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performed by the same physician. During the physical examinations, height (cm), weight (cm), waist and hip circumferences (cm) of the subjects were measured. BMI was calculated. Waist circumferences were measured at the plane between anterior superior iliac spines and lower costal margines at the narrowest part of the waistline while subjects were standing during slight expiration.

Subjects accepted to participate in the study were invited to the clinic in the next morning a er 12 hour fasting duration. For measuring 25 (OH)D, venous blood samples were collected into plain tubes, and serum was separated and stored at -70°C until analysis for a week. Levels of 25 (OH)D were estimated using a kit 25 (OH)D-Ria-CT (Bruxelles-Belgium). e treated samples were then assayed using a competitive binding radioimmunuassay (RIA) technique.

Statistical analysis

All statistical analyses were made by using the so ware SPSS for Windows V13.0. Normality of distribution of variables was tested by Shapiro-Wilk and Kolmogorov-Smirnov tests. Subjects were compared for di erences in anthropometric and biochemical data by two tailed Mann-Whitney U or Student's t test. Kruskal-Wallis test or Oneway ANOVA was performed for comparison of two or more independent samples. Correlation between variables were determined by Pearson correlation test or Spearman's Rho. Data are expressed as means ± SD. A p value below 0.05 (two tailed) was considered to be statistically signi cant.

Results

One hundred and two premenaposal women were recruited in the study between November 2008-April 2009. Age were similar among three groups (p=0.085). Anthropometric measurements of three groups can be seen on table 1.

Frequency of metabolic syndrome in vitamin D de ciency group was signi cantly more than that of the other groups (p=0.028; Figure 1). Number of metabolic syndrome criteria in Group 1 was more than that of Group 3 (p<0.0001; Table 2). Vitamin D level was negatively correlated with waist circumference, BMI, triglyceride and fasting plasma glucose, and positively correlated with HDL (r=-0.463, p<0.0001, r=-0.505, p<0.0001, r=-0.292, p=0.0028, r=-0.258, p=0.009; r=0.243, p=0.014, respectively; Figure 2). Among subjects without metabolic syndrome, vitamin D level in the group with more criteria was less than the other groups (p<0.0001; Table 3). Even we have taken only the subjects with waist circumference less than 88 cm, metabolic syndrome frequency in vitamin D de ciency group (n=4) was more than the other groups (p=0.001). Waist circumference was also greater in the vitamin D de cieny group (p<0.0001; Table 4).

		Group 1 (n=32)	Group 2 (n=31)	Group3 (n=39)	p
Age	year	34,47 ± 6,77	32,32 ± 6,68	31,64 ± 5,34	0,085
Systolic blood pressure	mmHg	114,44 ± 15,53	109,67 ± 12,64	116,79 ± 9,12	0,18
Diastolic blood pressure	mmHg	76,30 ± 9,26	71,67 ± 7,86	74,29 ± 4,75	0,14
Waist					

Conclusion

It was found that increased frequency of metabolic syndrome was associated with vitamin D de ciency independent of hyperparathyroidism. As a result, vitamin D de ciency may be an independent risk factor for metabolic syndrome.

Discussion

It was shown in this study that metabolic syndrome frequency was increasing with vitamin D de ciency. Moreover, vitamin D level was

negatively correlated with waist circumference, BMI, triglyceride and fasting plasma glucose and positively correlated with HDL.

Low levels of vitamin D are a ecting the cellular functions negatively in most tissues. In that manner, pancreas is one of those tissues. Vitamin D de ciency may deteroriate the e ect of insulin on adipose tissue. In a study of Reis et al., vitamin D de ciency was found to be related to abdominal obesity, metabolic syndrome, insulin resistance and type 2 diabetes [19]. Various mechanisms are responsible from this associaton. First, anormal calcium metabolism is related with weight gain [20]. Increase in intracellular calcium was shown to activate lipogenesis and to inhibit lipolysis [21]. Increased levels of intracellular calcium leads to accumulation of triglyceride in adipocytes and activation of lipogenesis and obesity. High calcium intake was investigated in the study of Zemel et al. according to this hypothesis and it was found that obesity risk is decreasing with high calcium intake in mice [20]. Other mechanisms related to that is associated with TNF- (Tumor necrosis factom mca-141(fatakerren)-106((TuIFN)--106((T700)]TJ EMC /Span <</MCID 3576>>BDC T* [

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