999-2002, showed that prevalence of overweight/obesity was 85.2%, whereas prevalence of obesity was 54.8%. [30] Overweight and obesity in early adulthood and weight gain at middle age were both independently

associated with the increased risk of T2DM in middle-aged men and women. [31]

WC and WHR were found statistically significant but BMI was insignificant all age groups in our study. These two variables (WC and WHR) have been used as measures of central adiposity and suggested a greater association with future metabolic risk than BMI (which is a measure of general obesity). Between WC and WHR, several studies have shown that WC is a better predictor of metabolic syndrome because of variations in the levels of hip measurements, and have different cut-off in various ethnic groups as well as in gender [32,33] which is contrary to our finding. In our study, the higher SCr levels were found in T2DM cases as compared to AMHCs, this might be due to higher WC and WHR but not BMI in our study groups. A Chinese study reported that WC is more strongly associated with CKD than BMI in patients with T2DM. [34] Similarly, results were reported from North America and they concluded that WC and WHR are the better markers for visceral fat in CKD. [35] On the other hand, general obesity measured by BMI could not distinguish fat and muscle mass while more muscle mass results in higher SCr concentration. [36] WC and its related values are being widely used as indicator of abdominal adiposity, as they are correlated with abdominal fat mass and are more related to CVD rather than BMI. [37] Despite variations in ethnicity, studies have shown that BMI is unable to discriminate between excess adipose tissue and high lean muscle mass and does not correspond for body fat distribution. [38]

Significantly higher mean FBS, PPBS and HbA1c were observed in T2DM as compared to AMHCs in all age groups as well as all BMI groups. A recently published study from Korea showed that poor glycemic control was observed in T2DM patients who have higher BMI. The findings of the present study indicated that more frequent screening for T2DM and strict glycemic control are required for overweight or obese T2DM patients,

because the prevalence of CVD in T2DM patients increased with increasing obesity. [39]

In another study, Mohan et al. observed that long-term survivors of T2DM patient had better blood sugar and blood pressure control with more favourable lipid profiles. [7] Sheth et al. also observed that obese T2DM patients, who have poorly controlled HbA1c, have shown dyslipidemia leading to increase CVD. [40]

In our study, the younger and middle aged patients had significantly elevated TG, TC & VLDL. This might be possible due to our presently increasing sedentary life style. Obesity in India has a direct correlation with the increasing prevalence of obesity-related co-morbidities like hypertension, dyslipidemia, T2DM and CVD. [41,42] An increased dyslipidemia is likely to increase FBS, PPBS and HbA1c and vice versa as the correlation between these parameters are directly proportional when comparing with age and BMI distribution in this study subjects. Reduction in HbA1c and blood sugar level in T2DM is associated with improved insulin sensitivity and better lipid parameters. [43]

In our study group SBP and DBP were found significant in age-wise as well as BMI-wise distribution of all groups. This study showed that mean SBP and prevalence of hypertension increased linearly with age, whereas DBP increased with age up to ~55 years and consistently low thereafter mainly in AMHCs. The age-related changes in SBP and DBP in the study are consistent with previous studies. [44,45] Changes in SBP and DBP in elderly individuals were shown to be an independent risk factor for CVD. [44]

Significantly higher mean SCr were observed in T2DM as compared to AMHCs in all age groups. Similarly, significantly higher mean SCr were observed in T2DM as compared to AMHCs in all BMI groups. In age group of 25-35 years, SCr is 2.9-folds higher in T2DM patients than AMHCs. This might be due to the poor glycemic control, because the HbA1c is 1.6-folds higher in T2DM patients than AMHCs in this age

group. Higher SCr, hypertension and poor glycemic control are the major causes of CKD with increasing age and obesity. [45,46] A study reported that High HbA1c levels associated with CKD in non-DM patients also. Renal function in patients with high HbA1c levels may need to be monitored regularly to prevent the development of CKD in the patients of T2DM as well as non-T2DM. [47]

It was observed that between 25 to 45 years of age groups FBS, PPBS, HbA1c, TG, TC and SCr level were found multiple folds higher in T2DM patients as compared to AMHCs. Higher blood sugar, disturbed lipid profile and SCr are the alarming stage in these early and middle age groups, might be due to lack of awareness and improper knowledge. This will invite early complications and related comorbidities of diabetes and obesity. Another study demonstrated that FBS had positive correlation with TC, TG and VLDL. Also, FBS had found to be the predictor of hypercholesterolemia. [48] One more possible reason that in our study group 49.3% and 48% obese were found in T2DM and AMHCs, respectively. One more concern is that as this is the most reproductive age groups (25 to 45 years), poor glycemic control might affect their fertility and reduce the chances to enjoy parenting. A study reports that a diabetic man who has uncontrolled blood sugar level has less chance of impregnating his partner and when he did, the risk of miscarriage and deformities are much higher. [49] McGrogan et al. has reported that the proportion of pregnancy losses is higher in women with either type I or type II diabetes (approximately 20%) as compared to that in general population (13.2%). [50] Poor glycemic control and impaired insulin sensitivity are associated with increased risk of erectile dysfunction. [51]

CONCLUSION

WC and WHR is an important marker for obesity than BMI in our study. WHR was the only significant predictor of BMI. In age and BMI wise distribution,

dyslipidemia, obesity and hypertension are significantly associated with poorly controlled fasting blood sugar, post-prandial blood sugar, glycated haemoglobin, and SCr in T2DM with AMHCs. Additionally, higher prevalence of dyslipidemia, obesity and hypertension with T2DM in the study subjects suggesting their possible role as biomarker and early detection for CVD, CKD, and other diabetic complications. Mainly, younger (25-35 years) and middle (36-45 years) age group have lipid profile abnormalities and poor glycemic controls in this study.

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Contributions

M.M.K., G.K.S., S.K.S., A.K., developed the concepts and design of the study. M.M.K., G.K.S., S.K.S., A.K., and R.A., S.M., M.S.K., gave final approval of the version to be published.

REFERENCES

1. Pathania M, Dutt HK, Gogoi JB, Rathaur V, Singh G, Singh P. Study the Impact of Diabetes Camps on Adherence to Medication and Glycaemic Control in Uttarakhand. *Journal of Clinical and Diagnostic Research*, 2016; Feb, Vol-10(2): OC22-OC26.
2. Ben-Salem A, Ajina M, Suissi M, Daher HS, Almawi WY, Mahjoub T. Polymorphisms of transcription factor-7-like 2 (TCF7L2) gene in Tunisian women with polycystic ovary syndrome (PCOS). *Gene*, 2014; 533: 554-7.
3. IDF Diabetes Atlas. International Diabetes Federation 7th Edition, 2015; Nov. 29.
4. Anjana RM, Pradeepa R, Deepa M, Datta M, Sudha V, Unnikrishnan R, et al. ICMR-INDIAB Collaborative Study Group Prevalence of diabetes and prediabetes (impaired fasting glucose and/or impaired glucose tolerance) in urban and rural India: Phase I results of the Indian Council of Medical Research-India DIABetes (ICMR-INDIAB) study. *Diabetologia*, 2011; 54:3022-7.
5. Mohan V, Sandeep S, Deepa R, Shah B & Varghese C. Epidemiology of type 2 diabetes: Indian scenario. *Indian J Med Res*, 2007; 125:217-230.
6. Mohan V, Shanthirani CS, Deepa M, Deepa R, Unnikrishnan RI, Datta M. Mortality rates due

- to diabetes in a selected urban South Indian population -the Chennai Urban Population Study (CUPS). *J Assoc Physicians India*, 2006; 54:113-7.
7. Mohan V, Rani CSS, Amutha A, Dhulipala S, Anjana RM, Parathasarathy B, Unnikrishnan R. Clinical Profile of Long-Term Survivors and Nonsurvivors With Type 2 Diabetes. *Diabetes Care*, 2013; 36: 2190-2197.
 8. Jensen MD, Ryan DH, Apovian CM, et al. 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: a report of the American College of Cardiology/ American Heart Association Task Force on Practice Guidelines and The Obesity Society. *Circulation*, 2014; 129: S102-38.
 9. Flegal KM, Kit BK, Orpana H, Graubard BI. Association of All-Cause Mortality with Overweight and Obesity Using Standard Body Mass Index Categories. *JAMA*, 2013; 309:71-82.
 10. World Health Organization (WHO). Obesity: preventing and managing the global epidemic. Report of a WHO consultation. (1-253). *World Health Organ Tech Rep Ser*, 2000; 894: i-xii.
 11. Geneva: WHO. *World Health Statistics*, 2012; November, 8.
 12. Fan H, Li X, Zheng L. Abdominal obesity is strongly associated with Cardiovascular Disease and its Risk Factors in Elderly and very Elderly Community-dwelling Chinese. *Sci Rep.*, 2016; 6:21521.
 13. Hanis CL, Redline S, Cade BE, et al. Beyond type 2 diabetes, obesity and hypertension: an axis including sleep apnea, left ventricular hypertrophy, endothelial dysfunction and aortic stiffness among Mexican Americans in Starr County, Texas. *Cardiovasc Diabetol.*, 2016; 15:86.
 14. Nayak SB, Maharaj N, Fatt LaL. Association between altered lipid profile, body mass index, low plasma adiponectin and varied blood pressure in Trinidadian type 2 diabetes and non-diabetic subjects. *Indian J Med Sci*, 2012; 66:214-21.
 15. Mandal A. Study of prevalence of type 2 diabetes mellitus and hypertension in overweight and obese people. *J Family Med Prim Care*, 2014; Jan-Mar; 3(1):25-28.
 16. American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care*, 2010; 33 (Suppl 1): S62-S69.
 17. Diabetes Control and Complications Trial Research Group: The absence of a glycemic threshold for the development of long-term complications: the perspective of the Diabetes Control and Complications Trial. *Diabetes*, 1996; 45:1289 -1298.
 18. UK Prospective Diabetes Study (UKPDS) Group: Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). *Lancet*, 1998; 352:837-853.
 19. Vikøren TB, Berg JP, Berg TJ. Sources of error when using haemoglobin A1c. *Tidsskr Nor Laegeforen* 2014 Feb 25; 134(4):417-21.
 20. The International Expert Committee. International expert committee report on the role of the A1C assay in the diagnosis of diabetes. *Diabetes Care*, 2009; 32:1327-34.
 21. Iglay K, Hannachi H, Joseph Howie P, Xu J, Li X, Engel SS, Moore LM, Rajpathak S. Prevalence and co-prevalence of comorbidities among patients with type 2 diabetes mellitus. *Curr Med Res Opin.*, 2016; Apr 4:1-10.
 22. American Diabetes Association. Standards of medical care in diabetes-2015. *Diabetes Care*, 2015; 38 Suppl 1: S1-S10.
 23. Executive summary: Standards of medical care in diabetes. *Diabetes Care*, 2010; 33(Suppl 1):S4-10.
 24. Alberti K, Zimmet P, Shaw J. Metabolic syndrome-a new world-wide definition. A consensus statement from International diabetes federation. *Diabet Med.*, 2006; 23(5): 469-480.
 25. Verma BL, Kumar A, Srivastava RN. Measurement of body build based on weight/height, an index for adults in an Indian population. *Ind J Pub Health*, 1982; 26, 133-143.
 26. Frisancho AR. New standards of weight and body composition by frame size and height for assessment of nutritional status in adults and elderly. *Am J Clin Nutr*, 1984; 40, 808-819.
 27. International Diabetes Federation. The IDF consensus worldwide definition of the metabolic syndrome. *International Diabetes Federation*, 2006.
 28. NICE guidelines [PH46] Published date: July 2013. BMI: preventing ill health and premature death in black, Asian and other minority ethnic groups. *NICE guidelines [PH46]*.
 29. Misra A, Khurana L. Obesity and the metabolic syndrome in developing countries. *J Clin Endocrinol Metab.*, 2008; 93(11):9-30.
 30. Eberhardt MS, Ogden C, CDC group et al. Prevalence of Overweight and Obesity Among Adults with Diagnosed Diabetes---United States, 1988-1994 and 1999-2002. *Morbidity and Mortality Weekly Report*, 2004; 53(45):1066-1068.
 31. Zhou L, Zhao L, Li Y, Guo M, Wu Y. [Relationship between body weight status in early adulthood and body weight change at middle age in adults and type 2 diabetes mellitus]. *Zhonghua Liu Xing Bing Xue Za Zhi.*, 2016; 37(3):339-43.
 32. Rajput R, Rajput M, Bairwa M, Singh J, Saini O, Shankar V. Waist height ratio: A universal screening tool for prediction of metabolic syndrome in urban and rural population of Haryana. *Indian J Endocr Metab*, 2014;

- 18:394-9.
33. Wang F, Wu S, Song Y, Tang X, Marshall R, Liang M, et al. Waist circumference, body mass index and waist to hip ratio for prediction of the metabolic syndrome in Chinese. *Nutr Metab Cardiovasc Dis*, 2009; 19:542-7.
 34. Xu L, Yu W, Huang P, Li C, Li Y, Wang M, Xu Q, Wang J, Zheng C, Qu B, Zhao Y, Niu M, Wang O, and Gong F. The age-specific association of waist circumference and risk of chronic kidney disease in patients with type 2 diabetes mellitus in Shandong, China. *International Journal of Endocrinology*, 2015; Article ID 715871, 6 pages. DOI 10.1155.2015.715871.
 35. Sanches FMR, Avesani CM, Kamimura MA et al. Waist circumference and visceral fat in CKD: a cross-sectional stud. *The American Journal of Kidney Diseases*, 2008; vol. 52, no. 1, pp. 66-73.
 36. Maric-Bilkan C,. Obesity and diabetic kidney disease. *Medical Clinics of North America*, 2013; vol. 97, no. 1, pp.59-74.
 37. Liu et al. Can body mass index, waist circumference, waist-hip ratio and waist-height ratio predict the presence of multiple metabolic risk factors in Chinese subjects? *BMC Public Health*, 2011; 11:35.
 38. Bener A, Yousafzai MT, Darwish S, et al. Obesity Index That Better Predict Metabolic Syndrome: Body Mass Index, Waist Circumference, Waist Hip Ratio, or Waist Height Ratio. *Journal of Obesity*, 2013; Volume, Article ID 269038,9 pages.
 39. Lee DH, Jung KY, Kim KM, Moon JH, Lim S, Jang HC, Choi SH. Characterization of Patients with Type 2 Diabetes according to Body Mass Index: Korea national Health and Nutrition Examination Survey from 2007 to 2011. *Endocrinol Metab*, 2015; 30:514-521.
 40. Sheth J, Shah A, Sheth F, Trivedi S, Nabar N, Shah N, Thakor P and Vaidya R. The association of dyslipidemia and obesity with glycated haemoglobin. *Clinical Diabetes and Endocrinology*, 2015. DOI 10.1186/s40842-015-0004-6.
 41. Gupta R, Gupta VP, Sarna M, et al. Prevalence of coronary heart disease and risk factors in an urban Indian population: Jaipur Heart Watch-2. *Indian Heart J*, 2002; 54(1):59-66.
 42. Gupta R, Misra A. Type 2 diabetes in india: Regional Disparities. *Br J Diabetes & Vascular Dis*, 2007; 7:12-16.
 43. Mahato RV, Gyawali P, Raut P, Regmi P, Singh K, Pandeya DR, et al. Association between glycemic control and serum lipid profile in type 2 diabetic patients: Glycated haemoglobin as a dual biomarker. *Biomedical Research*, 2011; 22(3):375-80.
 44. Franklin SS. Ageing and hypertension: The assessment of blood pressure indices in predicting coronary heart disease. *J Hypertens Suppl*, 1999; 17: S29-S36.
 45. Murakata Y, Fujimaki T, Yamada A. Age-related changes in clinical parameters and their associations with common complex diseases. *Biomedical Reports*, 2015; 3: 767-777.
 46. Lingling Xu, Weihong Yu, PingHuang, Chunying Li, Yan Li, Meng Wang, Qun Xu, Jing Wang, Caixia Zheng, Bin Qu, Yanping Zhao, Meng Niu, Ou Wang, and Fengying Gong. The Age-Specific Association of Waist Circumference and Risk of Chronic Kidney Disease in Patients with Type 2 Diabetes Mellitus in Shandong, China. *International Journal of Endocrinology*, 2015; Volume, Article ID 715871, 6 pages.
 47. Kang SH, Jung DJ, Choi EW, Cho KH, Park JW, Do JY. HbA1c Levels Are Associated with Chronic Kidney Disease in a Non-Diabetic Adult Population: A Nationwide Survey (KNHANES 2011-2013). *PLoS ONE*, 2015; 10(12): e0145827.
 48. Khadke S, Harke S, Ghadge A, Kulkarni O, Bhalerao S, Diwan A, Pnkaj M and Kulkalkar A. Association of Fasting Plasma Glucose and Serum Lipids in Type 2 Diabetics. *Indian J Pharm Sci*, 2015; 77(5):630-634.
 49. Agbaje I.M., Rogers D.A., McVicar C.M., McClure N., Atkinson A.B., Mallidis C.and Lewis S.E.M. Insulin dependent diabetes mellitus: implications for male reproductive function. *Hum. Reprod.*, 2007; 22 (7): 1871-1877.
 50. McGrogan A, Snowball J and de Vries CS. Pregnancy losses in women with Type 1 or Type 2 diabetes in the UK: an investigation using primary care records. *Diabet. Med.*, 2014; 31:357-365.
 51. Aviva E. Weinberg, Eisenberg M, Patel CJ, Chertow GM, and Leppert JT. Diabetes Severity, Metabolic syndrome, and the risk of Erectile Dysfunction. *J Sex Med.*, 2013; 10(12): 3102-3109.

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