#1979 - THE RELATIONSHIP OF APELINE AND ENDOCAN LEVELS WITH KIDNEY FUNCTION LOSS AND CARDIOVASCULAR DISEASE PARAMETERS IN TYPE 2 DIABETES PATIENTS

Authors and Affiliations	Esra GUVERCIN BALBAY Medeniyet University Goztepe Educational and Research Hospital, Department of Internal Medicine Gulsah SASAK KUZGUN Medeniyet University Goztepe Educational and Research Hospital, Department of Nephrology Ali Rıza ODABAS Medeniyet University Goztepe Educational and Research Hospital, Department of Nephrology
Corresponding Author and email	Gulsah SASAK KUZGUN, gulsahsasak@gmail.com

Body

Aim: Diabetes mellitus (DM) is an increasing public health problem in the world. The most common causes of death in this group of patients are cardiovascular disease (CVD) and renal failure due to diabetic nephropathy (DN). Apelin is adipocytokine, neuropeptide and also a cardiovascular peptide.

Apelin has been shown to play a role in endothelial oxidative system and coronary atherosclerotic plaque formation, and have a protective role in CVD (1). Plasma apelin levels increased in Type 1 DM patients and have shown to reduce DN progression (2). Endocan that is a proteoglycan, has been identified as a marker for systemic inflammation and microalbuminuria levels were positively correlated with endocan increase in Type 2 DM patients (3,4).

We aimed to determine the relationship between apelin and endocan levels with parameters indicating renal and cardiovascular dysfunction in patients with type 2 DM.

Method: Study was conducted with 100 Type 2 DM diagnosed patients. Initial and 6^{th} month visits of patients in accordance with the criteria were evaluated. Body mass index, waist and hip circumferences were calculated. Routine blood tests and urine tests including protein, creatinine and microalbumiria were recorded. Apelin and endocan parameters were added to initial routine blood tests and electrocardiograms (ECG) were also performed.

Echocardiography (ECHO) examinations, carotid intima media thickness (CIMT) and brachial artery flow-mediated dilatation (FMD) measurements were performed. Relation of apelin and endocan levels with CIMT, brachial artery FMD measurement, parameters showing diastolic dysfunction in ECO, loss of 6-month glomerular filtration rate (GFR), proteinuria and microalbuminuria were statistically evaluated.

Results: There was no correlation between endocan levels and CIMT *(p=0.363)*, brachial artery FMD measurements *(p=0.808)*; however in a model using apelin, endocan, GFR, microalbuminuria in 24-hour urine sample and DM year in the linear regression analysis for mitral E/A ratio; endocan levels were found to be an independent risk factor for diastolic dysfunction. There was no difference between t GFR losses at the ofhe 6-month when the patients were divided into two groups according to Endocan median values *(p=0.202)*. When the patients grouped according to microalbuminuria in 24-hour urine sample; endocan levels were the highest *(284.4±142.5, p=0.046*) in the group without microalbuminuria, and the lowest in the group with microalbuminuria *(163.8±107.3, p=0.046).* There was no correlation between apelin and CIMT *(p=0.534)*, brachial artery FMD *(p=0.446)* and Mitral E/A ratio *(p=0.417)*. Significant difference was found in the 6th month of GFR *(70.6±27.7 vs 82.3±19.6, p=0.046)* in patients with two groups according to Apelin median values but there was no difference in the 6-month of GFR loss between the groups *(-6.3±13.5 vs -3.5±11.8, p=0.461*). Apelin levels were not correlated with 24-hour urine microalbuminuria *(p=0.854)*. The group with lower apelin levels had a longer duration of DM *(13±9 vs 9±6, p = 0.036)*, with higher baseline uric acid levels *(5.8±1.7 vs. 5.1±1.3, p = 0.042).*

Conclusion: Endocan level is an independent risk factor for diastolic dysfunction in Type 2 DM patients and its use in combination with other parameters in this group of patients may be useful in determining cardiovascular risk. Although there was a negative correlation between microalbuminuria

	levels and endocan levels in our study, different results were obtained in studies in the literature. Apelin molecule has not been shown to be associated with cardiovascular risk markers in diabetic patients in our study, but we think that apelin molecule may be a useful marker for follow-up of renal function in diabetic nephropathic patients when used in combination with other parameters. In patients with lower Apelin levels, uric acid levels are higher and renal function is more impaired, suggesting that apelin and uric acid are effects on the renal system through inflammatory mediators and possibly interrelated mechanisms. In the case of diabetic nephropathy, there is a need for larger scale and longer follow-up studies for the use of the apelin Endocan molecule for the prediction of nephropathy progression and CVD.
References	1- Ashley E, Chun HJ, Quertermous T. Opposing cardiovascular roles for the angiotensin and apelin signaling pathways. J Mol Cell Cardiol. 2006;41(5):778-81. 2- Day RT, Cavaglieri RC, Feliers D. Apelin retards the progression of diabetic nephropathy. Am J Physiol Renal Physiol. 2013;304(6):F788-800. 3- Lee W, Ku SK, Kim SW, Bae JS. Endocan elicits severe vascular inflammatory responses in vitro and in vivo. Journal of cellular physiology. 2014;229(5):620-30. 4- Arman Y, Akpinar TS, Kose M, Emet S, Yuruyen G, Akarsu M, et al. Effect of Glycemic Regulation on Endocan Levels in Patients With Diabetes: A Preliminary Study. Angiology. 2016;67(3):239-44.
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