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## RESEARCH ARTICLE

## Serum levels of Chemerin and Omentin 1 in Obese Type 2 Diabetic Patients.

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**Abstract**

**Background:-** Peripheral insulin resistance is present in obese patients primarily due to the marked down regulation of the insulin receptors. Obesity is associated with elevated levels of chemerin that might influence dysregulation of glucose metabolism. Omentin-1 (Intelectin-1) is a newly identified protein that is highly and selectively expressed in visceral adipose tissue. Controversy has arisen concerning the regulatory mechanisms that modulate chemerin and omentin-1 expression and function, namely obesity, chronic inflammation, insulin resistance, or oxidative stress.

**Objectives:-** The aim of this work is to estimate the serum levels of chemerin and omentin 1 in obese patients with type 2 diabetes mellitus and their correlation to disease progress and activity.

**Subjects and methods:-** This prospective study was conducted on 80 individuals where 60 subjects were complaining of type 2 diabetes mellitus and 20 apparently healthy subjects serving as the control group. Individuals were subjected to full history taking, thorough clinical examination, anthropometric measurements, routine and specific laboratory investigations including glycated Hb, fasting insulin level, HOMA-IR, chemerin and omentin-1 serum levels.

**Results:-** We found that there were statistically high significant differences between the studied groups regarding anthropometric measurements, DM duration, fasting blood sugar, glycated hemoglobin, lipid profile, liver function tests and chemerin and omentin, while there was significant differences regarding insulin and HOMA. We found statistically high significant positive correlations between chemerin and fasting blood sugar, glycated hemoglobin, lipid profile, liver enzymes, HOMA-IR and body mass index. There were statistically high significant negative correlations between omentin and fasting blood sugar, glycated hemoglobin, lipid profile, liver enzymes, HOMA-IR and body mass index, significant positive correlations regarding duration and insulin.

**Conclusion:-** Chemerin levels were significantly increased in obese patients and were related to insulin resistance. Omentin-1 levels were decreased in morbid obesity (MO), were inversely associated with chronic inflammation and dyslipidemia and the main modulating factors seemed to be dyslipidemia, hyperglycemia and BMI.

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**Introduction:-**

Obesity is one of the most important concerns of the 21st century, and it is considered to be a major public health issue worldwide. A cluster of diseases such as type 2 diabetes mellitus, dyslipidemia, hypertension, coronary heart disease, obstructive sleep apnea, nonalcoholic fatty liver disease, gastroesophageal reflux disease and several malignancies are associated with obesity and threat to reduce life expectancy<sup>(1)</sup>.

Adipose tissue is considered to be an active endocrine organ that produces a different number of adipokines which are dysregulated in corpulence and affect a condition of incessant aggravation, oxidative anxiety and insulin resistance, so that may progress metabolic complications (2).

Chemerin, is a member of the adipocytokines family, is otherwise called tazarotene-incited quality 2 (TIG2) or retinoic corrosive receptor responder protein 2 (RARRES2). It was initially found as a chemotactic peptide coordinating macrophages and dendritic cells toward destinations of aggravation, being included in both versatile and inborn invulnerability<sup>(3)</sup>.

Enthusiasm for chemerin has developed since it was found in fat tissue as a novel adipokine discharged by fat tissue. Chemerin has been connected with autocrine/paracrine motioning for adipocyte separation and development and additionally with glucose uptake and lipolysis incitement in adipocytes(4).

Human chemerin is a protein discharged in a latent structure as prochemerin and actuated by provocative and coagulation serine proteases. Henceforth, nearby and systemic levels of bioactive chemerin rely on proteolytic preparing and are not just identified with chemerin protein focuses<sup>(5)</sup>.

Serum circulating form, identified by **Meder and Co.** is composed with 134 aminoacids, truncated in N- and C-terminus compared with precursor. Its levels in humans are related to body mass index (BMI), concentration of triglycerides and total cholesterol, levels of blood pressure and insulin resistance (IR). However, chemerin levels higher in hepatic vein blood samples than in systemic circulation indicate that it is synthesized and secreted by the liver<sup>(6)</sup>.

In humans, chemerin mRNA was found highly expressed not only in white adipose tissue, but in liver and lungs as well. Chemerin exerts its functions by binding the G protein- coupled receptor, chemokine receptor-like 1 (CMKLR1) (also known as chemerin receptor 23 [ChemR23])<sup>(7)</sup>.

Its expression has been defined in various leukocyte populations and a number of other cell types including preadipocytes and adipocytes, osteoclasts, chondrocytes, skeletal muscle cells and endothelial cells<sup>(8)</sup>.

**Roman et al.**<sup>(9)</sup> demonstrated that obesity is associated with elevated levels of chemerin that might influence dysregulation of glucose metabolism. Then again, corpulent patients with sort 2 diabetes regularly have hyperinsulinemia, which appears to expand serum chemerin levels. The ramifications of chemerin in digestion system and aggravation may give a connection amongst corpulence and its related issue, for example, sort 2 diabetes and cardiovascular maladies.

Omentin-1 (Intelectin-1) is a newly identified protein that is highly and selectively expressed in visceral adipose tissue. It may act as an endocrine factor affecting muscles, liver and omental adipose depot to enhance insulin sensitivity and glucose metabolism<sup>(10)</sup>.

Interestingly, circulating omentin-1 levels have been negatively correlated with obesity and insulin resistance. Omentin-1 plasma levels and the adipose tissue gene expression are decreased in obesity and there is a positive relationship with the plasma adiponectin and high thickness lipoprotein and a reverse connection with waist circuit, BMI, and insulin resistance(11).

Debate has emerged concerning the administrative instruments that balance chemerin and omentin-1 expression and capacity, namely obesity, chronic inflammation or insulin resistance<sup>(12)</sup>.

Therefore the present study was designed to estimate the serum levels of chemerin and omentin 1 in obese patients with type 2 diabetes mellitus and their correlation to disease progress and activity.

## Subjects and methods:-

This prospective study was conducted on 80 individuals, where 60 subjects were complaining of type 2 diabetes mellitus and 20 apparently healthy subjects serving as control group during the period from June 2015 till February 2016. The cases were chosen from patients attending the internal medicine department and outpatient clinic of Benha University Hospital. This study was approved by Ethical committee of Benha University.

### Subjects:-

The studied subjects were classified into four groups as follows:

**Group I:-** This group included 20 adult patients (10 males & 10 females ) complaining of type 2 diabetes mellitus with normal weight (BMI < 25 kg/m<sup>2</sup>) and their age between 40-65 years.

**Group II:-** This group included 20 adult patients (12 males & 8 females ) complaining of type 2 diabetes mellitus with overweight (BMI 25 -30 kg/m<sup>2</sup> ) and their age between 43-60 years.

**Group III:-** This group included 20 patients (11 males & 9 females) complaining of type 2 diabetes mellitus with obesity (BMI > 30 kg/m<sup>2</sup>) and their age between 39-56 years.

### Inclusion criteria:

All overweight and obese individuals with type 2 diabetes mellitus and agreed to participate in the study, receiving anti-diabetic therapy.

### Exclusion criteria:

1. Type 1 diabetes mellitus
2. Psychiatric disorders
3. Endocrine diseases other than D.M
4. Acute infectious or inflammatory conditions
5. Cardiac, hepatic or renal diseases
6. Cancers and systemic diseases.
7. Females taking hormonal therapy.

**Group IV:-** This group included 20 apparently healthy adults (12 males & 8 females) and their age between 41-56 years, they were non obese, non diabetic , not complaining of any disease and not receiving any medication or dietary supplements. This group served as control.

### Methods:-

Individuals of the studied 4 groups were subjected to the following:

1. Full history taking
2. Thorough clinical examination

Anthropometric measures including weight, height, BMI (Body Mass Index), waist circumference in centimeters.

### The laboratory investigations included:

#### Sampling:-

Nine milliliters of venous blood were withdrawn under aseptic precautions after fasting for 10 -12 hours and distributed as follows:

a- 2 milliliters whole blood was put in EDETA vacutainer (violet cap) and mixed up & down gently which was used to measure CBC & HBA1C.

b- 2 ml on Na Fluoride serum test tubes, centrifuged at (1500 rpm for 10 minutes). The separated serum is used for the assay of fasting blood sugar.

c- 1.5 plain test tubes without anticoagulant. The plain test tubes were left till coagulation. After coagulation, samples were centrifuged (at 1500 rpm for 15 minutes). The separated serum was used for the assay of the lipid profile.

d-The rest of blood volume was put in plain tube (red cap) and left to clot then centrifuged (at 2000 rpm for 10 mins). The separated serum was divided into two aliquots:

1. One was designated for the immediate assay of liver function tests, kidney function test, insulin.
2. The second aliquot was stored at -20°C for subsequent assay of chemerin and omentin-1 levels.

**Routine Laboratory Investigations:-**

1. **CBC** was done for all samples using a fully automated cell counter , **Mythic 18 (Orphee) from Switzerland.**
2. **Liver Function Tests:** Alanine Transaminase, Aspartate Transaminase applying kinetic method.
3. **Fasting blood sugar** applying glucose enzymatic colorimetric method  
All biochemical tests were done using Biosystem A15 auto-analyzer
4. **Lipid Profile:** (Total Cholesterol, High-density Lipoprotein Cholesterol, and Triglycerides) Biosystem A15 auto-analyzer applying colorimetric method. Low-density Lipoprotein Cholesterol was calculated according to "Friedwald's equation":  
 $LDL-C = Total\ cholesterol - (HDL-C + TG/5)$

**Specific Laboratory Investigations:-**

1. **Glycated HB:-** was done by **Stanbio Glycohemoglobin procedure, Cat. No. 0350, Lot. No. 26121 .**  
Principle of this method:- (Quantitative Colorimetric ) Glycohemoglobin is formed progressively and irreversibly in the erythrocyte during its 120 day life. The red cell glycohemoglobin concentration is dependent on the average blood glucose concentration over a period of weeks and is stable for the life of the cell.
2. **Fasting insulin level:-** using **CALBIOTECH –catalog No. IS130D** kit, for determination of human Insulin in human serum or plasma using the ELISA technique.
3. **HOMA- IR** (Homeostasis Model Assessment of Insulin Resistance) it was calculated using the equation  
 $HOMA-IR = \text{fasting glucose (mg/dl)} \times \text{fasting insulin (iu/ml)} / 405$   
Cutoff point to define insulin resistance is  $\geq 3.8$  .
4. **Chemerin serum levels:** Chemerin was measured using human ELISA (sandwich technique) kits provided by SUN RED, China. (catalog No. 201-12-1436) with assay range (40-10000 ng/ml).

**Principle of the test:-**

the kit uses a double-antibody sandwich enzyme-linked immunosubant assay (ELISA) to assay the level of human chemerin in samples. Add chemerin to monoclonal antibody enzyme , which is pre-coated with human chemerin monoclonal antibody. Incubate: then, add chemerin antibodies labeled with, and combined with streptavidin-HRP to form immune complex then carry out incubation and washing again to remove the uncombined enzyme. Then add chromogen solution A & B. The color of the liquid changes into the blue and at the effect of acid the color finally becomes yellow. The chroma of color and the concentration of the human substance chemerin of sample were positively correlated.

**Omentin serum levels:-** Omentin was measured using human ELISA (sandwich technique) kits provided by SUN RED, China. (catalog No. 201-12-1436) with assay range (6-1500 ng/ml).

**Principle of the test:-** The kit uses a double –antibody sandwich enzyme –linked immunosorbent assay (ELISA) to assay the level of human omentin in samples. Add omentin to monoclonal antibody enzyme well which is pre-coated with human omentin monoclonal anti body , incubation then, add omentin antibodies labeled with biotin , and combined with streptavidin –HRP to form immune complex ; then carry out incubation and washing again to remove the uncombined enzyme. Then add chromogen solution A, B, the color of the liquid changes into the blue, And at the effect of acid, the color finally becomes yellow. The chroma of color and the concentration of the human substance omentin of sample were positively correlated .

**Statistics:-**

Data were analyzed using Statistical Program for Social Science (SPSS) version 18.0. Quantitative data were expressed as mean  $\pm$  standard deviation (SD). Qualitative data were expressed as frequency and percentage.

- ❖ Independent-samples t-test of significance was used when comparing between two means.
- ❖ Chi-square ( $X^2$ ) test of significance was used in order to compare proportions between two qualitative parameters.
- ❖ Receiver operating characteristic (ROC curve) analysis was used to find out the over all predictivity of parameter in and to find out the best cut-off value with detection of sensitivity and specificity at this cut-off value.
- ❖ Sensitivity: Probability that a test result will be positive when the disease is present and Specificity: Probability that a test result will be negative when the disease is not present
- ❖ PPV (positive predictive value): probability that the disease is present when the test is positive and NPV (negative predictive value): probability that the disease is not present when the test is negative

❖ Probability (P-value)

### Results:-

A total of 60 subjects with types 2 diabetes mellitus, their ages range from 39-65 years (33 males and 27 females) and 20 normal subjects as control group, their ages range from 41 to 56 years {12 males (60%) and 8 females (40%)}. So, most of the studied cases were mainly among males. There were no significant differences between the studied groups according to demographic data (**table 1**). We found that there were statistically high significant differences between the studied groups regarding anthropometric measurements.

We found that there were statistically high significant differences between the studied groups regarding duration, fasting blood sugar and glycated hemoglobin ( $p < 0.001$ ) and significant differences regarding insulin and HOMA ( $p < 0.05$ ) (**table 2**).

We found that there was statistically a high significant difference between the studied groups regarding lipid profile and liver enzymes ( $p < 0.001$ ) (**table 3**).

There was a high significant difference between control group and case groups regarding serum chemerin and omentin-1 (**table 4**). There was statistically high significant negative correlation between chemerin and omentin ( $p < 0.001$ ).

There were statistically high significant positive correlations between chemerin and fasting blood sugar, glycated hemoglobin, total cholesterol, triglycerides, low-density lipoproteins, alanine aminotransferase, aspartate aminotransferase, weight and body mass index ( $p < 0.001$ ), significant negative correlations regarding duration of DM, Insulin and high-density lipoprotein ( $p < 0.05$ ) and non-significant correlations regarding height and age ( $p > 0.05$ ) (**table 5**).

There were statistically high significant negative correlations between omentin and fasting blood sugar, glycated hemoglobin, total cholesterol, triglycerides, high-density lipoprotein, low-density lipoprotein, alanine aminotransferase, aspartate aminotransferase, weight and body mass index ( $p < 0.001$ ), significant correlations regarding duration and insulin ( $p < 0.05$ ) and non-significant correlations regarding height and age ( $p > 0.05$ ) (**table 6**).

We found that at cutoff 1000 ng/ml chemerin give 78.3% sensitivity, 95% specificity, 97.9 PPV, 59.4 NPV with AUC .898 (**fig: 1**). While omentin-1 at cutoff 250 ng/ml give 90% sensitivity, 100% specificity, 100 PPV, 76.9 NPV with AUC.967 (**fig: 2**).

**Table 1:-** Comparison between the studied groups according to demographic data & anthropometric measurements

|                               | I            | II         | III          | IV           | F                    | P        |
|-------------------------------|--------------|------------|--------------|--------------|----------------------|----------|
| <b>Age ( years )</b>          |              |            |              |              |                      |          |
| X ± SD                        | 53.8 ± 7.8   | 51.1 ± 6   | 50.9 ± 6     | 49.9 ± 5.4   | 1.3                  | 0.2      |
| Range                         | 40-65        | 43 -60     | 49-64        | 41 - 56      |                      |          |
| <b>Gender</b>                 | <b>N %</b>   | <b>N %</b> | <b>N %</b>   | <b>N %</b>   | <b>X<sup>2</sup></b> | <b>p</b> |
| Male                          | 10 (50 %)    | 12 (60 %)  | 11 (55 %)    | 12 (60 %)    | 0.56                 | 0.9      |
| Female                        | 10 (50 %)    | 8 (40 %)   | 9 (45 %)     | 8 (40 %)     |                      |          |
| <b>Wt ( kg )</b>              |              |            |              |              |                      |          |
| X ± SD                        | *71.8 ± 7.2  | 77.1 ± 7.8 | 90.1 ± 11.2  | *72 .1 ± 5.9 | 21.2                 | < 0.001  |
| Range                         | 58 – 84      | 56 – 88    | 75 – 117     | 64 – 82      |                      | (HS)     |
| <b>HT (m)</b>                 |              |            |              |              |                      |          |
| X ± SD                        | *1.75 ± 0.07 | 1.66 ± 0.1 | 1.56 ± 0.11  | *1.75 ± 0.07 | 8.2                  | < 0.001  |
| Range                         | 1.62 – 1.85  | 1.39-1.83  | 1.49 – 1.96  | 1.62 – 184   |                      | (HS)     |
| <b>BM (KG/ M<sup>2</sup>)</b> |              |            |              |              |                      |          |
| X ± SD                        | *23.3 ± 1.0  | 27 ± 1.4   | 33.1 ± 2.5   | *23.3 ± 1.1  | 166.6                | < 0.001  |
| Range                         | 20.5 – 24.6  | 25.3 -30   | 30.1 – 39 .8 | 20.5 – 24.6  |                      | (HS)     |

\* P < 0.05 When compared with other group

+ P < 0.05 When compared with other group

**Table 2:** Comparison between the studied groups according to assessment of diabetic laboratory investigations

|                             | I          | II          | III           | IV           | F     | p       |
|-----------------------------|------------|-------------|---------------|--------------|-------|---------|
| <b>Duration of DM (yrs)</b> |            |             |               |              |       |         |
| X ± SD                      | 19.3 ± 7.1 | 20.7 ± 5.7  | *13.3 ± 4.1   | -            | 9.2   | < 0.001 |
| Range                       | 10 – 33    | 12 – 32     | 7 - 20        |              |       | (HS)    |
| <b>FBS (mg/dl)</b>          |            |             |               |              |       |         |
| X ± SD                      | 175.2 ± 21 | 198.7 ± 17  | +248.7 ± 30.9 | *85.8 ± 12.1 | 154.0 | < 0.001 |
| Range                       | 140 – 209  | 171 – 226   | 193 – 302     | 69 – 104     |       | (HS)    |
| <b>HbA1c (%)</b>            |            |             |               |              |       |         |
| X ± SD                      | +7.1 ± 4.5 | 8.1 ± 0.5   | 8.87 ± 0.7    | *5.3 ± 0.8   | 123   | < 0.001 |
| Range                       | 6.6 – 7.8  | 7.3 -8.9    | 7.8 – 10.4    | 4.1 – 6.7    |       | (HS)    |
| <b>Insulin (IU/ML)</b>      |            |             |               |              |       |         |
| X ± SD                      | 7.4 ± 4.5  | 6.6 ± 3.4   | *5.4 ± 2.4    | 10.0 ± 6.6   | 3.6   | < 0.05  |
| Range                       | 2.13 -15.1 | 2.04 - 14.1 | 1.93 – 9.46   | 2.81 – 21.19 |       | (S)     |
| <b>HOMA-IR</b>              |            |             |               |              |       |         |
| X ± SD                      | 3.0 ± 1.7  | 3.0 ± 1.3   | 3.2 ± 1.3     | *1.98 ± 1.2  | 3.04  | < 0.05  |
| Range                       | 0.73 -5.72 | 1.13 – 5.45 | 1.43 – 5.34   | 0.71 – 3.66  |       | (S)     |

\* P &lt; 0.05 When compared with other group

+ P &lt; 0.05 When compared with other group

**Table 3:-** Comparison between the studied groups according to lipid profile & liver enzymes

|                  | I            | II          | III         | IV           | F     | p       |
|------------------|--------------|-------------|-------------|--------------|-------|---------|
| <b>TG mg/dl</b>  |              |             |             |              |       |         |
| X ± SD           | 112.3 ± 27.9 | 152.9 ± 93  | 293.6 ± 103 | 100.2 ± 28.6 | 45.3  | < 0.001 |
| Range            | 75 – 160     | 102 – 216   | 163 – 425   | 59 – 146     |       | (HS)    |
| <b>TC mg/dl</b>  |              |             |             |              |       |         |
| X ± SD           | 220 ± 15.7   | 240 ± 17.5  | 272.9 ± 20  | 148.2 ± 18   | 170.0 | < 0.001 |
| Range            | 198 – 243    | 212 – 263   | 242 -304    | 121 -190     |       | (HS)    |
| <b>HDL mg/dl</b> |              |             |             |              |       |         |
| X ± SD           | 51.4 ± 10.9  | 45.7 ± 7.3  | 50.4 ± 8    | 40.6 ± 6.3   | 6.87  | < 0.001 |
| Range            | 37 – 73      | 32 -56      | 37 – 67     | 33 – 56      |       | (HS)    |
| <b>LDL mg/dl</b> |              |             |             |              |       |         |
| X ± SD           | 149 ± 13.6   | 174 .4 ± 13 | 196.7 ± 15  | 92.7 ± 15.1  | 198.9 | < 0.001 |
| Range            | 128 – 173    | 147 -193    | 170 – 220   | 71 -136      |       | (HS)    |
| <b>ALT (U/L)</b> |              |             |             |              |       |         |
| X ± SD           | 28.2 ± 6.9   | 44.4 ± 8.2  | 67.7 ± 5.1  | 23.6 ± 5.4   | 186.1 | < 0.001 |
| Range            | 19 – 39      | 31 – 57     | 58 – 47     | 16 – 32      |       | (HS)    |
| <b>AST (U/L)</b> |              |             |             |              |       |         |
| X ± SD           | 22.3 ± 5.1   | 37.6 ± 4.1  | 40.6 ± 4.8  | 19.4 ± 5.7   | 91.46 | < 0.001 |
| Range            | 15 – 31      | 30 – 43     | 34 – 48     | 12 -28       |       | (HS)    |

**TG:** Triglycerides, **TC:** total cholesterol, **HDL:** high density lipoprotein, **LDL:** low density lipoprotein, **ALT:** alanine aminotransferase, **AST:** aspartate aminotransferase

**Table 4:-** Comparison between the studied groups according to chemerin & omentin

|            | chemerin X ± SD (range)       | Omentin X ± SD (range)        |
|------------|-------------------------------|-------------------------------|
| <b>I</b>   | 1035.2 ± 138 (800 – 1246)     | 232.7 ± 13.3 (209.7 – 252.7 ) |
| <b>II</b>  | 1319.8 ± 145.6 (1028 – 1600 ) | 200.6 ± 28 ( 164.5 – 305.9 )  |
| <b>III</b> | 1730.2 ± 273.4 (1368 -2244)   | 189.4 ± 14.6 (176.9 – 245.2 ) |
| <b>IV</b>  | 677.9 ± 172.1 (412 -100 )     | 304.6 ± 24.8 (286 -340.3)     |
| <b>F</b>   | 109 .6                        | 120.0                         |
|            | p < 0.001 (HS)                | p < 0.001 (HS)                |

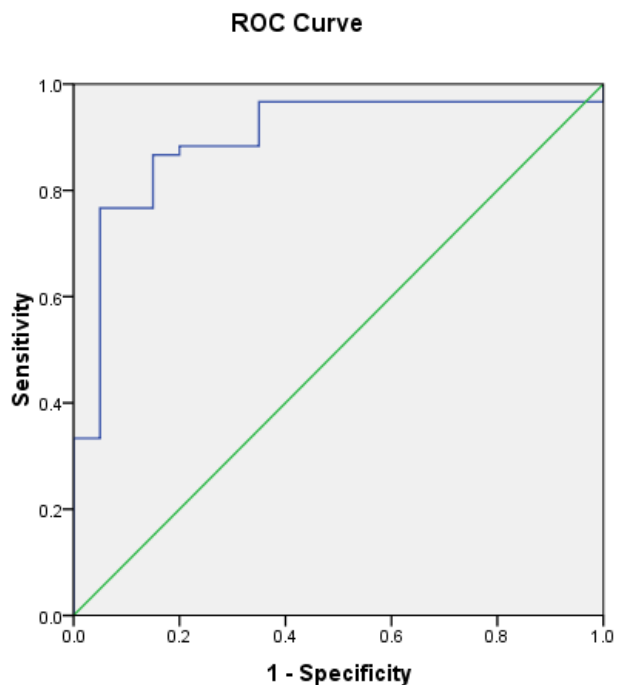
**F:** fisher exact test. **HS:** highly significant

**Table 5:** Correlation between chemerin and other parameters.

|                 | <b>R</b> | <b>P</b>     | <b>sig</b> |
|-----------------|----------|--------------|------------|
| <b>FBS</b>      | 0.83     | < 0.001      | (HS)       |
| <b>Duration</b> | -0.28    | < 0.05       | (S)        |
| <b>HBA1C</b>    | 0.8      | < 0.001 (HS) | (HS)       |
| <b>Insulin</b>  | -0.3     | < 0.001      | (HS)       |
| <b>T.C.</b>     | 0.83     | < 0.001      | (HS)       |
| <b>T.G.</b>     | 0.68     | < 0.001      | (HS)       |
| <b>HDL-c</b>    | 0.27     | < 0.05       | (S)        |
| <b>LDL-c</b>    | 0.84     | < 0.001      | (HS)       |
| <b>ALT</b>      | 0.82     | < 0.001      | (HS)       |
| <b>AST</b>      | 0.78     | < 0.001      | (HS)       |
| <b>Weight</b>   | 0.53     | < 0.001      | (HS)       |
| <b>Height</b>   | 0.1      | > 0.05       | (NS)       |
| <b>BMI</b>      | 0.79     | < 0.001      | (HS)       |
| <b>AGE</b>      | 0.02     | > 0.05       | (NS)       |
| <b>HOMA-IR</b>  | 0.27     | <0.05        | (S)        |

**Table 6:-** Correlation between omentin and other parameters.

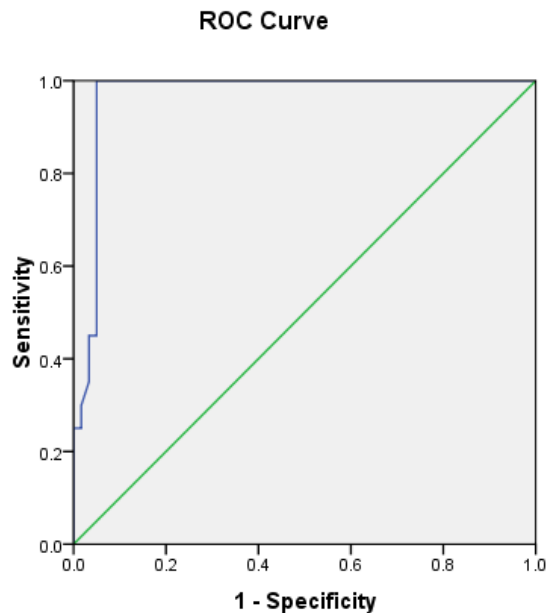
|                 | <b>R</b> | <b>P</b> | <b>Sig</b> |
|-----------------|----------|----------|------------|
| <b>FBS</b>      | -0.82    | < 0.001  | (HS)       |
| <b>Duration</b> | +0.18    | > 0.05   | (NS)       |
| <b>HBA1C</b>    | -0.78    | < 0.001  | (HS)       |
| <b>Insulin</b>  | 0.28     | < 0.05   | (S)        |
| <b>T.C.</b>     | -0.84    | < 0.001  | (HS)       |
| <b>T.G.</b>     | -0.53    | < 0.001  | (HS)       |
| <b>HDL-c</b>    | -0.34    | < 0.001  | (HS)       |
| <b>LDL-c</b>    | -0.85    | < 0.001  | (HS)       |
| <b>ALT</b>      | -0.68    | < 0.001  | (HS)       |
| <b>AST</b>      | -0.69    | < 0.001  | (HS)       |
| <b>Weight</b>   | -0.45    | < 0.001  | (HS)       |
| <b>Height</b>   | 0.13     | > 0.05   | (NS)       |
| <b>BMI</b>      | -0.66    | < 0.001  | (HS)       |
| <b>AGE</b>      | 0.03     | > 0.05   | (NS)       |
| <b>HOMA-IR</b>  | -0.28    | <0.05    | (S)        |



**Fig 1:-** diagnostic accuracy of chemerin at cutoff 1000 ng/ml

| Area Under the Curve                  |                         |                              |                                    |             |
|---------------------------------------|-------------------------|------------------------------|------------------------------------|-------------|
| Test Result Variable(s):chemerin      |                         |                              |                                    |             |
| Area                                  | Std. Error <sup>a</sup> | Asymptotic Sig. <sup>b</sup> | Asymptotic 95% Confidence Interval |             |
|                                       |                         |                              | Lower Bound                        | Upper Bound |
| .898                                  | .041                    | .000                         | .817                               | .978        |
| a. Under the nonparametric assumption |                         |                              |                                    |             |
| b. Null hypothesis: true area = 0.5   |                         |                              |                                    |             |





Diagonal segments are produced by ties.

**Fig 2:-** diagnostic accuracy of omentin at cutoff 250 ng/ml

| <b>Area Under the Curve</b>  |                         |                              |                                    |             |
|--|-------------------------|------------------------------|------------------------------------|-------------|
| Test Result Variable(s): omentin   |                         |                              |                                    |             |
| Area   | Std. Error <sup>a</sup> | Asymptotic Sig. <sup>b</sup> | Asymptotic 95% Confidence Interval |             |
|  |                         |                              | Lower Bound                        | Upper Bound |
| .967   | .019                    | .000                         | .929                               | 1.005       |
| The test result variable(s): omentin has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased. |                         |                              |                                    |             |
| a. Under the nonparametric assumption  |                         |                              |                                    |             |
| b. Null hypothesis: true area = 0.5  |                         |                              |                                    |             |

### Discussion:-

Peripheral insulin resistance is present in obese patients primarily due to the marked down regulation of the insulin receptors. Obesity is associated with elevated levels of chemerin that might influence dysregulation of glucose metabolism. Omentin-1 (Intelectin-1) is a newly identified protein that is highly and selectively expressed in visceral adipose tissue. Controversy has arisen concerning the regulatory mechanisms that modulate chemerin and omentin-1 expression and function, namely obesity, chronic inflammation, insulin resistance, or oxidative stress. The aim of this work is to estimate the serum levels of chemerin and omentin 1 in obese patients with type 2 diabetes mellitus and their correlation to disease progress and activity.

This study recruited 60 type 2 diabetic patients. They were subjected to careful history taking, thorough clinical examination, anthropometric measurements (including weight, height, body mass index and waist circumference), routine laboratory investigations (including complete blood count, liver enzymes, fasting blood sugar and lipid profile) and specific laboratory investigations (including glycated hemoglobin, fasting insulin level, HOMA-IR and serum chemerin and omentin-1 levels).

It was found that there were statistically high significant differences between the studied groups regarding anthropometric measurements, duration, fasting blood sugar, glycated hemoglobin, lipid profile, liver function tests and significant differences regarding insulin and HOMA-IR.

In this study, there were high significant differences between control group and case groups regarding chemerin and omentin. Also, there was statistically a high significant positive correlation between chemerin and omentin, as shown by Stejskal et al.<sup>(13)</sup>; Bozaoglu et al.<sup>(14)</sup>; Sell et al.<sup>(15)</sup>; Ernst and Sinal<sup>(16)</sup>; Sell et al.<sup>(17)</sup>.

This study found statistically high significant positive correlations between chemerin and fasting blood sugar, glycosylated hemoglobin, insulin, total cholesterol, triglycerides, low-density lipoproteins, alanine aminotransferase, aspartate aminotransferase, weight and body mass index, significant positive correlations regarding duration and high-density lipoprotein and non-significant correlations regarding height and age. This was in accordance with **Wang et al.**<sup>(18)</sup> and in contrast with **Jialal et al.**<sup>(19)</sup>, and this could be due to the fact that BMI is not considered to be the best tool for adipose tissue evaluation.

**Bozaoglu et al.**<sup>(20)</sup>; **Ernst and Sinal**<sup>(21)</sup>; **Hu and Fing**<sup>(22)</sup>; **Feng et al.**<sup>(23)</sup>; **Shin et al.**<sup>(24)</sup>; **Yoo et al.**<sup>(25)</sup>; **Sledzinski et al.**<sup>(26)</sup>; **Bobbert et al.**<sup>(27)</sup> and **Du et al.**<sup>(28)</sup> detected a strong association between chemerin and obesity. These findings indicated that chemerin may play a role in obesity and the development of obesity comorbidities.

**Kondaveeti et al.**<sup>(29)</sup> noticed that there was a direct correlation between the duration of diabetes and the development of microalbuminuria, because of a prolonged exposure to hyperglycaemia as well as deposition of advanced glycosylated end products.

**Sledzinski et al.**<sup>(26)</sup> reported chemerin to be associated with the cluster of features of metabolic syndrome such as high fasting glucose, triglycerides, low HDL cholesterol, elevated blood pressure and insulin resistance in obese, overweight but also in lean subjects.

On the other hand, **Jialal et al.**<sup>(19)</sup> noted and explained the lack of associations between chemerin levels and metabolic syndrome phenotypes by the dysregulation of metabolic processes associated with the development of type 2 diabetes which might have a disruptive effect.

**Rourke et al.**<sup>(30)</sup> expressed that chemerin is a proinflammatory marker delivered by the adipocytes and preadipocytes as it goes about as a chemoattractant for leukocytes to destinations of aggravation. In this manner, it may be conceivable that it assumes a part in constant aggravation that happens in corpulence.

It was exhibited that both insulin resistance and perpetual aggravation were BMI-autonomous indicators of raised serum chemerin focuses and conversely with **Sledzinski et al.**<sup>(26)</sup> who inferred that BMI was the primary indicator of serum chemerin fixation.

As per this study, chemerin is more identified with glucose digestion system and insulin resistance than abundance of fat tissue. In this admiration, **Shan et al.**<sup>(31)</sup> uncovered expanded level of chemerin in incline, overweight, and large patients with sort 2 diabetes autonomously of BMI while **Neuparth et al.**<sup>(32)</sup> reasoned that the expansion of serum chemerin was identified with the measure of aggregate rate of muscle to fat quotients in patients with no past history or proof of cardiovascular sickness, diabetes, hypertension or dyslipidemia.

Then again, while dissecting a gathering of corpulent sort 2 diabetic patients, **Weigert et al.**<sup>(33)</sup> underlined that elevated chemerin levels were more associated with inflammation rather than BMI, thereby suggesting that chemerin might be more like a marker of inflammation than obesity.

In this study, we found that there were statistically high negative significant correlations between omentin and fasting blood sugar, glycosylated hemoglobin, total cholesterol, triglycerides, high-density lipoprotein, low-density lipoprotein, alanine aminotransferase, aspartate aminotransferase, weight and body mass index, significant negative correlations regarding duration and insulin and non-significant correlations regarding height and age. **Fatima et al.**<sup>(34)</sup> have demonstrated reduced values of this novel adipokine in MO patients as compared to normal weight healthy subjects.

These findings go hand in hand with **Auguet et al.**<sup>(35)</sup> and in disagreement with **Moreno-Navarette et al.**<sup>(36)</sup> who studied two groups of obese men and women. Decreased levels of omentin-1 might explain the reduced insulin mediated glucose uptake in insulin sensitive tissues and might contribute to development of type 2 diabetes in obese patients. **Auguet et al.**<sup>(35)</sup> demonstrated in a group of MO women that plasma omentin levels inversely correlated with glucose metabolism parameters.

**Moreno-Navarette et al.**<sup>(36)</sup> showed that BMI and sex were the two independent contributors to circulating omentin variance after adjusting by fasting insulin. When studied according to sex, BMI was the only independent contributor to circulating omentin-1 variance after adjusting by fasting insulin.

### Conclusion:-

Chemerin levels were significantly increased in obese patients and were related to insulin resistance. Chemerin changes seemed to be predicted mainly by insulin resistance. Interestingly, we found relation between chemerin and anthropometric parameters, but this merits further investigation. Omentin-1 levels were decreased in obese, were inversely associated with chronic inflammation and dyslipidemia and the main modulating factors seemed to be dyslipidemia, hyperglycemia and BMI.

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