TÜRKİYE ENDOKRİNOLOJİ VE METABOLİZMA DERNEĞİ BÜLTENİ

Üç ayda bir yayımlanır • Üyelere ücretsiz olarak gönderilir

Sayı 57 • Ocak – Şubat – Mart - 2017

14. Mezuniyet Sonrası Hipertansiyon Eğitim Kursu, Muğla

14. Mezuniyet Sonrası Hipertansiyon Eğitim Kursu derneğimizin çatısı altında TEMD, Obezite, Lipid Metabolizması, Hipertansiyon Çalışma Grubu tarafından, 11-12 Mart 2017 tarihlerinde Muğla, Sıtkı Koçaman Üniversitesi Toplantı Salonunda yaklaşık 80 hekimimizin katılımı ile başarı ile gerçekleştirilmiştir.

Osteoporoz ve Metabolik Kemik Hastalıkları Kursu - IV

Osteoporoz ve Metabolik Kemik Hastalıkları Kursu, derneğimiz çatısı altında Osteoporoz ve Diğer Metabolik Kemik Hastalıkları Çalışma grubumuz tarafından 11 Mart 2017, Cumartesi günü Hilton İstanbul Bosphorus otelde yaklaşık 90 hekimimizin katılımı ile başarılı bir şekilde gerçekleştirilmiştir. Canlı olarak yayınlan kursumuza 50 katılımcı online olarak katılmıştır.

ENDOKRİN ACİLLER - VIII KURSU TAMAMLANDI

Endokrin Aciller VIII Kursu, 4 Mart 2017 tarihinde, Ottoman Palace Otel, Antakya'da 64 meslektaşımızın katılımı ile başarılı bir şekilde tamamlanmıştır.

ADRENAL VE GONAD HASTALIKLARI SEMPOZYUMU TAMAMLANDI

Adrenal ve Gonad Hastalıkları Kursu 25 Şubat 2017 tarihinde Erzurum'da 55 meslektaşımızın katılımı ile başarılı bir şekilde tamamlandı.

5. LİPİD METABOLİZMASI BOZUKLUKLARI EĞİTİM KURSU

5. Lipid Metabolizması Bozuklukları Eğitim Kursu derneğimiz çatısı altında Obezite, Lipid Metabolizması, Hipertansiyon Çalışma Grubu tarafından 17-18 Aralık 2016 tarihlerinde Adana'da Başkent Üniversitesi Kışla Yerleşkesi Konferans Salonu'nda yaklaşık 100 hekimimizin katılımı ile başarı ile gerçekleştirilmiştir.

Kongre, Kurslar ve Sempozyumlar

Bilimsel Kongreler, Ulusal ve Uluslararası Sempozyumlar

03-07 Mayıs 2017
39. Türkiye Endokrinoloji ve Metabolizma Hastalıkları Kongresi
Belek/Antalya
www.temd.org.tr
www.temhk2017.org

23-26 Mart 2017
WCO-IOF-ESCEO 2017
Fortezza da Basso, Florrence, Italy
http://www.wco-iof-esceo.org/

01-04 Nisan 2017
ENDO2017
Orange Country Convention Center
Orlando, FL
https://www.endocrine.org/endo-2017

15-16 Nisan 2017
6. Lipid Metabolizması ve Bozuklukları Eğitim Kursu
Trakya Üniversitesi BALKAN KONGRE MERKEZİ Konferans Salonu, Edirne

20 - 23 Mayıs 2017
19th European Congress of Endocrinology (ECE 2017)
Lisbon, Portugal
http://www.ece2017.org/

09-13 Haziran 2017
70th Scientific Sessions (ADA 2017)
San Diego, CA
http://ada-2017.org/?gclid=CJb_os-WO6tICFciRGwodWJ8AAg

11-15 Ekim 2017
19. Ulusal İç Hastalıkları Kongresi
Sueno Deluxe Hotel & Kongre Merkezi, Belek, Antalya
http://ichastaliklari2017.org/

19-22 Ekim 2017
Endo Bridge 2017
Regnum Carya Hotel, Antalya
http://www.endobridge.org/

Üyelerimizden Literatür Seçmeleri

EFFECT OF LIFESTYLE INTERVENTIONS WITH OR WITHOUT METFORMIN THERAPY ON SERUM LEVELS OF OSTEOPROTEGERIN AND RECEPTOR ACTIVATOR OF NUCLEAR FACTOR KAPPA B LIGAND IN PATIENTS WITH PREDIABETES.

Arslan MS[1], Tutal E[2], Sahin M[3], Karakose M[2], Ucan B[2], Ozturk G[4], Cakal E[2], Biyikli Gencturk Z[5], Ozbek M[2], Delibasi T[6].

Endocrine. 2017 Feb;55(2):410-415. doi: 10.1007/s12020-016-1121-4. Epub 2016 Oct 15.

Osteoprotegerin has been shown to be increased in cardiovascular disorders and type 2 diabetes mellitus. Prediabetes represents a high risk condition for diabetes and diabetic complications. Therefore, we aimed to find the relationship between prediabetes and osteoprotegerin with nuclear factor-B ligand, carotid intima media thickness, and metabolic markers. A total of 54 participants with prediabetes including impaired fasting glucose (n = 21), impaired glucose tolerance (n = 8), impaired fasting glucose and impaired glucose tolerance (n = 25), and 60 healthy individuals as a control were admitted to the study. Metabolic and anthropometric parameters, insulin resistance variables, osteoprotegerin, and nuclear factor-B ligand markers, carotid intima media thickness were examined at baseline for all participants. To evaluate the effect of therapy we determined the same parameters after the end of the study. Measurements of waist circumference, body mass index, body fat percentage and levels of fasting blood glucose, fasting insulin, homeostatic model assessment of insulin resistance, triglyceride levels and hsCRP and carotid intima media thickness were significantly higher in patients with prediabetes ($p < 0.05$). We also found higher osteoprotegerin and lower nuclear factor-B ligand levels in patients than in controls however, the value was non-significant ($p > 0.05$). Patients with prediabetes were under lifestyle interventions with (group 1, n = 33) or without metformin (group 2, n = 21) therapy. Baseline anthropometric and metabolic characteristics were not found statistically different in group 1 and group 2. Mean follow up period of the patients were 7.9 ± 2.2 month (min-max: 6-12 months). After the follow up period we evaluated the same parameters and found significant differences between waist circumference, body mass index, body fat percentage, fasting insulin, homeostatic model assessment of insulin resistance, and osteoprotegerin levels ($p < 0.05$). However, carotid intima media thickness, and nuclear factor-B ligand levels significantly different only in the group treated with metformin ($p < 0.05$). We also compared the variables after the treatment period with the control group and found significantly lower levels in terms of fasting insulin, homeostatic model assessment of insulin resistance, waist circumference, body mass index, body fat percentage, carotid intima media thickness, osteoprotegerin, and nuclear factor-B ligand values ($p < 0.05$). Correlation analysis revealed a negative relationship between nuclear factor-B ligand and body mass index, and body fat percentage in group 1 ($p = 0.05$, $r = -0.646$, $p = 0.01$, $r = -0.585$). Therapy of prediabetes was associated with a significant decrease in osteoprotegerin and certain metabolic variables together with an increase in nuclear factor-B ligand levels particularly in patients with under metformin therapy.

SERUM LEVELS OF FIBROBLAST GROWTH FACTOR-23, OSTEOPROTEGERIN, AND RECEPTOR ACTIVATOR OF NUCLEAR FACTOR KAPPA B LIGAND IN PATIENTS WITH PROLACTINOMA.

Arslan MS, Sahin M, Karakose M, Tutal E, Topaloglu O, Ucan B, Demirci T, Caliskan M, Ozdemir S, Ozbek M, Cakal E.
Endocr Pract. 2017 Mar;23(3):266-370. doi: 10.4158/EP161440.OR. Epub 2016 Nov 16.

Objective: The aim of this study to was to evaluate the effect of fibroblast growth factor-23 (FGF-23), osteoprotegerin (OPG), receptor activator nuclear κB ligand (RANKL), and vitamin D hormones on bone loss in patients with hyperprolactinemia due to pituitary prolactinoma.

Methods: We recruited 46 premenopausal female patients with prolactinoma and age and sex-matched healthy controls (Group 3, n = 20) for this cross-sectional study. Prolactinoma patients were divided into 2 groups as patients newly diagnosed (Group 1, n = 26) and those under cabergoline treatment (Group 2, n = 20). Anthropometric and metabolic variables; hormonal profiles; and osteocalcin, deoxypyridinoline (DOP), and bone mineral density measurements were performed for all participants. FGF-23, OPG, and RANKL levels were analyzed in all groups.

Results: FGF-23, OPG, calcium, phosphorus, and parathormone levels were similar between all groups despite significantly higher levels in the control group in terms of vitamin D and RANKL levels than in patients. Bone loss was found more in Group 2, particularly observed in Z scores of femur and spinal bone (P<.05). Correlation analysis revealed a negative correlation between FGF-23 and femur neck T score (r = -0.0433, P = .05) in patients with active prolactinoma. A positive correlation was also observed between parameters of DOP and OPG (r = 0.673, P = .02). In patients with remission there were a negative correlation between prolactin and luteinizing hormone (r = -600, P = .08). Additionally, a negative correlation was found between osteocalcin and osteoprotegerin in patients in remission (r = -0.73, P = .01).

Conclusion: Our data indicated that FGF-23 and OPG levels do not play a critical role on the development of bone decrease in patients with hyperprolactinemia. However, further prospective studies in larger numbers of participants should be designed to clarify this issue.

Abbreviations: BFP = body fat percentage BMD = bone mineral density BMI = body mass index CV = coefficient of variation DOP = deoxypyridinoline ELISA = enzyme-linked immunosorbent assay FGF-23 = fibroblast growth factor-23 HOMA-IR = homeostatic model assessment of insulin resistance OPG = osteoprotegerin RANKL = receptor activator nuclear κB ligand.

VISCERAL OBESITY MEDIATES THE ASSOCIATION BETWEEN METABOLIC SYNDROME AND OBSTRUCTIVE SLEEP APNEA SYNDROME.

Bozkurt NC[1], Beysel S[1], Karbek B[1], Unsal İO[1], Cakir E[1], Delibasi T[2].
Metab Syndr Relat Disord. 2016 May;14(4):217-21. doi: 10.1089/met.2015.0086. Epub 2016 Mar 22.

Background: Metabolic syndrome (MetS) and visceral obesity are more prevalent in obstructive sleep apnea syndrome (OSAS). We investigated the association of visceral fat (VF) measures with the components of MetS in OSAS patients with different severity levels, according to World Health Organization (WHO, 1999), National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III, 2001), and International Diabetes Federation (IDF, 2005) definitions.

Patients and methods: Study population was grouped according to polysomnography results as non-OSAS [who had apnea-hypopnea index (AHI) <5, n=51], mild OSAS (5 < AHI <15, n=52), moderate OSAS (15 < AHI <30, n=53), and severe OSAS (AHI >30, n=53). VF ratio was measured by abdominal bioimpedance analysis. Waist-to-hip ratio (WHR), homeostasis model assessment of insulin resistance (HOMA-IR), and lipid profiles were assessed in all subjects.

Results: The prevelance of MetS in OSAS patients was 30.0%, 35.6%, and 44.4% according to WHO, NCEP-ATP III, and IDF definitions, respectively. MetS was found in 27.5% non-OSAS and 72.8% OSAS according to at least one definition (P=0.012). Within OSAS group, 27.2% subjects had average, 38.0% had slightly excessive, and 34.8% had an excessive VF ratio. The prevelance of MetS was similar in various VF ratios (P>0.05). However HOMA-IR increased progressively with VF ratio after adjusting for age, gender, and body mass index (BMI; P=0.02). AHI increased progressively with BMI (P=0.02), WHR (P=0.03), VF ratio (P=0.01), HOMA-IR (P=0.02), and MetS (P=0.016).

Conclusion: Since severity of OSAS, in terms of AHI and insulin resistance, is both associated with VF rather than BMI, VF should be suggested to link OSAS and MetS. The IDF definition is more sensitive in OSAS patients to diagnose MetS, as central obesity and insulin resistance are obligatory components. This would allow clinicians to intervent earlier to adverse metabolic outcomes of OSAS.

DISCORDANCE BETWEEN GH AND IGF-1 LEVELS IN TURKISH ACROMEGALIC PATIENTS.

Cerit ET, Ağbaht K, Demir Ö, Şahin M, Gedik VT, Özcan C, Çorapçıoğlu D.
Endocr Pract. 2016 Dec;22(12):1422-1428. Epub 2016 Sep 15.

Objective: Discordance between insulin-like growth factor-1 (IGF-1) and growth hormone (GH) levels is an important problem in the follow-up of patients diagnosed with acromegaly. Our aims were to evaluate the discordance between IGF-1 and GH levels and compare the performance of different cut-off levels for the nadir in GH (GHn) in acromegalic patients.

Methods: The study included 63 acromegalic patients in a follow-up at a tertiary care university hospital facility. Levels of IGF-1,

IGF binding protein-3 (IGFBP-3), and GH were investigated. The baseline GH and GHn levels were evaluated after an oral glucose tolerance test (cut-offs of 0.4 and 1 ng/mL, respectively). The discordance rates between GHn and IGF-1 levels, and IGF-1/IGFBP-3 ratios were determined.

Results: We first adopted a GHn cut-off value of 1 ng/mL and found that 27 patients (42.9%) exhibited biochemical remission (BR) (IGF-1 <95th percentile, GH <1), and 25 patients (39.7%) had no BR (NBR) (IGF-1 ≥95th percentile, GH >1). Discordance in the presence of normal IGF-1 and nonsuppressed GH (DC1) occurred in 2 of 63 (3.2%) patients; discordance in the presence of high IGF-1 and suppressed GH (DC2) occurred in 9 of 63 (14.3%) patients. If the GHn cut-off value adopted was 0.4 ng/mL, the distributions were 17 of 63 (27.0%) patients in BR, 29 of 63 (46.0%) patients in NBR, 12 of 63 (19.0%) in DC1, and 5 of 63 (7.9%) patients in DC2. If only the baseline GH values were considered, the distributions were very similar to those with a GHn cut-off value of 0.4 ng/mL. The IGF-1/IGFBP-3 ratio was lowest in the BR group.

Conclusion: Adopting a GHn cut-off value of 0.4 ng/mL did not increase the test performance compared with baseline GH only. In contrast, in the follow-up of acromegalic patients, the IGF-1/IGFBP-3 ratio might be a useful measurement when discordance between IGF-1 and GH levels occurs. We propose that these values be considered in clinical practice.

Abbreviations: BR = biochemical remission DC1 = discordance group 1 DC2 = discordance group 2 DM = diabetes mellitus GH = growth hormone GHn = nadir in GH IGF-1 = insulin-like growth factor-1 IGFBP-3 = IGF binding protein-3 LAR = long-acting release NBR = not in biochemical remission OGTT = oral glucose tolerance test.

THE IMPACT OF PARATHYROIDECTOMY ON SERUM ADAMTS1, ADAMTS4 LEVELS, INSULIN RESISTANCE, AND SUBCLINICAL CARDIOVASCULAR DISEASE IN PRIMARY HYPERPARATHYROIDISM.

Karakose M[1], Caliskan M[2], Arslan MS[2], Demirci T[2], Karakose S[3], Cakal E[2].
Endocrine. 2017 Jan;55(1):283-288. doi: 10.1007/s12020-016-1175-3. Epub 2016 Nov 14.

Purpose: Primary hyperparathyroidism has been associated with increased incidence of morbidity and mortality of the cardiovascular system. The etiopathogenetic mechanisms underlying this association are still not completely clear. Accumulating evidence suggested that a disintegrin and metalloproteinase with thrombospondin-like motifs (ADAMTS) has a role in the development of inflammation and atherosclerosis. In this study, we aimed to determine whether there is a change in serum levels of ADAMTS1, ADAMTS4, carotid intima-media thickness, and cardiovascular risk score after the surgery and also whether there is a relationship between ADAMTS levels and cardiovascular risk score in hypercalcemic primary hyperparathyroidism patients.

Methods: The study included the 48 consecutive newly diagnosed patients with primary hyperparathyroidism. The patients were evaluated before and six months after parathyroidectomy. The Framingham score is used to calculate cardiovascular risk. Serum ADAMTS levels were determined by a human enzyme-linked immunoassay in all subjects.

Results: The fasting glucose, fasting insulin levels and HOMA values were decreased significantly in all patients after surgery compared to the pretreatment values ($p < 0.05$). ADAMTS1, ADAMTS4, and carotid intima-media thickness levels were significantly lower after surgical correction of primary hyperparathyroidism compared to the preoperative values ($p < 0.05$). cardiovascular risk score was decreased after parathyroidectomy however, the difference were not statistical significant ($p > 0.05$). There were statistically significant relationship between cardiovascular risk score and waist/hip ratio, calcium, LDL-cholesterol, carotid intima-media thickness, ADAMTS4 values.

Conclusion: Based on the results of the present study, fasting glucose, fasting insulin levels, ADAMTS1, ADAMTS4, and carotid intima-media thickness might be an additional parameters during the management of patients with primary hyperparathyroidism, since these factors might improve after surgery.

Keywords: ADAMTS; Atherosclerosis; Insulin resistance; Parathyroidectomy; Primary hyperparathyroidism

CCDC141 MUTATION IDENTIFIED IN ANOSMIC HYPOGONADOTROPIC HYPOGONADISM (KALLMANN SYNDROME) ALTERS GNRH NEURONAL MIGRATION.

Hutchins BI[1], Kotan LD[1], Taylor-Burds C[1], Ozkan Y[1], Cheng PJ[1], Gurbuz F[1], Tiong JD[1], Mengen E[1], Yuksel B[1], Topaloglu AK[1], Wray S[1].
Endocrinology. 2016 May;157(5):1956-66. doi: 10.1210/en.2015-1846. Epub 2016 Mar 25.

The first mutation in a gene associated with a neuronal migration disorder was identified in patients with Kallmann Syndrome, characterized by hypogonadotropic hypogonadism and anosmia. This pathophysiological association results from a defect in the development of the GnRH and the olfactory system. A recent genetic screening of Kallmann Syndrome patients revealed a novel mutation in CCDC141. Little is known about CCDC141, which encodes a coiled-coil domain containing protein. Here, we show that Ccdc141 is expressed in GnRH neurons and olfactory fibers and that knockdown of Ccdc141 reduces GnRH neuronal migration. Our findings in human patients and mouse models predict that CCDC141 takes part in embryonic migration of GnRH neurons enabling them to form a hypothalamic neuronal network to initiate pulsatile GnRH secretion and reproductive function.

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DUYURULAR

Yeni Yılınızı Kutlar, Sağlık Mutluluk ve Başarılar Dileriz.

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