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Original Research Assessment of serum Apelin level in type II diabetic mellitus patients

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ABSTRACT

Background: Central/abdominal obesity, systemic hypertension, insulin resistance (IR) or type 2 diabetes mellitus (T2DM), and atherogenic dyslipidemia are among the clinical disorders that make up metabolic syndrome (MetS). The present study was conducted to assess serum Apelin level in type II diabetic mellitus patients.

Materials & Methods:70 type II diabetes mellitus patients of both genders were selected. Fasting blood glucose (FBG), triglyceride (TG) and high-density lipoprotein cholesterol (HDL-C) and Apelin (ng/mL) level was measured among subjects with metabolic and non- metabolic syndrome.

Results: Out of 70 patients, 43 were males and 27 were females. The mean age (years) in age in patients with MetS and without MetS was 46.2 years and 45.4 years respectively. BMI (kg/m2) was 26.7 and 26.2, WC (cm) was 106.2 and 97.6, SBP (mm Hg) was 126.4 and 116.4, DBP (mm Hg) was 82.5 and 74.0, FBG (mg/dL) was 172.0 and 116.4, TG (mg/dL) was 196.4 and 130.6, HDL-C (mg/d L) was 41.5 and 46.2, Apelin level was 230.6 ng/mL and 428.4 ng/mL respectively. The difference was significant (P< 0.05).

Conclusion: The determination of serum apelin may contribute to the evaluation of theMetS occurrence in T2DM patients. **Keywords:** Diabetes mellitus, Metabolic syndrome, Obesity adjuvant of choice in patients undergoing elective open cholecystectomy.

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Introduction

Central/abdominal obesity, systemic hypertension, insulin resistance (IR) or type 2 diabetes mellitus (T2DM), and atherogenic dyslipidemia are among the clinical disorders that make up metabolic syndrome (MetS). T2DM and atherosclerotic cardiovascular disease (ASCVD) are multiplex risk factors for MetS.¹ It twice the risk of ASCVD and five times the risk of T2DM in non-diabetic people. With a prevalence of 30% to 40%, Met S is widespread throughout the world. In several other populations, the prevalence of MetS is varied. Numerous studies have demonstrated that MetS varies by gender and ethnic group.² According to reports, the prevalence of MetS varies between 8% and 24% for men and between 7% and 46.5% for women worldwide. The prevalence of MetS in European Americans and the European population is between 20% and 30% for men and women. It has demonstrated that MetS is becoming more common in Asian nations.³

The APLN gene codes for the 12-amino acid peptide that makes up apelin, which is expressed in human adipocytes.⁴ Bioactive hormones and mediators (adipokines) that control metabolic activity are

produced and secreted by adipose tissue. One of the recently identified adipokines in human adipocytes implicated in numerous obesity-related symptoms of Met S components is apelin.⁵ Animal insulin secretion and sensitivity are regulated by apelin. A number of studies, albeit not all of them, have shown that different metabolic disorders are brought on by elevated apelin levels in both people and animals. Aptin has recently emerged as a potential therapeutic target for a number of metabolic diseases, including IR, hyperinsulinemia, and diabetes mellitus.⁶The present study was conducted to assess serum Apelin level in type II diabetic mellitus patients.

Materials & Methods

The study was carried out on 70 type II diabetes mellitus patients of both genders. All gave their written consent to participate in the study.

Data such as name, age, gender etc. was recorded. The 5-mL blood samples were provided for all subjects after 12 h overnight fast. After the serum separation, it was used to determine biochemical parameters. Fasting blood glucose (FBG), triglyceride (TG) and high-density lipoprotein cholesterol (HDL-C) were

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measured by commercial kits using an automated method. Apelin (ng/m L) level was also measured among subjects with metabolic and non- metabolic syndrome. Results thus obtained were subjected to

statistical analysis. P value < 0.05 was considered significant.

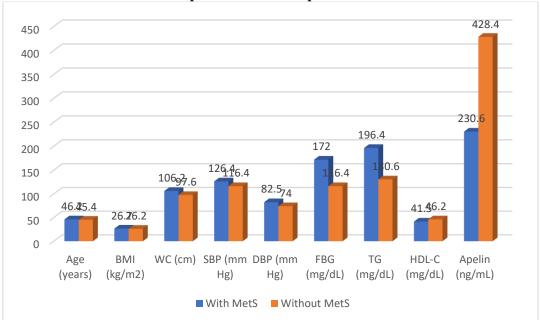
Results

 Table: I Distribution of patients					
Total- 70					
Gender	Male	Female			
Number	43	27			

Table I shows that out of 70 patients, 43 were males and 27 were females.

Parameters	Table: II Assessment of pa With Met S	Without Met S	P value
Age (years)	46.2	45.4	0.83
BMI (kg/m2)	26.7	26.2	0.91
WC (cm)	106.2	97.6	0.05
SBP (mm Hg)	126.4	116.4	0.04
DBP (mm Hg)	82.5	74.0	0.03
FBG (mg/dL)	172.0	116.4	0.01
TG (mg/dL)	196.4	130.6	0.02
HDL-C (mg/dL)	41.5	46.2	0.03
Apelin (ng/mL)	230.6	428.4	0.01

Table II, graph I shows that mean age (years) in age in patients with Met S and without Met S was 46.2 years and 45.4 years respectively. BMI (kg/m2) was 26.7 and 26.2, WC (cm) was 106.2 and 97.6, SBP (mm Hg) was 126.4 and 116.4, DBP (mm Hg) was 82.5 and 74.0, FBG (mg/dL) was 172.0 and 116.4, TG (mg/dL) was 196.4 and 130.6, HDL-C (mg/dL) was 41.5 and 46.2, Apelin level was 230.6 ng/mL and 428.4 ng/mL respectively. The difference was significant (P< 0.05).



Graph: I Assessment of parameters

Discussion

Met S may raise the chance of developing certain illnesses, such heart disease, which can be fatal.⁷ The specific pathophysiology of Met S is still unknown, however apelin has been demonstrated to affect a variety of organs and tissues, including the kidney, gut, heart, and brain.^{8,9} The discovery of apelin in

adipocytes may provide insight into apelin's endocrine function as an adipokine. Increased apelin levels in humans and animals have been linked to a number of metabolic diseases, according to some but not all research.¹⁰ With its anti-obesity and anti-diabetic properties, apelin has emerged as a useful adipokine that may be a useful therapeutic target for a number of

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metabolic issues.¹¹ The present study was conducted to assess serum Apelin level in type II diabetic mellitus patients.

We found that out of 70 patients, 43 were males and 27 were females. Zhang et al¹² assessed insulin resistance (HOMA-IR) and β-cell function. Plasma apelin-17 levels were determined bv radioimmunoassay (RIA). Plasma apelin levels were significantly lower in the diabetic group compared with the control subjects (0.11 vs. 0.25 ng/ml, P <0.0001). Apelin levels were negatively correlated with CRP (r = -0.357, P = 0.001), HOMA-IR (r = -0.509, P < 0.0001), FPG (r = -0.607, P < 0.0001), and A1C (r = -0.467, P < 0.0001) and positively correlated with HOMA-IS (r = 0.566, P < 0.0001). Multiple stepwise regression analysis showed that apelin increased 565 fg/ml along with 1 SD, which increased in HOMA-IS in our subjects (R2 = 0.107, P = 0.002). Compared with the lowest quartile, the highest quartile of HOMA-IS had significantly higher plasma apelin level (0.27 vs. 0.07 ng/ml, P < 0.0001). Such results were also obtained in FPG and A1C (P < 0.0001 for both).

We observed that mean age (years) in age in patients with MetS and without MetS was 46.2 years and 45.4 years respectively. BMI (kg/m2) was 26.7 and 26.2, WC (cm) was 106.2 and 97.6, SBP (mm Hg) was 126.4 and 116.4, DBP (mm Hg) was 82.5 and 74.0, FBG (mg/dL) was 172.0 and 116.4, TG (mg/dL) was 196.4 and 130.6, HDL-C (mg/dL) was 41.5 and 46.2, Apelin level was 230.6 ng/mL and 428.4 ng/mL respectively. Cavallo et al¹³ recruited 369 subjects, 119 with T2D, 113 with T1D and 137 non-diabetic controls. All subjects underwent a complete clinical examination, including anthropometric and laboratory measurements. Serum apelin levels were determined by EIA (immunoenzyme assay). Patients with T2D had significantly higher serum apelin levels compared to controls (1.23 \pm 1.1 ng/mL vs 0.91 \pm 0.7 ng/mL, P<0.001) and to T1D subjects (0.73 \pm 0.39 ng/mL, P<0.001). Controls and T1D subjects did not differ significantly in apelin levels. Apelin concentrations were directly associated with fasting blood glucose (FBG), body mass index (BMI), basal Disposition Index (DI-0), age, and diagnosis of T2D at bivariate correlation analysis. Multiple regression analysis confirmed that diagnosis of T2D, basal DI-0 and FBG were all determinants of serum apelin levels independently from age and BMI. Bariatric surgery performed in a subgroup of obese diabetic subjects (n = 12) resulted in a significant reduction of apelin concentrations compared to baseline levels (P = 0.01). Yu et al¹⁴ investigated whether chemerin and apelin play an important role in the pathophysiologic proceeding of diabetes. This study enrolled 81 newly diagnosed obese type 2 diabetes mellitus (T2DM) patients (T2DM group, n = 81). All the patients were randomly assigned to DM1 group treated with metformin (n = 41) and DM2 group treated with pioglitazone (n = 40). After hypoglycemic agents

treatment, patients under better blood glucose control were chosen to be given antioxidant treatment. Another 79 subjects without T2DM were recruited as normal control group (NC group), including 40 subjects (NC1 group) with normal body mass index (BMI) and 39 obese subjects (NC2 group). Levels of chemerin, apelin, BMI, tumor necrosis factor-α (TNFα), homeostasis model assessment of IR (HOMA-IR) and 8-isoprotaglandim F2a (8-iso-PGF2a) were examined at baseline and post-treatment. The relationship between chemerin, apelin and BMI, TNF- α , HOMA-IR, 8-iso-PGF2 α was analyzed. The baseline levels of chemerin, apelin, TNF-α, HOMA-IR and 8-iso-PGF2a in T2DM group were significantly higher than normal control group (P <0.001). All indices mentioned above were significantly decreased after treatment (P < 0.05). In T2DM patients treated with pioglitazone, indices mentioned above except for HOMA-IR, were decreased significantly compared with patients treated with metformin (P < 0.05). After antioxidant treatment using lipoic acid, levels of chemerin, apelin, TNF- α and 8-iso-PGF2 α were further significantly decreased (P < 0.05). Correlation analysis showed that the levels of chemerin and apelin correlated positively with BMI, TNF- α , HOMA-IR and 8-iso-PGF2 α before and after treatment with hypoglycemic agents (P < 0.01). The levels of chemerin and apelin also had positive correlation with TNF-a and 8-iso-PGF2a after antioxidant treatment (P < 0.05).

The shortcoming of the study is small sample size.

Conclusion

Authors found that the determination of serum apelin may contribute to the evaluation of the MetS occurrence in T2DM patients.

References

- 1. Fonseca VA. Rationale for the use of insulin sensitizers to prevent cardiovascular events in type 2 diabetes mellitus. Am J Med 2007; 120 (9 Suppl 2): S18-S25.
- 2. Kadoglou NP, Tsanikidis H, Kapelouzou A, Vrabas I, Vitta I, Karayannacos PE, et al. Effects of rosiglitazone and metformin treatment on apelin, visfatin, and ghrelin levels in patients with type 2 diabetes mellitus. Metabolism 2010; 59: 373-379.
- Weisberg SP, McCann D, Desai M, Rosenbaum M, Leibel RL, Ferrante AW Jr. Obesity is associated with macrophage accumulation in adipose issue. J Clin Invest 2003; 112: 1796-1808.
- 4. Roh SG, Song SH, Choi KC, Katoh K, Wittamer V, Parmentier M, et al. Chemerin-a new adipokine that modulates adipogenesis via its own receptor. Biochem Biophys Res Commun 2007; 362: 1013-1018.
- 5. Ernst MC, Sinal CJ. Chemerin: at the crossroads of inflammation and obesity. Trends Endocrinol Metab 2010; 21: 660-667.
- Soriguer F, Garrido-Sanchez L, Garcia-Serrano S, Garcia-Almeida JM, Garcia-Arnes J, Tinahones FJ, Garcia-Fuentes E. Apelin levels are increased in morbidly obese subjects with type 2 diabetes mellitus. Obesity surgery. 2009 Nov;19:1574-80.

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- Kahn SE, Hull RL, Utzschneider KM (2006) Mechanisms linking obesity to insulin resistance and type 2 diabetes. Nature 444: 840–846.
- 8. Deng Y, Scherer PE (2010) Adipokines as novel biomarkers and regulators of the metabolic syndrome. Ann N Y Acad Sci 1212: 1–19.
- 9. Tatemoto K, Takayama K, Zou MX, Kumaki I, Zhang W, et al. (2001) The novel peptide apelin lowers blood pressure via a nitric oxide-dependent mechanism. Regul Pept 99: 87–92.
- Boucher J, Masri B, Daviaud D, Gesta S, Guigné C, et al. (2005) Apelin, a newly identified adipokine upregulated by insulin and obesity. Endocrinology 146: 1764–1771.
- 11. Medhurst AD, Jennings CA, Robbins MJ, Davis RP, Ellis C, et al... (2003) Pharmacological and immunohistochemical characterization of APJ receptor and its endogenous ligand apelin. J Neurochem 84;1162–1172.
- Zhang Y, Shen C, Li X, Ren G, Fan X, Ren F, Zhang N, et al. Low plasma apelin in newly diagnosed type 2 diabetes in Chinese people. Diabetes Care. 2009;32(12):150.
- Cavallo MG, Sentinelli F, Barchetta I, Costantino C, Incani M, Perra L, Capoccia D, Romeo S, Cossu E, Leonetti F, Agati L. Altered glucose homeostasis is associated with increased serum apelin levels in type 2 diabetes mellitus. PloS one. 2012 Dec 5;7(12):51236.
- 14. Yu S, Zhang Y, Li MZ, Xu H, Wang Q, Song J, Lin P, et al. Chemerin and apelin are positively correlated with inflammation in obese type 2 diabetic patients. Chin Med J (Engl). 2012;125(19):3440-3444.
- Nainani P, Singh HP, Paliwal A, Nagpal N. A rare case report of clear cell variant of oral squamous cell carcinoma. J Clin Diagn Res. 2014 Dec;8(12):QD07-9. doi: 10.7860/JCDR/2014/11536.5339.
- Singh HP, Yadav M, Nayar A, Verma C, Aggarwal P, Bains SK. Ameloblastomatous calcifying ghost cell odontogenic cyst - a rare variant of a rare entity. Ann Stomatol (Roma). 2013 Mar 20;4(1):156-60. doi: 10.11138/ads.0156.
- Singh HP, Kumar P, Goel R, Kumar A. Sex hormones in head and neck cancer: Current knowledge and perspectives. Clin Cancer Investig J. 2012;1(1):2-5. <u>https://doi.org/10.4103/2278-0513.95011</u>.
- Sharma A, Singh HP, Gupta AA, Garg P, Moon NJ, Chavan R. Granulocytic sarcoma in non-leukaemic child involving maxillary sinus with long term follow up: A rare case report. Ann Maxillofac Surg 2014;4:90-5.
- 19. Puri N, Rathore A, Dharmdeep G, Vairagare S, Prasad BR, Priyadarshini R, et al. A clinical study on comparative evaluation of the effectiveness of carbamazepine and combination of carbamazepine with baclofen or capsaicin in the management of TrigeminalNeuralgia. Niger J Surg 2018;24:95-9.
- Singh HP, Yadav M, Nayar A, Verma C, Aggarwal P, Bains SK. Ameloblastomatous calcifying ghost cell odontogenic cyst - a rare variant of a rare entity. Annali di Stomatologia 2013; IV (1): 156-160