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Exploring Insulin Resistance in Obesity: A Cross-Sectional Study from Arunachal Pradesh

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Abstract:

Background: Obesity and metabolic syndrome are prominent worldwide health issues that substantially elevate the likelihood of developing a range of diseases, including cancer. Chronic, mild inflammation and a decreased body response to insulin are important variables that link fat to cancer development. The aim of this study was to examine the relationship between insulin resistance and the likelihood of developing cancer in overweight or obese patients, regardless of whether they have metabolic syndrome or not.

Methods: We conducted a cross-sectional investigation with a total of 200 participants. We categorised the participants into four distinct groups: 50 individuals in good health, 50 overweight individuals, 50 obese individuals without metabolic syndrome, and 50 obese individuals with metabolic syndrome. We used the enzyme-linked immunosorbent assay (ELISA) to determine serum insulin levels and the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) to evaluate insulin resistance. We also assessed anthropometric and biochemical markers.

Results: Compared to the control group (4.7 3.0 IU/ml), individuals who were overweight (10.8±0.8 µIU/ml), obese without metabolic syndrome (14.2±2.4 µIU/ml), and obese with metabolic syndrome (20.3±6.3 µIU/ml) had significantly higher levels of serum insulin. The HOMA-IR values were higher in the overweight and obese groups compared to the control group, suggesting increased insulin resistance. These data indicate that obesity worsens insulin levels and insulin resistance, perhaps leading to an increased risk of cancer.

Conclusion: The study emphasises a substantial correlation between obesity, insulin resistance, and increased cancer susceptibility. To effectively reduce this risk, it is essential to implement interventions that focus on weight loss and improving metabolic health. Timely detection and proactive measures are critical for effectively managing obesity issues and decreasing cancer incidence.

Keywords:Serum Insulin, Overweight, Obesity, Metabolic Syndrome, Insulin resistance

Introduction:

Obesity has become a significant worldwide health issue, with a concerning rise in the number of people categorised as overweight or obese. This trend is supported by multiple studies that indicate a significant increase in obesity rates globally, including in India. Recent global figures indicate that there are around 650 million individuals who are obese, and approximately 1.9 billion who are overweight. In India, the prevalence of obesity has significantly increased from 11.8% to 31.3%, and the occurrence of central obesity has climbed from 16.9% to 36.3% as reported by the ICMR-INDIAB study done in 2015 [1]. Despite these alarming statistics, obesity has not yet garnered the recognition it deserves as a significant health risk. Unemployment, decreased socioeconomic productivity, and social inequality have a major impact on the prevalence of obesity [2]. Studies indicate that obesity is mostly caused by an inequity between the amount of energy consumed and the amount of energy expended, often accompanied by chronic inflammation in certain forms of obesity. This syndrome is defined by the expansion of adipose tissue, which experiences cellular and molecular modifications that subsequently disrupt and hinder systemic metabolism [3].

The current body of research unambiguously indicates that obesity substantially heightens the likelihood of developing a range of illnesses, including type 2 diabetes, cardiovascular diseases, and multiple forms of cancer—such as those impacting the liver, kidney, colon, prostate, ovaries, breast, endometrium, and gall bladder. Central obesity, specifically, significantly contributes to metabolic syndrome, which increases the incidence of these concomitant illnesses by two-fold [4-6]. On a global scale, the prevalence of metabolic syndrome in adults is roughly 20-25%, while in India it is around 30% [7]. Obesity is a well-documented effect of causing insulin resistance, which is a physiological condition characterised by reduced responsiveness of the body's cells to insulin. Insulin resistance is a characteristic feature of metabolic syndrome and has been linked to an elevated risk of cancer, as evidenced by multiple epidemiological studies [8-12]. Insulin, secreted by the β cells of the pancreas, enhances the absorption of glucose into cells from the bloodstream, namely in adipocytes, hepatocytes, and skeletal muscle cells. Obesity often leads to insulin resistance due to disruption in the insulin signalling cascade, particularly in the IRS/PI-3K/PK-B pathway [13,14].

Recent studies on tumours connected to obesity have found a strong link between metabolic dysregulation and the occurrence of cancer. An elevation in body mass index (BMI) has been associated with a 20-50% increase in the detection of tumours that are related to obesity. Research has also demonstrated that the presence of insulin in the bloodstream can indicate the likelihood of developing cancer [15]. Insulin resistance is a well-documented part of the physiological consequences of metabolic syndrome. However, the specific processes by which fat causes insulin resistance are still not fully understood. There is a significant lack of research explicitly investigating the impact of insulin resistance on cancer related with obesity. In order to fill this void, our research sought to evaluate the levels of serum insulin in three specific categories of individuals in Arunachal Pradesh: individuals who are overweight, individuals who are obese but do not have metabolic disorders, and individuals who are obese and have metabolic diseases. The aim was to establish a correlation between serum insulin levels and several biochemical and anthropometric characteristics, in order to improve our comprehension of the connection between obesity, insulin resistance, and the risk of developing cancer.

The participants were classified into three groups, and their serum insulin levels were assessed using the Enzyme-Linked Immunosorbent Assay (ELISA). The assessment of insulin resistance was conducted using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR). The study assessed anthropometric measures, including BMI and waist circumference, as well as biochemical parameters, to investigate the correlation between obesity, insulin resistance, and the chance of developing cancer. The findings revealed a substantial increase in blood insulin levels in overweight and obese people in comparison to healthy controls. Significantly, people who are obese and have metabolic problems displayed the most elevated levels of serum insulin and HOMA-IR values, indicating a strong resistance to insulin. These findings indicate that fat worsens insulin resistance, perhaps leading to an increased risk of cancer. This study highlights the significance of promptly identifying and intervening in patients who are at risk of developing metabolic syndrome and its associated consequences. Implementing preventive strategies, such as reducing body weight and enhancing metabolic health, is essential for reducing the likelihood of developing cancer linked to obesity and insulin resistance. Additional investigation is required to clarify the fundamental processes that connect fat to insulin resistance and cancer, thereby creating opportunities for precise treatment approaches. Comprehending the complex correlation between obesity, insulin resistance, and the likelihood of developing cancer is crucial for the formulation of efficient public health strategies and interventions. By tackling obesity and its correlated metabolic dysfunctions, we can effectively diminish the impact of cancer and other linked disorders.

Materials and Methods:

Study design

The Tomo Riha Institute of Health & Medical Sciences in Naharlagun, Arunachal Pradesh recruited 200 volunteers, ranging in age from 20 to 70 years, for this study. We categorised the participants into four

groups: 50 individuals with overweight, 50 individuals with obesity but without metabolic syndrome, 50 individuals with obesity and metabolic syndrome, and 50 healthy controls. Before conducting the study, we sought approval from the Institutional Ethics Committee and gathered informed consent from all participants. The study excluded people who were obese due to secondary factors, had a documented medical history of diabetes, cardiovascular illnesses, cancer, or were undergoing relevant medication therapy.

Anthropometric Measurements

We used a beam balance and a wall-mounted tape to measure the height and weight, respectively. We recorded the weight to an accuracy of 0.1 kg and the height to the nearest centimeter. We computed the Body Mass Index (BMI) by dividing the weight (in kilogrammes) by the square of the height (in metres). We assessed the waist-to-hip ratio (WHR) by measuring the circumference of the waist at the umbilicus level and the diameter of the hips at their widest point. We then calculated the WHR by dividing the waist circumference by the hip circumference. We measured the neck circumference (NC) with precision, using a non-stretchable plastic tape at a point halfway between the mid-cervical spine and the mid-anterior neck, with an accuracy of 1 mm. We measured the NC just below the laryngeal prominence in men [8].

Categorization and Diagnosis Criteria

We categorised the participants into two groups based on their body mass index (BMI): overweight (BMI between 25 and 29.9) and obese (BMI greater than 30).The International Diabetes Federation (IDF) established criteria to diagnose metabolic syndrome. People are said to have metabolic syndrome if they are overweight (waist circumference more than 94 cm for men and more than 80 cm for women) and have at least two of the following conditions: triglyceride levels equal to or greater than 150 mg/dl, HDL levels less than 40 mg/dl for men and less than 50 mg/dl for women, blood pressure equal to or greater than 130/85 mm Hg, and fasting blood glucose levels above 100 mg/dl.We obtained venous blood samples from all participants after they abstained from food for a 12-hour period. We isolated and preserved the serum at a temperature of -20 °C until its examination.

Insulin Quantification

We conducted an enzymatic analysis on the lipid profile, which included total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C), using an automated analyzer called Agappe. We used the GPO-PAP method to test serum triglycerides (TG), and the GOD-POD method to measure fasting blood glucose. We used ELISA (Diametra) to quantify serum insulin levels. We determined the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) by multiplying the fasting glucose level (in milligrammes per deciliter) by the fasting insulin level (in microunits per millilitre), and then dividing the result by 22.5. We used mercury sphygmomanometers to record the blood pressure values of all subjects [8].

Statistical analysis

We conducted the statistical analysis using SPSS version 23.0 (SPSS Inc., Chicago, IL, USA). Descriptive statistics provide a summary of categorical variables in terms of frequencies and percentages, as well as continuous variables in terms of mean plus or minus standard deviation (SD). The inferential statistics utilised in this study encompassed t-tests and correlation analyses for numerical data, as well as the chi-square test for categorical data. We employed the Pearson correlation coefficient at a significance level of 5%.This methodology guarantees a thorough analysis of the correlation between obesity, metabolic syndrome, and insulin resistance in the population under study. The comprehensive techniques offer a precise framework for reproducing the study and verifying its results [9] .

Results:

We examined a cohort of 200 subjects, comprising 69 men and 131 women, with ages ranging from 20 to 70 years. This cohort was divided into four groups: 50 healthy controls, 50 overweight individuals, 50

obese individuals without metabolic syndrome, and 50 obese individuals with metabolic syndrome. Forty-eight subjects were excluded from the study due to refusal to participate, incomplete data, or pre-existing diagnoses of various disorders. Table 1 displays the biochemical and anthropometric characteristics of the study participants. Anthropometric parameters were notably higher in overweight individuals and those with obesity, regardless of metabolic syndrome status, compared to healthy controls. However, waist-to-hip ratio (WHR) did not significantly differ among the groups. Serum insulin levels exhibited a significant increase in overweight individuals (10.8±0.8 µIU/ml), obese individuals without metabolic syndrome (14.2±2.4 µIU/ml), and obese individuals with metabolic syndrome (20.3±6.3µIU/ml) ($p<0.001$) compared to the control group (4.7±3.0µIU/ml). These findings highlight the association between obesity, particularly in the presence of metabolic syndrome, and elevated serum insulin levels, indicating a potential link to insulin resistance (Table-1) .

Table 1: Comparison between control and overweight, obese with and without metabolic syndrome

Parameter	Control (N= 50)	Overweight (N= 50)	Obese without Met S(N= 50)	Obese with Met S (N= 50)
Age (Years)	41.5 ± 13.4	37.1 ± 10.9	40.6 ± 12.7	42.0 ± 9.2
BMI (Kg/m^2)	21.5 ± 1.8	27.2 ± 1.2**	31.4 ± 1.9**	32.8 ± 2.7**
WHR	0.8 ± 0.1	0.8 ± 0.1 ł	0.9 ± 0.0**	0.9 ± 0.1**
NC	14.1 ± 0.8	14.6 ± 0.7*	15.8 ± 1.0**	16.2 ± 1.1**
Glucose(mg/dl)	90.2 ± 8.8	95.2 ± 10.7*	94.9 ± 8.5*	108.5 ± 10.4**
TC(mg/dl)	167.5 ± 32.3	177.1 ± 37.7ł	190.4 ± 26.8*	219.6 ± 42.3**
TG(mg/dl)	121.9 ± 33.0	162.8 ± 6.8**	150.2 ± 41.2**	242.0 ± 9.61**
HDL-C(mg/dl)	48.8 ± 11.6	43.2 ± 9.1*	49.1 ± 6.2 ł	40.6 ± 7.0*
LDL-C(mg/dl)	92.2 ± 26.2	104.3 ± 36.1ł	107.5 ± 22.4*	135.0± 40.5**
VLDL(mg/dl)	24.1 ± 6.8	32.5 ± 13.6*	29.8 ± 8.2*	48 ± 18.8**
S.B.P (mmHg)	116.2 ± 6.4	117.6 ± 5.6ł	117.8 ± 6.8 ł	133.8 ± 10.7**
D.B.P (mmHg)	79.6 ± 6.7	79.4 ± 5.5 ł	79.8 ± 6.8 ^{NS}	87.8 ± 8.2**
Insuin(µIU/ml)	4.7±3.0	10.8±0.8**	14.2±2.4**	20.3±6.3**
HOMA-IR	1.1±0.6	2.5±0.4**	3.3±0.7**	5.4±1.6**

** $p<0.001$, * $p<0.005$, ł $p>0.05$

BMI – Body Mass Index, WHR – Waist to Hip Ratio, NC – Neck Circumference

TC – Total Cholesterol, TG – Triglycerides, HDL-C – High Density Lipoprotein- Cholesterol, LDL-C – Low Density Lipoprotein- Cholesterol, VLDL – Very Low Density Lipoprotein, S.B.P – Systolic Blood Pressure, D.B.P – Diastolic Blood Pressure, Met S – Metabolic Syndrome

The relationship between serum insulin, biochemical and anthropometric parameters for all the subjects are shown in Table 2. The Pearson correlation analysis revealed significant associations between serum insulin levels and various anthropometric and biochemical variables among the study subjects. Body Mass Index (BMI) exhibited a strong positive correlation with serum insulin levels ($r = 0.76$, $p = 0.001$), indicating that higher BMI values were associated with elevated insulin levels. Similarly, Waist to Hip Ratio (WHR) ($r = 0.44$, $p = 0.001$) and Neck Circumference (NC) ($r = 0.55$, $p = 0.001$) showed significant positive correlations with serum insulin levels. Glucose levels ($r = 0.41$, $p = 0.001$), Total Cholesterol (TC) ($r = 0.37$, $p = 0.001$), Triglycerides (TG) ($r = 0.44$, $p = 0.001$), Very Low Density Lipoprotein (VLDL) ($r = 0.44$, $p = 0.001$), Systolic Blood Pressure (S.B.P) ($r = 0.49$, $p = 0.001$), and Diastolic Blood Pressure (D.B.P) ($r = 0.34$, $p = 0.001$) also exhibited positive correlations with serum insulin levels. However, High Density Lipoprotein- Cholesterol (HDL-C) showed a negative correlation ($r = -0.19$, $p = 0.002$) with serum insulin levels. Additionally, Low Density Lipoprotein- Cholesterol (LDL-C) ($r = 0.34$, $p = 0.001$) and Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) ($r = 0.27$, $p = 0.001$) demonstrated significant positive correlations with serum insulin levels. These results underscore the intricate relationship between insulin levels and various anthropometric and biochemical parameters,

suggesting a potential role of insulin in metabolic dysregulation associated with obesity and related conditions.

Table 2: Pearson correlation analysis between serum Insulin and anthropometric, biochemical variables of the study subjects.

Parameter	r value	p value
BMI	0.76	0.001
WHR	0.44	0.001
NC	0.55	0.001
Glucose	0.41	0.001
TC	0.37	0.001
TG	0.44	0.001
HDL-C	-0.19	0.002
LDL-C	0.34	0.001
VLDL	0.44	0.001
S.B.P	0.49	0.001
D.B.P	0.34	0.001
HOMA-IR	0.27	0.001

BMI – Body Mass Index, WHR – Waist to Hip Ratio, NC – Neck Circumference
TC – Total Cholesterol, TG – Triglycerides, HDL-C – High Density Lipoprotein- Cholesterol, LDL-C – Low Density Lipoprotein- Cholesterol, VLDL – Very Low Density Lipoprotein, S.B.P – Systolic Blood Pressure, D.B.P – Diastolic Blood Pressure.

Table 3 presents a comparison between males and females across different groups based on various anthropometric and biochemical parameters. In Group I, comprising healthy controls, males exhibited a mean age of 42.1 years ± 12.7, while females had a mean age of 41.1 years ± 13.9. In Group II, which consisted of overweight individuals, both males and females showed an increase in BMI compared to Group I, with males at 27.0 kg/m2 ± 1.2 and females at 27.2 kg/m2 ± 1.1 ($p<0.001$). Similarly, Waist to Hip Ratio (WHR) and Neck Circumference (NC) were significantly higher in both males and females in Group II compared to Group I ($p<0.001$). Group III, composed of obese individuals without metabolic syndrome, exhibited further increases in BMI, WHR, NC, and other parameters in both males and females compared to Group II ($p<0.001$). Group IV, representing obese individuals with metabolic syndrome, showed the highest values across all parameters in both males and females compared to the other groups ($p<0.001$). Notably, serum insulin levels and Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) were markedly elevated in both males and females in Group IV compared to the other groups ($p<0.001$). Overall, these findings highlight significant gender-specific differences in anthropometric and biochemical parameters among individuals with varying degrees of obesity and metabolic health.

Table 3: Comparison between males and females of different groups.

Parameter	Group I		Group II		Group III		Group IV	
	Males (N= 17)	**Females (N= 33)**	**Males(N= 13)**	**Females (N= 37)**	**Males(N= 16)**	**Females (N= 34)**	**Males(N= 23)**	**Females (N= 27)**
AGE (Years)	42.1±12.7	41.1±13.9	37.6±12.2	36.8±10.6	42.1± 10.8	39.8± 13.5	41.5±9.1	42.3±9.5
BMI (Kg/m^2)	21.7±1.7	21.3±1.7	27.0±1.2**	27.2±1.1**	30.8±1.2**	31.7±2.1**	33.1±3.3**	32.5±1.9**

WHR	0.78±0.06	0.78±0.06	0.8±0.07ł	0.81±0.06ł	0.86±0.05**	0.86±0.04**	0.88±0.05**	0.87±0.04**
NC	14.6±0.69	13.8±0.74	14.9±0.65ł	14.5±0.68*	16.4±1.0**	15.4±0.87**	16.4±1.0**	15.9±1.0**
Glucose (mg/dl)	88.8±8.0	90.8±9.2	95.7±15.1ł	95.0±8.8ł	96.6±11.6ł	93.9±6.5ł	108.2±9.8**	108.7±11.0**
TC (mg/dl)	164.6±37.5	168.9±29.7	166.9±40.4ł	180.6±36.5ł	183.1±25.8ł	193.8±26.9**	223.0±41.0**	216.6±43.9**
TG (mg/dl)	120.6±34.3	122.5±32.7	171.7±61.9*	159.6±70.6*	137.8±18.2*	156.0±47.5**	261.1±132.1**	225.6±45.1**
HDLC (mg/dl)	48.5±11.6	48.9±11.7	41.3±6.5ł	43.8±9.8ł	47.0±3.4ł	50.1±6.9ł	39.3±7.1**	41.7±6.7**
LDL (mg/dl)	87.4±29.7	94.5±24.2	93.6±33.6ł	107.9±36.6ł	103.5±25.5ł	109.3±20.9*	139.9±38.1**	130.7±42.5**
VLDL (mg/dl)	24.0±6.9	24.1±6.8	34.2±12.3*	31.8±14.1*	27.3±3.6ł	30.9±9.4*	51.5±25.8**	45.0±9.0**
SBP (mmHg)	118.2±8.0	115.1±5.0	116.9±6.3ł	117.8±5.3ł	119.3±6.8ł	117.0±6.7ł	135.2±11.6**	132.5±9.8**
DBP (mmHg)	78.2±6.3	80.3±6.8	79.2±4.9ł	79.4±5.7ł	80.0±6.3ł	79.7±7.1ł	88.2±10.2**	87.4±5.9**
Insuin(µIU/ml)	4.3±2.8	4.9± 3.0	10.6±0.8**	10.7±0.8**	15.2±3.0**	13.7±1.8**	18.5±5.9**	21.8±6.1**
HOMA-IR	0.9±0.6	1.0±0.7	2.5±0.5**	2.5±0.3**	3.6±0.9**	3.2±0.5**	4.9±1.4**	5.8±1.7**

** p<0.001, * p<0.005, ł p>0.05

BMI – Body Mass Index, WHR – Waist to Hip Ratio, NC – Neck Circumference

TC – Total Cholesterol, TG – Triglycerides, HDL-C – High Density Lipoprotein- Cholesterol, LDL-C – Low Density Lipoprotein- Cholesterol, VLDL – Very Low Density Lipoprotein, S.B.P – Systolic Blood Pressure, D.B.P – Diastolic Blood Pressure.

Table 4 illustrates the Pearson correlation analysis between serum Insulin and various anthropometric and biochemical variables of the study subjects, stratified by gender. Among males (N=69), significant positive correlations were observed between serum Insulin levels and BMI (r=0.75, p=0.001), Waist to Hip Ratio (WHR) (r=0.44, p=0.001), Neck Circumference (NC) (r=0.43, p=0.001), Glucose levels (r=0.31, p=0.009), Total Cholesterol (TC) (r=0.38, p=0.001), Triglycerides (TG) (r=0.40, p=0.001), Low Density Lipoprotein-Cholesterol (LDL-C) (r=0.40, p=0.001), Very Low Density Lipoprotein (VLDL) (r=0.39, p=0.001), Systolic Blood Pressure (S.B.P) (r=0.43, p=0.001), Diastolic Blood Pressure (D.B.P) (r=0.30, p=0.01), and Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) (r=0.25, p=0.01). In females (N=131), similar positive correlations were observed between serum Insulin levels and BMI (r=0.76, p=0.001), WHR (r=0.43, p=0.001), NC (r=0.63, p=0.001), Glucose levels (r=0.48, p=0.001), TC (r=0.36, p=0.001), TG (r=0.48, p=0.001), LDL-C (r=0.29, p=0.001), VLDL (r=0.48, p=0.001), S.B.P (r=0.53, p=0.001), D.B.P (r=0.35, p=0.001), and HOMA-IR (r=0.28, p=0.01). However, there was a negative correlation observed between serum Insulin levels and High Density Lipoprotein-Cholesterol (HDL-C) levels among females (r=-0.18, p=0.03). These findings underscore the significant associations between serum Insulin levels and various anthropometric and biochemical parameters, with some gender-specific variations.

Table 4: Pearson correlation analysis between serum Insulin and anthropometric, biochemical variables of the study subjects.

Parameter	Males (N=69)	Females (N=131)

	r value	p value	r value	p value
BMI	0.75	0.001	0.76	0.001
WHR	0.44	0.001	0.43	0.001
NC	0.43	0.001	0.63	0.001
Glucose	0.31	0.009	0.48	0.001
TC	0.38	0.001	0.36	0.001
TG	0.40	0.001	0.48	0.001
HDL-C	-0.22	-0.06	-0.18	- 0.03
LDL-C	0.40	0.001	0.29	0.001
VLDL	0.39	0.001	0.48	0.001
S.B.P	0.43	0.001	0.53	0.001
D.B.P	0.30	0.01	0.35	0.001
HOMA-IR	0.25	0.01	0.28	0.01

BMI – Body Mass Index, WHR – Waist to Hip Ratio, NC – Neck Circumference
TC – Total Cholesterol, TG – Triglycerides, HDL-C – High Density Lipoprotein- Cholesterol, LDL-C – Low Density Lipoprotein- Cholesterol, VLDL – Very Low Density Lipoprotein, S.B.P – Systolic Blood Pressure, D.B.P – Diastolic Blood Pressure.

Discussion:

Between 2020 and 2040, there will likely be a significant increase in cancer cases in India, with an estimated 2.08 million cases forecast. This represents a remarkable 57.4% surge compared to previous years [1]. The increasing trend emphasizes the urgent need to understand the fundamental elements that are causing this rise. Recent studies have shed light on the complex relationship between environmental factors, lifestyle choices, and the risk of cancer [9]. Eating choices undoubtedly play a crucial role in determining one's health, with meals high in fats and carbs linked to the disruption of cellular metabolism. This disruption increases the likelihood of developing obesity, insulin resistance, and metabolic syndrome [10]. Our study examines the intricate connection between obesity, insulin dynamics, and the likelihood of developing cancer. We specifically concentrate on patients classified according to their weight status and metabolic health. The results of our study showed a significant increase in serum insulin levels and Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) in overweight and obese patients, regardless of whether they had metabolic syndrome or not. This is consistent with emerging evidence that connects insulin resistance and high insulin production to increased vulnerability to cancer [11–13].

Our study revealed the precise mechanisms that connect insulin signaling pathways to cancer cell growth and multiplication. Our findings confirm the presence of insulin receptors on the surfaces of tumour cells. Insulin engagement triggers a signaling cascade that involves the activation of the IRS/PI-3K/PKB pathway and subsequently stimulates mTORC. This cascade promotes the proliferation of cancer cells through many methods, such as the manufacture of ribosomes, regulation of the cell cycle, stimulation of angiogenesis, and repression of autophagy [10, 11]. In addition, our study emphasised the complex connection between anthropometric characteristics and insulin dynamics, revealing positive associations between insulin levels and measurements such as body mass index (BMI) and neck circumference (NC). Adipose tissue plays a critical role in promoting tumor formation and progression by releasing proteins and hormones that support tumor cell proliferation [13].

All weight and metabolic syndrome categories showed increased fasting glucose levels, with a clear association between serum insulin and fasting glucose. The results show that high blood sugar has an indirect effect on cancer cells. This is because it raises insulin and IGF-1 levels and releases cytokines that cause inflammation [12–15]. Several researchers have identified an imbalanced lipid metabolism as a significant factor in the advancement of cancer. Overweight and obese patients exhibit heightened levels of total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and very low-density lipoprotein (VLDL). In contrast, there was a negative correlation between insulin levels and high-

density lipoprotein cholesterol (HDL-C) levels [13]. Our study further emphasised the intricate relationship between obesity, hypertension, and insulin resistance, showing notable increases in systolic blood pressure (SBP) and diastolic blood pressure (DBP) in obese people with metabolic syndrome. The results highlight the various complex ways in which fat contributes to hypertension, such as the stimulation of the sympathetic nervous system and insulin resistance [10-13]. Lifestyle interventions become critical in reducing the risk of cancer and improving health outcomes. Physical activity, including aerobic and resistance training, shows potential for reducing central obesity and visceral fat, which in turn improves insulin sensitivity and reduces cancer-promoting pathways [13]. Nevertheless, the limitations of our study, such as the absence of thorough evaluations of physical activity and nutritional habits, emphasize the necessity for full assessments of lifestyle in future research endeavors.

Conclusion:

Our study emphasizes the complex relationship between obesity, insulin function, and the likelihood of developing cancer. It highlights the crucial influence of dietary choices and metabolic health on cancer outcomes. Increased levels of insulin in the blood, disrupted processing of fats, and measurements related to the size and shape of the body have been identified as important factors in the advancement of cancer. This highlights the importance of making changes to one's lifestyle in order to reduce the chance of developing cancer. Our findings emphasise the significance of comprehensive methods that include dietary changes, physical exercise, and metabolic management in reducing the increasing cancer burden in India and other places.

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