

TÜRKİYE ENDOKRİNOLOJİ VE METABOLİZMA DERNEĞİ BÜLTENİ



Üç ayda bir yayımlanır • Üyelere ücretsiz olarak gönderilir

Sayı 66 • Nisan – Mayıs – Haziran - 2019

41. TÜRKİYE ENDOKRİNOLOJİ VE METABOLİZMA HASTALIKLARI KONGRESİ TAMAMLANDI

"41. Türkiye Endokrinoloji ve Metabolizma Hastalıkları Kongresi" bu yıl Regnum Carya Hotel Antalya'da 27 Nisan-01 Mayıs 2019 tarihleri arasında, 1190 meslektaşımızın katılımı ile başarıyla tamamlandı.

Bu yıl kongre bünyesinde "Temel Tiroid Ultrasonografi Kursu", "Nadir Metabolik Hastalıklar Kursu" ve "Adrenal Gonad Görüntüleme Kursu" olmak üzere 3 kurs ve kongrenin ilk günü "Beslenme ve Egzersiz Sempozyumu" gerçekleştirildi. 41. TEMHK bilimsel programında, 12 konferans, 21 panel, 5 uzmanına danış, 2 karşıt görüş, 8 sözlü bildiri oturumu, 9 uydu sempozyum yer aldı. 9'u yurt dışından davetli olmak üzere toplam 211 konuşmacı ve oturum başkanı bilimsel programda görev aldı. Bir önceki yıl ilkini gerçekleştirdiğimiz genç endokrinologların çalışmalarını sunma fırsatı buldukları "TEMMD GENÇ Paneli" bu yıl da bilimsel program içinde yerini aldı. Bu yıl "Genç Araştırmacı Ödülü"nü almaya, Dr. Sema Çiftçi Doğanşen hak kazandı. Dr. Sema Çiftçi Doğanşen açılış töreninde "Prolaktinomalarda Dopamin Agonist ilişkili Dürtü Kontrol Bozukluğu" başlıklı konferansını verdi.

41. Türkiye Endokrinoloji ve Metabolizma Kongresi'nde 48 Sözlü, 217 Poster olmak üzere toplam 265 bildiri sunuldu. Bilimsel kurul tarafından yapılan değerlendirmeler sonucunda her yıl olduğu gibi bu yıl da en iyi 3 sözlü ve 3 poster bildiriye ödül verildi. TURKJEM Makale Yarışmasının sonuçları da kongremiz sırasında açıklandı ve ödüller sahiplerini buldu.

Kongremizde emeği geçen tüm meslektaşlarımıza teşekkür eder, başarılar dileriz.



**41. Türkiye
Endokrinoloji
ve Metabolizma
Hastalıkları
Kongresi'nden kareler..**



41. Türkiye Endokrinoloji ve Metabolizma Hastalıkları Kongresi Sözlü, Poster Bildiri ve Genç Araştırmacı Ödülleri

ÖDÜL ALAN BİLDİRLER

● GENÇ ARAŞTIRMACI ÖDÜLÜ

Prolaktinomalarda Dopamin Agonist İlişkili Dürtü Kontrol Bozukluğu
Sema Çiftçi Doğanşen



● SÖZLÜ BİLDİRİ BİRİNCİLİK ÖDÜLÜ (S-44)

Akromegali Hastalarında Uzun Dönem Tedavi Sonuçları: 10 Farklı Merkezde Takip Edilen 547 Akromegali Hastasının Verilerinin Değerlendirilmesi

Çağlar Keskin, Özgür Demir, Alper Çağrı Karıcı, Dilek Berker, Zeynep Cantürk, Güzin Fidan Yaylalı, Şenay Topsakal, Reyhan Ersoy, Fahri Bayram, Melek Eda Ertörer, Emre Bozkırlı, Filiz Haydardedeoğlu, Esra Nur Ademoğlu Dilekçi, Seyid Ahmet Ay, Güven Barış Cansu, Mustafa Şahin, Rıfat Emral, Demet Çorapçıoğlu.

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⁷Başkent Üniversitesi, Adana Dr. Turgut Noyan Eğitim ve Araştırma Hastanesi, Endokrinoloji ve Metabolizma Hastalıkları Bilim Dalı, Adana

⁸Abant İzzet Baysal Üniversitesi Tıp Fakültesi, Endokrinoloji ve Metabolizma Hastalıkları Bilim Dalı, Bolu

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● SÖZLÜ BİLDİRİ İKİNCİLİK ÖDÜLÜ (S-45)

Akromegalik kardiyomyopatinin patofizyolojisine yeni bir bakış: Akromegalik hastalarda serum FSTL1 düzeyleri ve FSTL1 polimorfizmlerinin kardiyak MRG bulguları ile ilişkisi
Süleyman Nahit Şendur, Tuncay Hazirolan, Büşra Aydın, İncilay Lay, Mehmet Alikışifoğlu, Tomris Erbaş

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● SÖZLÜ BİLDİRİ ÜÇÜNCÜLÜK ÖDÜLÜ (S-32)

Mikropartikül Yüklü Biyoaktif Yara Örtüsünün Diyabetik Ayak Ülserlerinde Klinik Açısından Değerlendirilmesi

Şevki Çetinkalp, Evren Homan Gökçe, İlgin Yıldırım Şimşir, Sakine Tuncay Tanıverdi, Fatma Doğan, Çiğir Biray Avcı, İpek Eroğlu, Tülin Utku, Cumhuriyet Gündüz, Özgen Özer

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⁴Hacettepe Üniversitesi Eczacılık Fakültesi, Eczacılık Temel Bilimleri Anabilim Dalı, Ankara

● POSTER BİLDİRİ BİRİNCİLİK ÖDÜLÜ (P-154)

Büyük (≥ 8 cm) Adrenal Kitlelerde Histopatolojik Bulgularımız.

Mehmet Muhittin Yalçın, Başak Bolayır, Begüm Algül, Mustafa Akhoroz, Mehmet Feyiz Altınsoy, Alev Eroğlu Altınova, Müjde Aktürk, Ayhan Karakoç, Sinan Sözen, Aylar Poyraz, Füsün Balış Törüner.

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³Gazi Üniversitesi Tıp Fakültesi Üroloji Anabilim Dalı,

⁴Gazi Üniversitesi Tıp Fakültesi Patoloji Anabilim Dalı, Ankara

● POSTER BİLDİRİ İKİNCİLİK ÖDÜLÜ (P-063)

Tip 1 Gaucher Hastalarında Kemik Bulgularının ve Enzim Replasman Tedavisinin Kemik Mineral Yoğunluğu Üzerine Etkilerinin Değerlendirilmesi

Gökтуğ Sarıbeytiler, Sakin Tekin, Ramazan Çakmak, Bülent Canbaz, Gülşah Yenidünya Yalın, Özlem Soyluk Selçukbiricik, Nurdan Gül, Ayşe Kubat Üzümlü, Ferihan Aral, Refik Tanakol
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● POSTER BİLDİRİ ÜÇÜNCÜLÜK ÖDÜLÜ (P-135)

Subakut Tiroidit: 2015-2019 Tarihleri Arasında Tanı Alan 100 Hastanın Klinik Karakteristikleri ve Tedavi Sonuçları
Ersen Karakılıç, Emre Saygılı

¹Çanakkale 18 Mart Üniversitesi Tıp Fakültesi, Endokrinoloji ve Metabolizma Hastalıkları Bilim Dalı,

²Çanakkale Devlet Hastanesi, Endokrinoloji ve Metabolizma Hastalıkları Kliniği



TURKJEM 2. ÖDÜLLÜ MAKALE YARIŞMASI ÖDÜLLERİ

TURKJEM 2. ÖDÜLLÜ MAKALE YARIŞMASI BİRİNCİLİK ÖDÜLÜ

Relationship of Decreased Circulating Apelin Levels with Growth Hormone, Insulin-like Growth Factor, Carotid Intima-media Thickness, and Epicardial Fat Thickness in Acromegaly

Mehmet Çalan, Mustafa Demirpençe

Department of Internal Medicine, Division of Endocrinology and Metabolism, İzmir Bozyaka Training and Research Hospital, İzmir



TURKJEM 2. ÖDÜLLÜ MAKALE YARIŞMASI İKİNCİLİK ÖDÜLÜ

Relation of 18F-FDG PET/CT Positivity with Tumor Cytopathology, Galectin-3, PTEN, Ki-67 and NIS Expressions in Thyroid Nodules.

Güzin Çakmak, Berna İmge Aydoğan, Cevriye Cansız Ersöz*, Elgin Özkan**, Serpil Dizbay Sak*, Sevim Güllü.

Ankara University Faculty of Medicine, Department of Endocrinology and Metabolism, Ankara, Turkey

*Ankara University Faculty of Medicine, Department of Pathology, Ankara, Turkey

**Ankara University Faculty of Medicine, Department of Nuclear Medicine, Ankara, Turkey



TURKJEM 2. ÖDÜLLÜ MAKALE YARIŞMASI ÜÇÜNCÜLÜK ÖDÜLÜ

Nodular Thyroid Disease and Papillary Thyroid Carcinoma in Functional Pituitary Adenomas

Sema Çiftçi Doğanşen, Gülşah Yenidünya Yalın, Seher Tanrıku, Sema Yarman.

Istanbul University İstanbul Faculty of Medicine, Department of Internal Medicine, Division of Endocrinology and Metabolism, İstanbul, Turkey



DIYABETTE GÜNCELLEME VE OLGU TARTIŞMA TOPLANTISI TAMAMLANDI



TEMED Diyabet Çalışma Grubu "Diyabette Güncelleme ve Olgu Tartışma Toplantıları - 24", 6 Nisan 2019 tarihinde Gaziosmanpaşa Üniversitesi Tıp Fakültesi ev sahipliğinde Tokat'ta gerçekleştirilmiştir. Toplantıya bölgedeki endokrinoloji uzmanları, iç hastalıkları uzmanları ve aile hekimlerinden toplam 74 kişi katılmıştır.

Programdaki olgu sunumları yakın merkezler olan Sivas Cumhuriyet Üniversitesi, Çorum Hitit Üniversitesi, Amasya Üniversitesi ve Yozgat Şehir Hastanesi'nde görev yapmakta olan genç akademisyen meslektaşlarımız tarafından yapılmıştır. Her oturum, iki başkan tarafından interaktif şekilde, deneyim paylaşımları yapılarak yönetilmiştir. Bu şekildeki programın öğrenim hedeflerine ulaşmada daha verimli bir yöntem olduğu bir kez daha deneyimlenmiştir.

Emeği geçen üyelerimize teşekkür eder, başarılarının devamını dileriz.

HİPOFİZ ADENOMLARI KURSU TAMAMLANDI

TEMED Hipofiz Çalışma Grubu olarak 15-16 Haziran 2019 tarihinde Renaissance Bosphorus Otel İstanbul'da düzenlemiş olduğumuz "HİPOFİZ ADENOMLARI KURSU" başarı ile tamamlanmıştır. Toplantıya 135 meslektaşımızın katılımı gerçekleşmiştir.

Emeği geçen tüm üyelerimize teşekkür eder, saygılarımızı sunarız.



24. TİROİDOLOJİ KURSU (TİROKURS) TAMAMLANDI



TEMED, Tiroid Çalışma Grubu yıllık etkinliklerinden olan ve Türkiye çapında değişik bölgelerimizde yapılan 'Pratik Tiroidoloji' kurslarının yirmidördüncüsü TİROKURS-24, 22 Haziran 2019 tarihinde, Abant İzzet Baysal Üniversitesi Tıp Fakültesi, Endokrinoloji ve Metabolizma Hastalıkları Bilim Dalı öğretim Üyesinin değerli katkısı ve Merck® Türkiye'nin, koşulsuz desteği ile Bolu, Gazella Resort Otel'de yapılmıştır.

Aile hekimleri, İç hastalıkları, Genel Cerrahi, Nükleer Tıp uzmanları ve araştırma görevlilerinden oluşan 60 hekimin katılımı ile gerçekleştirilen kurs başarı ile tamamlanmıştır.

Emeği geçen tüm üyelerimize teşekkür eder, saygılarımızı sunarız.

Kongre, Kurslar ve Sempozyumlar



Bilimsel Kongreler, Ulusal ve Uluslararası Sempozyumlar

- 4-7 Nisan 2019
IOF-WCO-IOF –ESCEO
Paris
- 15-19 Nisan 2020
42. Türkiye Endokrinoloji ve Metabolizma Hastalıkları Kongresi
Antalya
- 24-28 Nisan 2019
28th AACE Annual Scientific & Clinical Congress
Los Angeles, CA
- 18-21 Mayıs 2019
ECE 2019 - 21st European Congress of Endocrinology
Lyon, France
- 07-11 Haziran 2019
79th Scientific ADA
Sanfiranisco, CA
- 04-09 Ağustos 2019
70th Annual ISE Meeting
South Africa
- 07-10 Eylül 2019
42nd Annual Meeting of ETA
Budapest - Hungary
- 16-20 Eylül 2019
55th EASD Congress
Barcelona, Spain
- 28 Eylül 2019
Endokrinologlar İçin İleri Tiroid ve Boyun Ultrasonografisi Kursu
Movenpick Hotel, Ankara
- 04-06 Ekim 2019
Mezuniyet Sonrası Eğitim Kursu – ENDOKURS 4 (Atatürk'ün Samsun'a çıkışının 100. yılı onuruna)
Anemon Hotel, Samsun
<http://endokurs.org/>
- 18-19 Ekim 2019
5. Metabolik Kemik Hastalıkları Sempozyumu, Prof. Dr. Miyase Bayraktar Onuruna
Hacettepe Kongre Merkezi, Ankara
- 19 Ekim 2019
İç Hastalıkları Uzmanları İçin Pratik Tiroidoloji Kursu - TİROKURS - 25
Hatay
- 24-27 Ekim 2019
EndoBridge 2019
Antalya
<http://www.endobridge.org/>
- 30 Ekim - 3 Kasım 2019
89th Annual Meeting of the ATA
Chicago, IL
- 08-09 Kasım 2019
15. Hipofiz Hastalıkları Sempozyumu ve 2. Hipofiz Görüntüleme Kursu
Sheraton Otel, Ankara
<http://www.hipofiz2019.org/>
- 15-16 Kasım 2019
1st International Meeting on Thyroid Ultrasound-guided, Minimally Invasive Therapies
Reggio Emilia, Italy
<http://www.nordestcongressi.it/site/event/1st-tnt-meeting-thyroid-nodule-therapies/?lang=en>
- 23 Kasım 2019
Metabolik Kemik Hastalıkları Kursu - OSTEOKURS
Bursa
- 2-6 Aralık 2019
IDF 2019 Congress
Busan Korea
- 7-8 Aralık 2019
TEMĐ 18. Hipertansiyon, Dislipidemi ve Obezite Eğitim Sempozyumu
Harran Üniversitesi Tıp Fakültesi Dekanlığı
Konferans Salonu, Şanlıurfa
<http://temd.org.tr/>
- 13-14 Aralık 2019
9. Adrenal Gonad ve Nöroendokrin Tümörler Sempozyumu
Divan Express Otel, Eskişehir
<http://www.adrenalgonad.org/>
- 15-19 Nisan 2020
42. Türkiye Endokrinoloji ve Metabolizma Hastalıkları Kongresi
Sueno Kongre Merkezi, Antalya
<http://www.temhk2020.org/>

Üyelerimizden Literatür Seçmeleri

UPDATE ON THERAPEUTIC OPTIONS IN LIPODYSTROPHY.

Akinci B^{1,2}, Meral R¹, Oral EA³.*Curr Diab Rep.* 2018 Oct 29;18(12):139. doi: 10.1007/s11892-018-1100-7.**PURPOSE OF REVIEW:** The purpose of this review is to summarize the therapeutic approach for lipodystrophy syndromes with conventional treatment options and metreleptin therapy in detail and to point out the current investigational treatments in development.**RECENT FINDINGS:** The observation of leptin deficiency in patients with lipodystrophy and the potential of leptin replacement to rescue metabolic abnormalities in animal models of lipodystrophy were followed by the first clinical study of leptin therapy in patients with severe lipodystrophy. This and several other long-term studies demonstrated important benefits of recombinant human leptin (metreleptin) to treat metabolic abnormalities of lipodystrophy. These studies ultimately led to the recent FDA approval of metreleptin for the treatment of generalized lipodystrophy and EMA approval for both generalized and partial lipodystrophy. Additional research efforts in progress focus on novel treatment options, predominantly for patients with partial lipodystrophy. Current treatment of generalized lipodystrophy includes metreleptin replacement as an adjunct to diet and standard treatment approach for metabolic consequences of lipodystrophy. Beyond metreleptin, a number of different compounds and treatment modalities are being studied for the treatment of partial lipodystrophy.PHENOTYPIC AND GENETIC CHARACTERISTICS OF
LIPODYSTROPHY: PATHOPHYSIOLOGY, METABOLIC
ABNORMALITIES, AND COMORBIDITIES.Akinci B^{1,2}, Meral R¹, Oral EA³.*Curr Diab Rep.* 2018 Nov 8;18(12):143. doi: 10.1007/s11892-018-1099-9.**PURPOSE OF REVIEW:** This article focuses on recent progress in understanding the genetics of lipodystrophy syndromes, the pathophysiology of severe metabolic abnormalities caused by these syndromes, and causes of severe morbidity and a possible signal of increased mortality associated with lipodystrophy. An updated classification scheme is also presented.**RECENT FINDINGS:** Lipodystrophy encompasses a group of heterogeneous rare diseases characterized by generalized or partial lack of adipose tissue and associated metabolic abnormalities including altered lipid metabolism and insulin resistance. Recent advances in the field have led to the discovery of new genes associated with lipodystrophy and have also improved our understanding of adipose biology, including differentiation, lipid droplet assembly, and metabolism. Several registries have documented the natural history of the disease and the serious comorbidities that patients with lipodystrophy face. There is also evolving evidence for increased mortality rates associated with lipodystrophy. Lipodystrophy syndromes represent a challenging cluster of diseases that lead to severe insulin resistance, a myriad of metabolic abnormalities, and serious morbidity. The understanding of these syndromes is evolving in parallel with the identification of novel disease-causing mechanisms.DOES PRIMARY HYPERPARATHYROIDISM HAVE AN
ASSOCIATION WITH THYROID PAPILLARY CANCER? A
RETROSPECTIVE COHORT STUDY.Çetin K¹, Sıkar HE², Temizkan Ş³, Ofluoğlu CB², Özderya A⁴, Aydın K⁴, Gül AE⁵, Küçük HF².*World J Surg.* 2019 May;43(5):1243-1248. doi: 10.1007/s00268-019-04920-4.**BACKGROUND:** To investigate the relationship between primary hyperparathyroidism (pHPT) and papillary thyroid cancer (PTC).**METHODS:** The perioperative findings of 275 patients with pHPT who underwent surgery between January 2014 and December 2017 were retrospectively reviewed. Thirty-one patients were diagnosed with pHPT and PTC concurrently. Pathology results and demographic findings of these patients were compared with 186 patients who underwent thyroidectomy and diagnosed with PTC at the same time interval.**RESULTS:** The co-occurrence of pHPT and PTC was 11.3% (31/275). The median ages of thepHPT, pHPT + PTC, and PTC groups were 55, 57, and 50 years old, respectively ($p < 0.001$). The diameter of tumor was smaller in the pHPT + PTC group [median 7 mm (range 0.5-25 mm) vs. 15 mm (range 1-100 mm)], with higher rates of microcarcinomas ($p < 0.001$), than the patients in the PTC group. Examination of tumor morphology showed higher rates of tumor capsule invasion and multicentricity in the pHPT + PTC group than those in the isolated PTC group ($p = 0.02$, $p = 0.04$, respectively).**CONCLUSION:** The pHPT + PTC group had significantly smaller tumor diameter than the PTC group. This result may support the idea that pHPT leads to overdiagnosis of PTC. However, observation of high rates of tumor capsule invasion and multicentricity in the pHPT + PTC group may suggest an associative etiology with more aggressive PTC.DOPAMINE AGONIST-INDUCED IMPULSE CONTROL
DISORDERS IN PATIENTS WITH PROLACTINOMA: A
CROSS-SECTIONAL MULTICENTER STUDY.Dogansen SC^{1,2}, Cikrikçili U³, Oruk G⁴, Kutbay NO⁵, Tanrikulu S⁶, Hekimsoy Z⁷, Hadzalic A⁸, Gorar S⁹, Omma T¹⁰, Mert M², Akbaba G¹¹, Yalin GY¹, Bayram F⁸, Ozkan M³, Yarman S¹.*J Clin Endocrinol Metab.* 2019 Jul 1;104(7):2527-2534. doi: 10.1210/je.2018-02202.**CONTEXT:** Dopamine agonist (DA)-induced impulse control disorder (ICD) in patients with prolactinomas is not sufficiently known.**OBJECTIVE:** To evaluate the prevalence of DA-induced ICDs and possible risk factors related to these disorders in patients with prolactinoma.**DESIGN, SETTING, AND PARTICIPANTS:** This is a cross-sectional multicenter study involving 308 patients with prolactinoma followed up in tertiary referral centers who received at least three months of DA therapy. DA-induced ICDs (pathological gambling, hypersexuality, compulsive shopping, and compulsive eating) and impulsivity were assessed using the Questionnaire for Impulsive-Compulsive Disorders in Parkinson Disease and the Barratt Impulsiveness Scale-11, respectively. Patients were evaluated in terms of parameters related to ICD development.**RESULTS:** Any ICD prevalence was 17% ($n = 51$). Hypersexuality was most common (6.5%). Although any ICD and hypersexuality were more common in male patients ($P = 0.009$, $P < 0.001$, respectively), compulsive eating was more common in female patients ($P = 0.046$). Current smoking, alcohol use, and gambling history were more frequent ($P = 0.033$, $P = 0.002$, $P = 0.008$, respectively) in patients with any ICD. In Barratt Impulsiveness Scale-11 total, attentional, motor, and nonplanning scores were higher in patients with any ICD ($P < 0.001$). Current smoking and alcohol use were more frequent ($P = 0.007$, $P = 0.003$, respectively) and percentage increase of testosterone levels at last visit was higher ($P = 0.021$) in male patients with prolactinomas with hypersexuality.**CONCLUSION:** Any ICD may be seen in one of six patients with prolactinoma who are receiving DA therapy. Endocrinology specialists should be aware of this side effect, particularly in male patients with a history of gambling, smoking, or alcohol use.EVALUATION OF THE NATURAL COURSE OF THYROID
NODULES IN PATIENTS WITH ACROMEGALY.Dogansen SC¹, Salmalıoğlu A², Yalin GY³, Tanrikulu S³, Yarman S³.*Pituitary.* 2019 Feb;22(1):29-36. doi: 10.1007/s11102-018-0923-1.**PURPOSE:** To investigate the nodular thyroid disease (NTD) and the natural course of thyroid nodules in patients with acromegaly.**METHODS:** 138 patients with acromegaly (73 F/65 M), whose initial thyroid ultrasonography performed in our university hospital, were included in this study. The frequencies of NTD, papillary thyroid cancer (PTC) and associated factors on nodule formation were investigated at initial assessment. Patients who had NTD continued to follow-up ($n = 56$) were re-evaluated with an ultrasonography performed after a mean 7-years follow-up period. The nodule size changes were compared with the initial data and the factors affecting nodule growth were investigated.**RESULTS:** The frequency of NTD was found 69%. Patients with NTD were older ($p = 0.05$), with higher baseline IGF-1%ULN (upper limit of normal) ($p = 0.01$). In patients with NTD,

the majority had similar nodule size (45%), decreased nodule size in 30% and nodule growth in 25%. In patients with active acromegaly at last visit, nodule growth was more significant ($p < 0.001$). For one unit change in the IGF-1 levels, nodule growth increased by 1.01 folds and presence of active acromegaly disease was related with ninefolds increase in nodule growth. The frequency of PTC was 14% in patients with nodule growth and PTC was diagnosed 11% of all acromegalic patients.

CONCLUSION: Both NTD and nodule growth is more frequent in active acromegalic patients. Thyroid nodules may show dynamic changes according to the disease activity and nodule growth should be closely monitored due to the risk of malignancy in patients with active acromegaly disease.

PITUITARY DYSFUNCTION DUE TO SPORTS-RELATED TRAUMATIC BRAIN INJURY.

Hacıoglu A¹, Kelestimur F², Tanriverdi F³.

Pituitary. 2019 Jun;22(3):322-331. doi: 10.1007/s11102-019-00937-z.

PURPOSE: After traumatic brain injury was accepted as an important etiologic factor of pituitary dysfunction (PD), awareness of risk of developing PD following sports-related traumatic brain injury (SR-TBI) has also increased. However there are not many studies investigating PD following SR-TBIs yet. We aimed to summarize the data reported so far and to discuss screening algorithms and treatment strategies.

METHODS: Recent data on pituitary dysfunction after SR-TBIs is reviewed on basis of diagnosis, clinical perspectives, therapy, screening and possible prevention strategies.

RESULTS: Pituitary dysfunction is reported to occur in a range of 15-46.6% following SR-TBIs depending on the study design. Growth hormone is the most commonly reported pituitary hormone deficiency in athletes. Pituitary hormone deficiencies may occur during acute phase after head trauma, may improve with time or new deficiencies may develop during follow-up. Central adrenal insufficiency is the only and most critical impairment that requires urgent detection and replacement during acute phase. Decision on replacement of growth hormone and gonadal deficiencies should be individualized. Moreover these two hormones are abused by many athletes and a therapeutic use exemption from the league's drug policy may be required.

CONCLUSIONS: Even mild and forgotten SR-TBIs may cause PD that may have distressing consequences in some cases if remain undiagnosed. More studies are needed to elucidate epidemiology and pathophysiology of PD after SR-TBIs. Also studies to establish screening algorithms for PD as well as strategies for prevention of SR-TBIs are urgently required.

NEUROENDOCRINE CHANGES AFTER ANEURYSMAL SUBARACHNOID HAEMORRHAGE.

Karaca Z¹, Hacıoglu A², Kelestimur F³.

Pituitary. 2019 Jun;22(3):305-321. doi: 10.1007/s11102-018-00932-w.

INTRODUCTION: The prevalence of pituitary dysfunction is high following aneurysmal subarachnoid hemorrhage (aSAH) and when occurs it may contribute to residual symptoms of aSAH such as decreased cognition and quality of life. Hypopituitarism following aSAH may have non-specific, subtle symptoms and potentially serious consequences if remained undiagnosed.

METHODS: We reviewed the literature on epidemiology, pathophysiology, diagnostic methods and management of neuroendocrine changes after aSAH as well as on the impact of pituitary dysfunction on outcome of the patient.

RESULTS: The prevalence rates of pituitary dysfunction after aSAH varies greatly across studies due to different diagnostic methods, though growth hormone deficiency is generally the most frequently reported followed by adrenocorticotrophic hormone, gonadotropin and thyroid stimulating hormone deficiencies. Pituitary deficiency tends to improve over time after aSAH but new onset deficiencies in chronic phase may also occur. There are no clinical parameters to predict the presence of hypopituitarism after aSAH. Age of the patient and surgical procedures are risk factors associated with development of hypopituitarism but the effect of pituitary dysfunction on outcome of the patient is not clear. Replacement of hypocortisolemia and hypothyroidism is essential but treatment of other hormonal insufficiencies should be individualized.

CONCLUSIONS: Hypopituitarism following aSAH necessitates screening despite lack of gold standard evaluation tests and cut-off values in the follow up, because missed diagnosis may lead to untoward consequences.

"FAT SHADOWS" FROM DXA FOR THE QUALITATIVE ASSESSMENT OF LIPODYSTROPHY: WHEN A PICTURE IS WORTH A THOUSAND NUMBERS.

Meral R¹, Ryan BJ², Malandrino N³, Jalal A¹, Neidert AH¹, Muniyappa R^{3,4}, Akıncı B⁵, Horowitz JF², Brown RJ³, Oral EA⁶.

Diabetes Care. 2018 Oct;41(10):2255-2258. doi: 10.2337/dc18-0978.

OBJECTIVE: Lipodystrophy syndromes are a heterogeneous group of disorders associated with selective absence of fat. Currently, the diagnosis is established only clinically.

RESEARCH DESIGN AND METHODS: We developed a new method from DXA scans called a "fat shadow," which is a color-coded representation highlighting only the fat tissue. We conducted a blinded retrospective validation study to assess its usefulness for the diagnosis of lipodystrophy syndromes.

RESULTS: We evaluated the fat shadows from 16 patients (11 female and 5 male) with generalized lipodystrophy (GL), 57 (50 female and 7 male) with familial partial lipodystrophy (FPLD), 2 (1 female and 1 male) with acquired partial lipodystrophy, and 126 (90 female and 36 male) control subjects. FPLD was differentiated from control subjects with 85% sensitivity and 96% specificity (95% CIs 72-93 and 91-99, respectively). GL was differentiated from nonobese control subjects with 100% sensitivity and specificity (95% CIs 79-100 and 92-100, respectively).

CONCLUSIONS: Fat shadows provided sufficient qualitative information to infer clinical phenotype and differentiate these patients from appropriate control subjects. We propose that this method could be used to support the diagnosis.

COMPREHENSIVE GENOTYPING OF TURKISH WOMEN WITH HIRSUTISM.

Polat S¹, Karaburgu S², Ünlühizarcı K², Dündar M³, Özkul Y³, Arslan YK⁴, Karaca Z², Kelestimur F^{2,5}.

J Endocrinol Invest. 2019 Feb 27. doi: 10.1007/s40618-019-01028-3. [Epub ahead of print]

INTRODUCTION: Hirsutism is a medical sign rather than a disease affects 5-8% of women of reproductive age. Hirsutism is associated with hyperandrogenemia in most patients excluding those with idiopathic hirsutism (IH). The most common cause of hirsutism is polycystic ovary syndrome (PCOS) followed by IH and idiopathic hyperandrogenemia (IHA); however, the clinical presentation of non-classical congenital adrenal hyperplasia (NCAH) in females is often indistinguishable from other hyperandrogenic disorders with common clinical signs such as hirsutism.

OBJECTIVE: The primary aim of the study is to examine the physical properties of the three genes and to make a detailed comparison of the mutations with the clinical data to contribute the etiology of hirsutism.

SUBJECTS AND METHODS: 122 women admitted to the Endocrinology Clinic at Erciyes University Hospital with hirsutism were enrolled in the study between 2013-2014. All the participants were clinically evaluated. Protein-encoding exons, exon-intron boundaries of CYP21A2 (including proximal promoter), CYP11B1 and HSD3B2 genes were analyzed via state-of-the-art genetic studies.

RESULTS: DNA sequencing analyses revealed two homozygous and three compound heterozygous 21-hydroxylase deficient (21OHD) NCAH patients. Additionally, three novel CYP21A2 mutations (A89V, M187I and G491S) and two novel CYP11B1 mutations (V188I and G87A) were determined. The frequencies of heterozygous mutations in CYP21A2 (including promoter), CYP11B1 and HSD3B2 genes were determined as 26.5% (15% coding region, 11.5% promoter), 11.5% and 0%, respectively.

CONCLUSION: 21OHD-NCAH prevalence was determined to be ~4%. Unexpectedly, high heterozygous mutation rates were observed in CYP11B1 gene and CYP21A2 promoter region. CYP11B1 and HSD3B2 deficiencies were not prevalent in Turkish women with hirsutism despite the existence of higher heterozygous mutation rate in CYP11B1.

**ASSESSMENT OF STATIC AND DYNAMIC PLANTAR DATA
OF PATIENTS WITH ACROMEGALY.**Sendur SN¹, Oguz S², Dagdelen S², Erbas T².*Pituitary. 2019 Aug;22(4):373-380. doi: 10.1007/s11102-019-00964-w.***PURPOSE:** To determine both static and dynamic plantar data of acromegalic subjects while barefoot.**METHODS:** Seventy acromegalic patients and 48 age-, sex-, weight- and height-matched healthy controls were included. Plantar variables were measured using the footscan gait system. The data included the width and length of each foot, relative force distribution in each quadrant, mean force applied to each foot and maximum pressure while walking. Maximum pressure data were obtained from ten parts of the foot. Injury risk assessments of five different regions were performed. To analyze balance, center of pressure (CoP) measurements were performed. The patients with acromegaly were compared with the controls. Furthermore, a comparison of patients with active and controlled acromegaly was performed.**RESULTS:** The foot was wider in acromegalic patients. The mean force on each foot was higher in cases of acromegaly (acromegaly: 1027 ± 180 N, control: 908 ± 180 N, $p = 0.001$). In the acromegalic individuals, the maximum pressure in the midfoot was higher, while the medial heel maximum pressure was lower (midfoot maximum pressure acromegaly: 11.3 ± 3.5 N/cm², control: 8.9 ± 3.7 N/cm², $p = < 0.001$). Injury risk was similar. CoP measurements elicited intact balance. In terms of static and dynamic plantar data, there was no difference between patients with active and controlled acromegaly.**CONCLUSIONS:** This is the first study to demonstrate that compared with healthy controls, patients with acromegaly experience great force on their feet while standing and high pressure in the midfoot during walking. Podiatric evaluation, custom molded orthotics and individualized rehabilitation programs for acromegalic patients may provide better force and pressure distribution throughout the foot and improve gait and skeletal symptoms.**TOXIC NODULAR GOITER AND THYROID CANCER: IS
HYPERTHYROIDISM PROTECTIVE AGAINST THYROID
CANCER?**Tam AA¹, Ozdemir D², Alkan A³, Yazicioglu O⁴, Yildirim N⁵, Kilicyazgan A⁶, Ersoy R², Cakir B².*Surgery. 2019 May 16. pii: S0039-6060(19)30134-5. doi: 10.1016/j.surg.2019.03.012.**[Epub ahead of print]***BACKGROUND:** The suppressive effect of the increase in thyroid hormone in patients with toxic nodular goiter is thought to protect the extranodular thyroid tissue from thyroid malignancy. In this study, we aimed to evaluate the prevalence and features of thyroid cancer in patients with toxic nodular goiter who underwent thyroidectomy.**METHODS:** Medical data of patients who had solitary toxic or nontoxic nodules and underwent total thyroidectomy were reviewed retrospectively. We reviewed the clinical, laboratory, and histopathologic features of patients with toxic nodular goiter and nontoxic solitary nodules.**RESULTS:** There were 73 patients with toxic nodular goiter and 366 patients with nontoxic solitary nodules. Median age was greater in the toxic nodular goiter compared with nontoxic solitary nodules patients (50 years; range: 18-73 vs 42 years; range: 18-83, $P < .001$). Median nodule diameters were 40.9 mm (range: 11.0-98.0) and 23.3 mm (range: 4.9-99.0) in patients with toxic nodular goiter and nontoxic solitary nodules, respectively ($P < .001$). Histopathologic examination revealed thyroid cancer in 14 patients (19%) with toxic nodular goiter and 132 (36.1%) patients with nontoxic solitary nodules ($P = .008$). Median tumor diameters were 6 mm (range: 1-50) in toxic nodular goiter and 14 mm (range: 1-80) in nontoxic solitary nodules ($P = .150$). The malignant nodule was the hyperfunctioning nodule in 7 patients with toxic nodular goiter; 4 were follicular and 3 were papillary thyroid cancer. The other 7 malignant foci were located in the suppressed contralateral lobe, and all were papillary microcarcinomas. The incidence of thyroid cancer outside the main nodule was similar in 2 groups ($P = .934$).**CONCLUSION:** Thyroid cancer in patients operated for toxic nodular goiter was 19%, which is not as rare as previously thought. A careful histopathologic examination of both the hyperfunctioning nodule and the extranodular thyroid tissue might help to disclose an unexpected tumor foci when thyroidectomy is performed in patients with toxic nodular goiter.**A CLINICAL AND PATHOPHYSIOLOGICAL APPROACH
TO TRAUMATIC BRAIN INJURY-INDUCED PITUITARY
DYSFUNCTION.**Temizkan S¹, Kelestimur F².*Pituitary. 2019 Jun;22(3):220-228. doi: 10.1007/s11102-019-00941-3.***PURPOSE:** This review aimed to evaluate the data underlying the pathophysiology of TBI-induced hypothalamo-pituitary dysfunction.**METHODS:** Recent literature about the pathophysiology of TBI-induced hypothalamo-pituitary dysfunction reviewed.**RESULTS:** Traumatic brain injury (TBI) is a worldwide epidemic that frequently leads to death; TBI survivors tend to sustain cognitive, behavioral, psychological, social, and physical disabilities in the long term. The most common causes of TBI include road accidents, falls, assaults, sports, work and war injuries. From an endocrinological perspective, TBIs are important, because they can cause pituitary dysfunction. Although TBI-induced pituitary dysfunction was first reported a century ago, most of the studies that evaluate this disorder were published after 2000. TBI due to sports and blast injury-related pituitary dysfunction is generally underreported, due to limited recognition of the cases.**CONCLUSION:** The underlying pathophysiology responsible for post-TBI pituitary dysfunction is not clear. The main proposed mechanisms are vascular injury, direct traumatic injury to the pituitary gland, genetic susceptibility, autoimmunity, and transient medication effects.

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Aday makaleler biçim özellikleri ile Türkiye Endokrinoloji ve Metabolizma Derneği'nin bilimsel yayın organı "Turkish Journal of Endocrinology and Metabolism" makale yazım kurallarına göre hazırlanmalıdır. Bilgi için <http://www.turkjem.org/> sayfasına giriş yapınız.

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