Section: Physiology

Original Research Article

CORRELATION BETWEEN INFLAMMATORY MARKERS IN OBESE TYPE 2 DIABETIC SUBJECTS

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ABSTRACT

Background: A tremendous burden is imposed on an individual with diabetes mellitus as well as on a health care system due to secondary pathophysiologic changes which occurs in multiple organ system. Secondary pathophysiologic changes are caused due to metabolic dysregulation occurring in DM. the objective is to assess the association between inflammatory markers in obese diabetic and healthy adults. **Materials and Methods:** The present hospital based analytical Cross-sectional study was conducted in OPD of Rajiv Gandhi Centre for Diabetes and Endocrinology and Department of Physiology, JNMCH, A.M.U., Aligarh on patients of Type 2 Diabetes Mellitus(T2DM) who were obese as well, having BMI more than 30 Kg/m² .The period of study was from 2018 to 2020. **Result:** Levels of inflammatory markers increase in diabetic obese as compared to healthy controls. Subclinical inflammation is present in diabetics and low grade chronic inflammation is present in obesity. Therefore, presence of diabetes combined with obesity showed increase levels of inflammatory markers. **Conclusion:** Current study shows how diabetes and obesity is associated with inflammatory markers.

INTRODUCTION

Obesity in India has increased dramatically and has become number 1 lifestyle disease. Obesity is not only life threatening but also the core cause of many associated deadly diseases such as type 2 diabetes mellitus, cardiac ailments, cancers (breast cancer, colon cancer and even pancreatic cancer) and sleep apnoea.[1]

India is undergoing a rapid epidemiological transition with increased urbanization and socio-economic development which has resulted in a dramatic change in lifestyle, consisting of physical inactivity, diet rich in fat, sugar and salt coupled with a high level of mental stress. This has led to increased incidence of lifestyle diseases like hypertension, type 2 Diabetes Mellitus, dyslipidemia, obesity and ischemic heart diseases. Obesity also contributes to this disease.[2] Asian Indians have lower BMIs, and for any given BMI, Asian Indians have higher central obesity and abdominal fat than do Europeans.[3,4]

In Indian subjects, an association between diabetes and obesity was found in a number of studies. Even with a small increase in body weight and Body Mass Index, glucose tolerance is adversely affected. Obesity prevalence is on the rise in school children also, which may be responsible for the increasing prevalence of type 2 diabetes in young adults. Diabetes showed a positive and independent association with age, BMI, WHR, family history of diabetes, monthly income and sedentary physical activity. Diabcare Asia Study indicates that type 2 diabetes begins almost a decade earlier in Asians, especially South Asians and Indians. The observations also showed that obesity is an important association of diabetes in urban Indians and a BMI of more than 25 was found in 39% of the patients. Also, Indians have a higher incidence of upper body obesity despite a lean body mass index and there is a strong correlation between central obesity and type 2 diabetes. Asian Indian phenotype is characterized by a high percentage of body fat and increased WHR which predisposes to diabetes and metabolic syndrome.[5]

It has been found that there is a significant association of elevated levels of IL-6 and CRP with type-2 diabetes risk. It has been further supported that chronic inflammation is a predictor of type-2 diabetes development.[6]

Inflammatory markers like TLC, ESR and CRP are significantly elevated in patients with diabetes, suggesting the important role of inflammation, prompting need for interventions reducing inflammation in effective diabetes management. WHR is significantly associated with elevated

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inflammatory markers suggesting the role of abdominal obesity in diabetes.[7]

Tumor necrosis factor (TNF-alpha) and C-reactive protein (CRP) are positively associated with the risk of chronic kidney disease in patients with type 2 diabetes.[8]

In addition, several cross-sectional studies have shown an increase of CRP levels in patients with diabetes,[9] and the increase of CRP, IL-6, and TNF-alpha in subjects with IGT.

Inflammatory Markers includes C-reactive protein (CRP), Erythrocyte Sedimentation Rate (ESR), and several other acute phase proteins. In this study C-Reactive Protein, ESR and IL-6 were analyzed.[10]

Inflammatory Cytokines: Pro-inflammatory cytokines plays an important role in development of inflammation in obesity. There are higher circulating concentrations of inflammatory cytokines in obese individuals as compared to lean individuals. It is believed that they play an important role in causing insulin resistance. Adipose tissue is the main source of pro-inflammatory cytokines in obesity. They are mainly produced by infiltrating macrophages although adipocytes play an important role. Blood concentrations of these cytokines are lowered when weight loss occurs.[11,12]

Markers of Inflammation: Several chronic diseases involve an inflammatory response which is characterized by the increase of cytokines and serum concentrations of acute-phase reactants (markers of active inflammation) such as C-reactive protein (CRP), fibrinogen, serum amyloid A, complement, sialic acid, haptoglobin and low albumin concentrations.[12] Acute-phase reactants are synthesized in the liver and its production is regulated by cytokines, including TNF-alpha and Interleukin-6.[13,14]

MATERIALS AND METHODS

The present hospital based analytical Cross-sectional study was conducted in OPD of Rajiv Gandhi Centre for Diabetes and Endocrinology and Department of Physiology, JNMCH, A.M.U., Aligarh on patients of Type 2 Diabetes Mellitus(T2DM) who were obese as well, having BMI more than 30 Kg/m². The period of study was from 2018 to 2020. Total 60 subjects were taken. Out of which 30 were selected as cases for further study, who met the inclusion and exclusion criteria and gave the valid consent after explaining the procedure to the subject prior to entering for further investigations. After approval from the ethical committee of JNMCH, A.M.U., Aligarh, 30 healthy controls were taken who went through same investigations as of the cases.

Selected cases of T2DM with obesity having age between 29 to 69 years were assessed for inflammatory markers.

Inclusion Criteria

- Subjects (male/female) with a diagnosis of diabetes and obesity, patients who have given informed consent.
- Type 2 DM patients aged 29 – 69 years.

Criteria for diagnosing diabetes mellitus:

- Symptoms of diabetes plus Random blood glucose concentration >200 mg/dl
- Fasting plasma glucose > 126 mg/dl or
- HbA1C > 6.5% [105].

Exclusion Criteria:

The following patients will be excluded from the study:

- Patients aged < 29 years and > 69 years.
- Previous history of systemic condition related to diabetes (Trauma, alcoholic neuropathy, renal failure).
- Patients taking calcium, Vitamin D/ Mineral supplements (including herbal drugs) within past 4 weeks.
- Post menopausal women on hormone replacement therapy.
- Malignancies or history of chemotherapy or radiotherapy within past 1 year.
- Patients on any inflammatory drugs.
- Patient with any other autoimmune & rheumatic disease or cancer.
- Patients with recent venous thromboembolism episode.
- Patients already known for:
  - Inflammatory bowel disease
  - Malabsorption Syndrome
  - Gastric bypass surgery
  - Hyerphosphatemia
  - AIDS Patients
  - Patients taking Corticosteroids within past 4 weeks

Procedure: Proper history was taken in T2DM patients and then BMI was calculated after measuring weight and height of the patients. Only those patients were selected whose BMI > 30 Kg/m². Clinical assessment was done after taking valid consent. Estimation of Inflammatory markers was done in cases of Obese Diabetic Patients (study group) and healthy controls. Blood samples were collected under all aseptic precautions. Blood for fasting and post prandial glucose estimation was collected on the same day. Investigation for HbA1C was also done. Venous blood samples were taken. Serum was separated from blood and stored at -20 degree Celsius and then it was analyzed for IL-6 by ELISA method. Determination of CRP in Serum was done by means of particle enhanced turbidimetric immunoassay. ESR was done using EaSeR pipetes by Westergren method.

Inflammatory Markers

- C-Reactive protein
- ESR
- IL-6

Normal Values

- CRP: negative (- ve) [108]

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- ESR: ≤15 mm/hour for males and ≤20 mm/hour for females. [109]
- IL – 6 : Normal values 0 to 16.4 pg/ml [110]

Statistical Analysis

All the data was compiled on Microsoft Office Excel 2015.Analysis was performed using SPSS version 20.0 statistical package for windows (SPSS,Chicago,IL).Continuous variables were expressed as mean + Standard Deviation (S.D.) and qualitative data was expressed in percentages. **Unpaired —t tests** for independent samples were used for comparison of means between two groups. The association between continuous variables was tested by linear correlation using Pearson's coefficient (r). All tests were two tailed and were calculated and a p-value of <0.05 was considered statistically significant. To study the association of qualitative variables Chi-square test/Fischer-Exact test were used.

RESULTS

The mean age of subjects in cases were 51.93 years and in controls were 38.73 years. There were 6 male subjects and 24 female subjects out of total 30 cases. There were 24 male subjects and 6 female subjects out of total 30 controls.

Table 1: Distribution of age in case and control

Group	N	Mean	Std. Deviation
Case	30	51.93 years	8.43
Control	30	38.73 years	8.01

(Unpaired t- test was applied for mean age, p- value < 0.001)

Mean of age in case were 51.93 ± 8.43 years and in control were 38.73 ± 8.01 years. Unpaired t-test was applied and p-value <0.001.

Table 2: Distribution of BMI in case and control

	Group	N	Mean	Std. Deviation
BMI (kg/m2)	Case	30	35.03	4.41
	Control	30	22.38	2.25

(Unpaired t- test was applied for BMI, p -value < 0.001)

Mean of BMI in case was 35.03 ± 4.41 kg/m2 and in control was 22.38 ± 2.25 kg/m2 .Unpaired t-test was applied and p-value <0.001.

Table 3: Gender wise distribution of fasting blood glucose levels

	Sex	N	Mean	Std. Deviation
Fasting Blood Sugar(mg/dl)	Female	24	175.46	57.00
	Male	6	224.50	105.29

(Unpaired t- test for Fasting Blood Sugar (Amongst cases only), p- value <0.126)

Mean value of fasting blood sugar level in female cases was found to be 175.46 ± 57.00 mg/dl and in male cases was found to be 224.50 ± 105 mg/dl. Unpaired t-test was applied and p-value<0.126.

Table 4: Gender wise distribution of HbA1C levels

	Sex	N	Mean	Std. Deviation
HbA1C(%)	Female	24	9.63	1.86
	Male	6	9.83	2.32

(Unpaired t- test for HbA1C (Amongst cases only), p- value <0.817)

Mean value of HbA1C in female cases was found to be 9.63 ± 1.86% and in male cases was 9.83± 2.32% and p-value<0.817.

Table 5: Glycemic parameters in cases

	N	Mean	Std. Deviation
Fasting Blood Sugar(mg/dl)	30	185.27	69.90
HbA1C (%)	30	9.67	1.92

Mean value of fasting blood sugar was 185.27 ± 69.90 mg/dl and HbA1C was 9.67 ± 1.92% amongst cases.

Table 6: Status of CRP in case and control

		CRP (+/-)		Total
		Positive	Negative	
Group	Case	24	6	30
	Control	0	30	30
Total		24	36	60

(Chi -square test for CRP, p- value <0.001)

Out of 30 cases, 24 subjects were found to be CRP positive and 6 were CRP negative. Then, in 30 controls, 0 subjects was CRP positive and 30 were CRP negative .Chi-square test was applied and Fischer's Exact test was used and p-value came out to be <0.001, which is statistically significant.

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Table 7: Distribution of ESR levels in case and control

	Group	N	Mean	Std. Deviation
ESR (mm/hr)	Case	30	36.17	9.99
	Control	30	5.00	3.17

(Unpaired t- test for ESR, p- value <0.001)

Mean level of ESR amongst cases was found to be 36.17 ± 9.99mm/hr and amongst control was found to be 5.00 ± 3.17 mm/hr.
Unpaired t-test was applied and p-value <0.001 is statistically significant.

Table 8: Distribution of IL-6 levels in case and control

	Group	N	Mean	Std. Deviation
IL-6 (pg/ml)	Case	30	34.40	9.98
	Control	30	6.50	1.45

(Unpaired t- test for IL-6, p- value<0.001)

Mean level of serum IL-6 amongst cases was found to be 34.40 ± 9.98 pg/ml and amongst control was found to be 6.50 ± 1.45 pg/ml. Unpaired t-test was applied and p-value <0.001 is statistically significant.

DISCUSSION

Obesity and type – 2 diabetes mellitus are associated with low grade inflammation. Chronic inflammation plays a specific role in people with type 2 DM and coexisting obesity. The sources of inflammatory cytokines that modulate inflammatory reactions in these patients are both immune cells which are activated by hyperglycemia and associated metabolic disorders, and adipocytes.
Obesity and Type-2 diabetes is threatening the health and wellbeing in every country of the world. The prevalence of both the diabetes and obesity has assumed epidemic proportions all over the world. The obesity epidemic is likely to increase the prevalence of type 2 diabetes mellitus in the near future than the present scenario. India has the largest number of diabetics in the world and obesity is also on the rise. Central obesity predisposes Indians to a higher risk of type 2 diabetes and metabolic syndrome even at lower levels of BMI.
Most importantly BMI was found to be high, in Patel et al. it was 27.07, in Sharma et al. 26.67, in Balkees 29.3 and in Pandya et al. it was 26.6. Virtually in all studies, diabetics are found to be obese.[15]
Subclinical inflammation and presence of almost all indicators of systemic inflammation are found in type 2 diabetic patients. The rise in the inflammatory cytokines is the essential step in glucotoxicity and lipotoxicity induced mitochondrial injury, oxidative stress and beta cell apoptosis in type 2 DM. In a recent study on Indian population mean values of serum levels of inflammatory markers were found to be very high in obese diabetic patients.[16]
Assessment and evaluation of the pro inflammatory biomarkers have been postulated to play an important role in early detection of the type 2 diabetes. This helps to halt not only the progression of the diabetes but also that of the associated co-morbidities. In the present study the BMI of the cases was significantly higher in cases as compared to the controls.
Results of earlier studies have indicated that the serum levels of inflammatory markers like IL-6 and CRP are higher in obese individuals. The results may be attributed to secretion of adipokines from the adipose tissue.[17]
Higher levels of ESR and IL-6 were found in T2DM patients as compared to healthy controls which indicate the role of inflammation in the pathogenesis of the disease.[18]
All the three inflammatory markers ESR, CRP and IL – 6 levels were raised in the cases as compared to the controls.
ESR: In this study, Mean value of ESR levels in the cases were found to be (36.17 + 9.99 mm/hr) which was higher in value as compared to the mean value of the controls (5.00 + 3.17 mm/hr) The finding is similar to the findings of Vidya Baleguli et. al. and Ashish Sharma et. al.[19,20]
CRP: In this study, 24 cases were CRP positive and 6 were CRP negative out of total 30 cases. Whereas all 30 controls were CRP negative. The finding is similar to the findings of other studies [6, 19, 8, 9, 21,22]. shows that the Inflammatory markers like TLC, ESR and CRP are significantly elevated in patients with diabetes, suggesting the important role of inflammation, prompting need for interventions reducing inflammation in effective diabetes management. WHR is significantly associated with elevated inflammatory markers suggesting the role of abdominal obesity in diabetes. Ashish Sharma et. al. study showed that CRP and ESR positively correlated with obesity. It suggests that the adipose tissue is a site for synthesis of many mediators of inflammation, including interleukin 6 (IL-6). This leads to an increase in acute phase reactants in obese individuals.
Increase in the levels of the pro-inflammatory cytokines lead to increase production of acute phase reactants including C Reactive Proteins from the liver which in turn lead to progression of diabetes.[23]

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IL-6: In this study, Mean value of IL – 6 levels was found to be increased in the cases (34.40 + 9.98 pg/ml) as compared to that of the controls which has mean level of (6.50 + 1.45 pg/ml) The findings in the present study were similar to the findings by other studies.[6,9,20] showed that it is possible that inflammation directly causes elevations in blood glucose and/or insulin resistance and subsequent diabetes. In the past decade, the role of inflammatory markers in the development of type-2 diabetes has been increasingly recognized. However, the biological mechanisms through which the levels of IL-6 and CRP increase the risk of type 2 diabetes are not well understood. Potential environmental triggers such as infection, chemicals, and over nutrition can be detected through a series of sentinel cells, which are a major component of innate immunity. On and in sentinel cells, a variety of germ line–encoded pattern recognition receptors recognize and bind potentially harmful substances, which activates signaling pathways and releases pro-inflammatory cytokines mainly including IL-6. IL-6 may contribute to the pathology and physiology of type 2 diabetes through its interaction with insulin- signaling pathways and b-cell function. In addition, IL-6 stimulates the production of CRP. In the current study, our findings showed that IL-6 was more strongly associated with type 2 diabetes than CRP. Ashish Sharma et. al. study showed that the adipose tissue is a site for synthesis of many mediators of inflammation, including interleukin 6 (IL-6). This leads to an increase in acute phase reactants in obese individuals.

There appears a strong correlation between blood glucose levels and IL-6. Studies have shown that hyperglycemia stimulates IL-6 production. On the other hand higher levels of IL-6 are indicative of the inflammatory processes in diabetes [124].

The relationship between hyperglycemia, obesity and IL-6 have has been reported in earlier studies as well.[16]

CONCLUSION

Levels of inflammatory markers increase in diabetic obese as compared to healthy controls. Subclinical inflammation is present in diabetics and low grade chronic inflammation is present in obesity. Therefore, presence of diabetes combined with obesity showed increase levels of inflammatory markers.

There is scope for further studies to be done on analyzing levels inflammatory markers in obese diabetics. Few markers show decrease level in some studies and normal to increased levels in another studies. Therefore, more work can be done on the same.

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