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Çocuk Endokrinolojisinde Güncel Yaklaşımlar

1st TAPES International Scientific Conference
Current Approaches in Pediatric Endocrinology

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İçindekiler

Contents

1. Diyabet / Diabetes Mellitus **8**

01 **Beyond the Initial Diagnosis: Clinical Clues, Diagnostic Challenges, and the Impact of Molecular Diagnosis in Pediatric MODY** 9
Özge Kaynar Sidimi, Ceren Karakoyunlu, Hande Turan

02 **Characteristics of Children Presenting with Diabetic Ketoacidosis and Evaluation of Diabetic Ketoacidosis Acute Complications** 10
Ibrahim Dikmen, Zhala Abdullayeva, Ilkyaz Türktan et al.

03 **Lipid Profile in Children and Adolescents with Type 1 Diabetes: Relationship with Glycemic Control and Diabetes Duration** 11
Ayşe Karadağ, Emine Kaygı, Aynura Aliyeva et al.

04 **The Effect of Continuous Glucose Monitoring on Health-Related Quality of Life and Glycemic Control in Children with Type 1 Diabetes** 14
Kürşat Çetin, Yasemin Funda Bahar, Ali Tırtar, Hale Tuhan, Mesut Parlak

05 **Comparative Analysis of Type 1 Diabetes Mellitus in Children** 15
Mammadova N.M., Ibrahim N.I., Kazımzade Z.V., Taghiyeva A.N., Eynalova M.K.

06 **Real-World Continuous Glucose Monitoring with the Anytime 5 System: On-Target GMI May Mask Hypoglycaemia in Type 1 Diabetes** 16
Gunduz Ahmadov

07 **HbA1c Changes Following Reimbursed Sibionics Use in Türkiye and Associations with CGM Metrics Across Different Reporting Periods** 17
Ilayda Altun, Özge Kaynar Sidimi, Olcay Evliyaoğlu

2. Obezite ve Metabolik Bozukluklar / Obesity and Metabolic Disorders **18**

08 **Childhood Obesity and Urine Chemistry: Uncovering Early Clues to Stone Formation** 19
Neşe Akcan, İlke Beyitler

09 **Can Mid-Upper Arm Circumference Predict Insulin Resistance and Dyslipidemia in Children and Adolescents with Obesity?** 20
Şükriye Tuğçe Çelebi, Fatma Kanat, Mehmet Bulut, Hakan Onur, Mehmet Nuri Özbek

10 **Micronutrient Deficiencies in Obese Children and Adolescents and Their Relationship with Glucose Metabolism** 23
Meltem Buhur Pirimoğlu

11 **MASLD in Adolescents with Obesity: Joint Evaluation of Central Adiposity, Dyslipidemia, and Genetic Susceptibility** 24
Shahri Khudiyeva, Sacide Pehlivan, Aylin Yetim Şahin

12 **Endocrine Comorbidity in Children with Bronchial Asthma** 25
T.T. Panakhova

3. Tiroid Hastalıkları / Thyroid Disorders **26**

13 An Overlooked Finding in Children Presenting with Elevated TSH: Iodine Deficiency 27
Seçil Çakır Gündoğan, Behiye Sarıkaya Özdemir, Şenay Savaş Erdeve

14 Clinical Characteristics and Long-Term Outcomes of Pediatric Graves' Disease: A Single-Center Experience 30
Mert Uçar, Elvan Bayramoğlu

15 Evaluation of Fatty Liver in Adolescents with Hashimoto's Thyroiditis Using Ultrasound Attenuation Scores: A Prospective Study 32
Fatma Özgüç Çömlek, Emine Uysal

4. Hipofiz ve Büyüme Bozuklukları / Pituitary and Growth Disorders **33**

16 Clinical and Laboratory Characteristics of Children with Growth Hormone Deficiency in the Republic of Uzbekistan 34
Kamola Kalanxodjayeva, Nasiba Alimova, Khurshidakhon Nasirova

17 Evaluation of Clinical, Hormonal and Treatment Characteristics in Pediatric Prolactinoma Cases: A Single-Center Experience 36
Aynura Aliyeva, Ayşe Karadağ, Emine Kaygı, Ülkü Gül Şiraz, Nihal Hatipoğlu

5. Puberte Bozuklukları / Disorders of Puberty **38**

18 Height SDS 'Catch-Down' During GnRH Analogue Treatment in Central Precocious Puberty and Achievement of Target Height 39
Emine Kaygı, Ayşe Karadağ, Aynura Aliyeva, Ülkü Gül Şiraz, Nihal Hatipoğlu

19 Clinical, Laboratory, and Cranial MRI Characteristics and Evaluation of Post-Treatment BMI and BMI SDS in Girls Diagnosed with Central Precocious Puberty: A Single-Center Experience 41
Ebru Gök

20 Inhibin A and Inhibin B in Predicting the Pubertal LH Response in Girls 44
Saliha Ahmetoğlu

21 IGF-1 and IGFBP-3 Cut-off Values for Predicting Pubertal Activation in 6–8-Year-Old Girls with Tanner Stage 2 Breast Development and Their Relationship with Gonadotropins, Estradiol and DHEA-S: A Retrospective Evaluation 45
Saliha Ahmetoğlu, İsmail Topal

6. Adrenal Bez Hastalıkları / Adrenal Disorders **46**

22 Evaluation of Metabolic Health Indicators in Children Diagnosed with Congenital Adrenal Hyperplasia 47
Zhala Abdullayeva, Ibrahim Dikmen, Eda Didem Kayakıran et al.

23 Examination and Treatment of a Patient Diagnosed with Adrenogenital Syndrome, Virilizing Form 48
S.M. Nasirova

24 3β-Hydroxysteroid Dehydrogenase Type 2 Deficiency: Clinical and Genetic Characterization and Prenatal Diagnosis in a High-Risk Family 49
Sultanova Sh.T., Alimov A.V., Salikhova Z.A., Kobilova S.B., Froyanchenko G.A.

25 Solid Right Adrenal Mass with Antenatal Presentation: Challenges in Differential Diagnosis 50
K.I. Khalmatova, G.N. Rakhimova, Z.Y. Khalimova, N.U. Alimova, Sh.T. Sultanova, A.A. Siddikov

7. Kalsiyum, Kemik ve Paratiroid Bozuklukları / Calcium, Bone and Parathyroid Disorders **51**

26 **A Diagnostically Challenging Case of Pseudohypoparathyroidism Type 1B: Endocrine Awareness in Bone Cysts** 52
Ibrahim Dikmen, Zhala Abdullayeva, Mustafa Doğan et al.

27 **Clinical Spectrum and Inheritance Patterns in IPPSD2 and IPPSD3: A Single-Center Experience** 54
Rabia Çoban, Seda Çakmaklı, Eda Çelebi Bitkin, Esra Deniz Papatya Çakır

28 **Contribution of Four-Dimensional Computed Tomography to the Localization of Parathyroid Lesions in Pediatric Primary Hyperparathyroidism** 55
Ertuğrul Sancak, Muammer Büyükinan, Ahmet Fatih Yılmaz et al.

8. Cinsiyet Gelişim Bozuklukları / Disorders of Sex Development **56**

29 **Clinical Characteristics and Genetic Diagnoses in Patients with Undescended Testes** 57
Gökçe Velioğlu Haşlak, Muhammed Furkan Yücer, Olcay Evliyaoğlu

30 **Hereditary Disorders in Patients with Cryptorchidism: Clinical and Genetic Aspects** 58
Sultanova Sh.T., Alimov A.V., Samadova U.S. et al.

31 **Timing of Orchidopexy in Cryptorchidism and Long-Term Testicular and Spermatogenic Outcomes** 59
Sultanova Sh.T., Alimov A.V., Samadova U.S., Kobilova S.B., Froyanchenko G.A.

9. Endokrin Otoimmünite ve Diğer Konular / Endocrine Autoimmunity and Other Topics **60**

32 **Immunological Abnormalities in Children with Autoimmune Endocrine Diseases: Association with Multiple Autoimmunity** 61
Yasemin Kınalı Çetin, Şule Büyük Yaytokgil, Emine Vezir

33 **Performance of a Large Language Model on Pediatric Endocrinology Test Questions Across Common Disorders** 62
Yayra Abdullaeva, Nasiba Alimova

10. Geç Gelen Bildiriler / Late-Breaking Abstracts **63**

34 **Epidemiology of Type 1 Diabetes in Children in Azerbaijan: Trends from 2017 to 2025 and Projections for 2026–2030** 64
Zulfiyya Kazımzade, Gunduz Ahmadov

BÖLÜM 1 · SECTION 1

Diyabet

Diabetes Mellitus

7 bildiri · 7 abstracts

BİLDİRİ № 01 · ABSTRACT 01 DOI: 10.5281/zenodo.23659710

Beyond the Initial Diagnosis: Clinical Clues, Diagnostic Challenges, and the Impact of Molecular Diagnosis in Pediatric MODY

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Keywords: MODY; Monogenic diabetes; GCK; HNF1A

Background and Objective: Maturity-onset diabetes of the young (MODY) is a heterogeneous group of monogenic diabetes disorders that may present with clinical features overlapping those of type 1 and type 2 diabetes, leading to diagnostic challenges and potential misclassification. Early recognition of MODY is essential for accurate diabetes classification, guiding genotype-specific treatment strategies, avoiding unnecessary therapies, and facilitating family screening. This study aimed to characterize the clinical and genetic features of pediatric patients with MODY, identify clinical clues relevant to the diagnostic process, and evaluate changes in diabetes classification and therapeutic management following molecular diagnosis.

Methods: This retrospective study included 20 patients followed at a pediatric endocrinology clinic in whom genetic variants associated with MODY had been identified. Demographic characteristics, age at diabetes diagnosis, clinical presentation, HbA1c levels, C-peptide concentrations, pancreatic autoantibody status, family history of diabetes, initial diabetes classification, molecular genetic findings, and treatment regimens before and after molecular diagnosis were recorded. Diagnostic delay was defined as the interval between the initial diabetes diagnosis and molecular confirmation. Changes in diabetes classification and therapeutic management following molecular diagnosis were assessed.

Results: A total of 20 patients [8 females (40%) and 12 males (60%)] were included. The mean age at the most recent evaluation was 13.6 ± 4.7 years, and the mean age at diabetes diagnosis was 9.7 ± 4.4 years. The median HbA1c level at presentation was 6.4% (range, 4.6–17.8%). Mean baseline C-peptide and insulin levels were 1.68 ± 0.97 ng/mL and 6.92 ± 4.69 µIU/mL. All patients tested negative for GAD, ICA, and IAA autoantibodies. The most frequently identified genes were GCK (n = 8, 40%) and HNF1A (n = 6, 30%). One patient presented with diabetic ketoacidosis. A family history of diabetes spanning at least two generations was documented in 76.5% of patients with available data (13/17), while seven patients exhibited mild, stable fasting hyperglycemia. Before molecular diagnosis, six patients (30%) had been classified as having other types of diabetes, including four initially diagnosed with type 1 diabetes and two with type 2 diabetes. Among the 12 patients with available data, the median diagnostic delay was 5 months (range, 0–32 months). Following molecular diagnosis, changes in therapeutic management were observed in five patients (25%), including sulfonylurea use in patients with HNF1A associated diabetes and complete discontinuation of insulin therapy in one patient with an HNF1A variant.

Conclusion: Negative pancreatic autoantibodies, preserved endogenous insulin secretion, mild and stable hyperglycemia, and a multigenerational family history of diabetes are important clinical clues that may raise suspicion of MODY in children. Nevertheless, the heterogeneity of clinical presentation may contribute to misclassification as type 1 or type 2 diabetes. In this cohort, changes in diabetes classification and therapeutic management were observed in 30% and 25% of patients, respectively, following molecular diagnosis. These findings highlight the importance of early recognition of MODY in children with suggestive clinical features, as molecular genetic evaluation may refine diabetes classification and inform individualized therapeutic decisions.

BİLDİRİ № 02 · ABSTRACT 02 DOI: 10.5281/zenodo.23659730

Characteristics of Children Presenting with Diabetic Ketoacidosis and Evaluation of Diabetic Ketoacidosis Acute Complications

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Keywords: Diabetic ketoacidosis; Type 1 diabetes; Electrolyte disturbances; Children

Introduction and Aim: Diabetic ketoacidosis (DKA) is a serious complication of type 1 diabetes that may lead to,electrolyte disorders,hyperchloremic metabolic acidosis, acute kidney injury, pancreatitis and cerebral edema.This study aimed to investigate the characteristics of acute complications in children with DKA at admission and during initial treatment.

Materials and Methods: Data of patients treated for DKA according to ISPAD criteria between 2010 and 2025 were retrospectively reviewed.Complications were asessed at presentation and during initial treatment.

Results: Seventy-four patients(mean age 8.18±4.02 years) were included.DKA was severe in 44.6%, moderate in 31.1% and mild in 24.3%. At presentation, complications were observed in 65% episodes, most commonly hypophosphatemia (35.1%), elevated creatinine (27%), hypochloremia (23%), hypermagnesemia (19.4%) and hyponatremia (14%).Elevated amylase and lipase were found in 7.8% and 4%, hypertriglyceridemia in 55.2%. Patients with hypophosphatemia had significantly higher HbA1c levels (13.16% vs. 12.13%). Mean insulin infusion duration was 16.1 ± 8.6 hours and time to acidosis resolution 13.7 ± 8.6 hours.

During initial treatment, complications occurred in 76% cases. The most frequent were hypokalemia (57%), hypomagnesemia (54%), hypophosphatemia (50%), hypocalcemia (24%) and hyperchloremic acidosis (10%).Cerebral edema and acute kidney injury each occured in one patient. Hyperchloremic acidosis was associated with longer insulin infusion and DKA resolution time.Hypokalemia and hypophosphatemia were significantly associated with severe DKA, whereas complication frequency was unrelated to age, puberty, or HbA1c.Most electrolyte disturbances developed within the first 12 hours of treatment.

Conclusion: Electrolyte disturbances are common during pediatric DKA,affecting approximately 80% of episodes. Hypokalemia and hypophosphatemia are more frequent in severe DKA. Hyperchloremic metabolic acidosis may prolong treatment, highlighting the importance of carefully evaluating chloride administration in initial fluid therapy Higher HbA1c in hypophosphatemic patients may reflect the effect of prolonged hyperglycemia on phosphate balance.Close monitoring of electrolytes from the first hours of treatment is essential, particularly in children with severe DKA.

BİLDİRİ № 03 · ABSTRACT 03 DOI: 10.5281/zenodo.23659734

Lipid Profile in Children and Adolescents with Type 1 Diabetes: Relationship with Glycemic Control and Diabetes Duration

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Keywords: Type 1 diabetes; Dyslipidemia; HbA1c; Albuminuria

Introduction and Aim: Dyslipidemia is a modifiable risk factor for early atherosclerosis in type 1 diabetes (T1D). We evaluated associations of the lipid profile with glycemic control, diabetes duration, insulin dose and renal parameters in children and adolescents with T1D.

Methods: This cross-sectional study included patients with T1D followed at a pediatric endocrinology clinic. Associations between lipid parameters and clinical variables were analyzed using Spearman correlation.

Results: Of 204 patients, 52.8% were male and 85.1% pubertal. Mean age at diagnosis was 7.46±3.54 years, diabetes duration 6.59±3.85 years and HbA1c 8.90±1.90%. Median whole-blood thiamine pyrophosphate was 97.1 (29.8–183.6) µg/L and median pyridoxal-5'-phosphate 14.1 (10.8–18.9) µg/L. Mean total cholesterol was 172.3±38.3 mg/dL, LDL 94.2±30.9 mg/dL, HDL 56.9±13.9 mg/dL, and median triglycerides 91.9 (62.3–126.3) mg/dL. Dyslipidemia was present in 17% (29/171). HbA1c correlated positively with total cholesterol ($r=0.288$; $p<0.001$), LDL ($r=0.262$; $p=0.001$) and triglycerides ($r=0.381$; $p<0.001$), and negatively with HDL ($r=-0.165$; $p=0.042$). Mean HbA1c over the previous 12 months showed the strongest correlation with triglycerides ($r=0.406$; $p<0.001$). Diabetes duration correlated with total cholesterol ($r=0.215$; $p=0.008$) and LDL ($r=0.229$; $p=0.005$); basal insulin dose correlated positively with triglycerides ($r=0.222$; $p=0.006$) and negatively with HDL ($r=-0.236$; $p=0.004$). Albuminuria was detected in 16.7%, and the urine albumin-to-creatinine ratio correlated with triglycerides ($r=0.279$; $p=0.001$).

Conclusion: In children with T1D, poor glycemic control, particularly elevated long-term HbA1c, is associated with an atherogenic lipid profile. Lipid levels increase with longer diabetes duration and accompany albuminuria. Improving glycemic control appears to be a primary target for improving

the lipid profile.

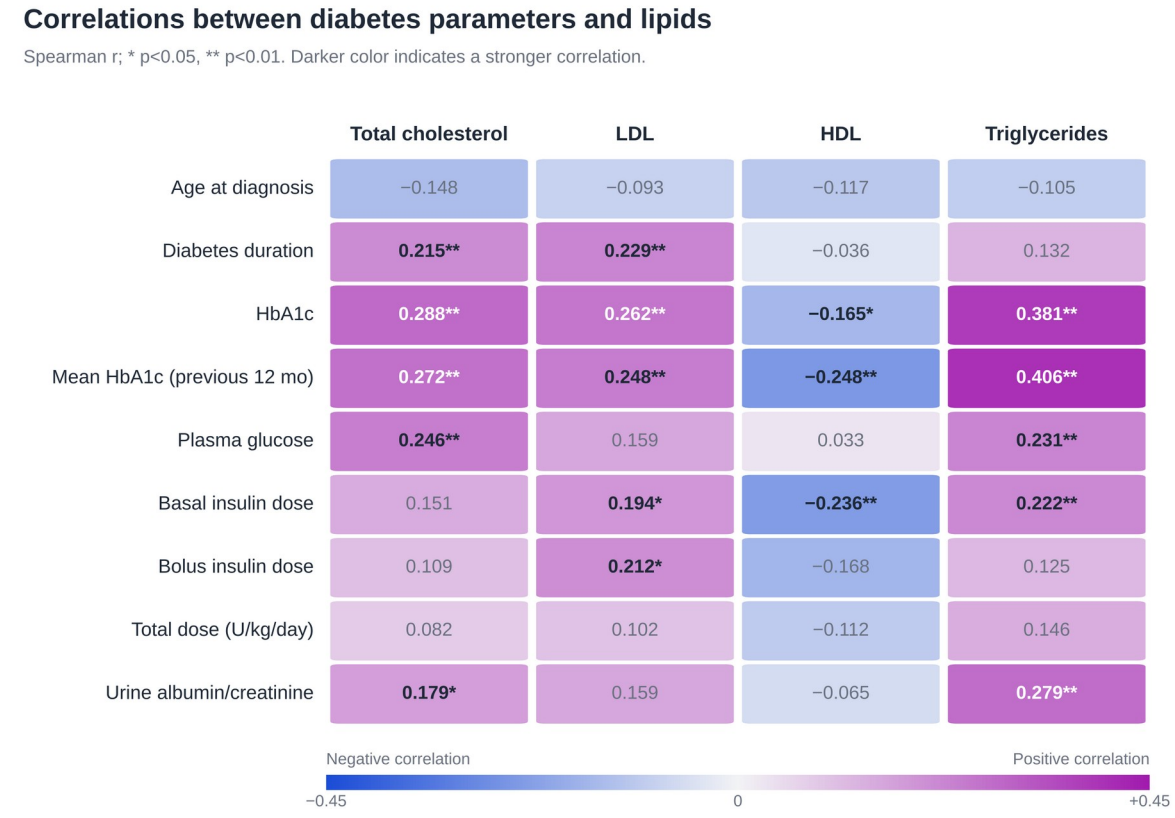

Figure 1. Spearman correlations between diabetes-related parameters and serum lipids. * p<0.05; ** p<0.01.

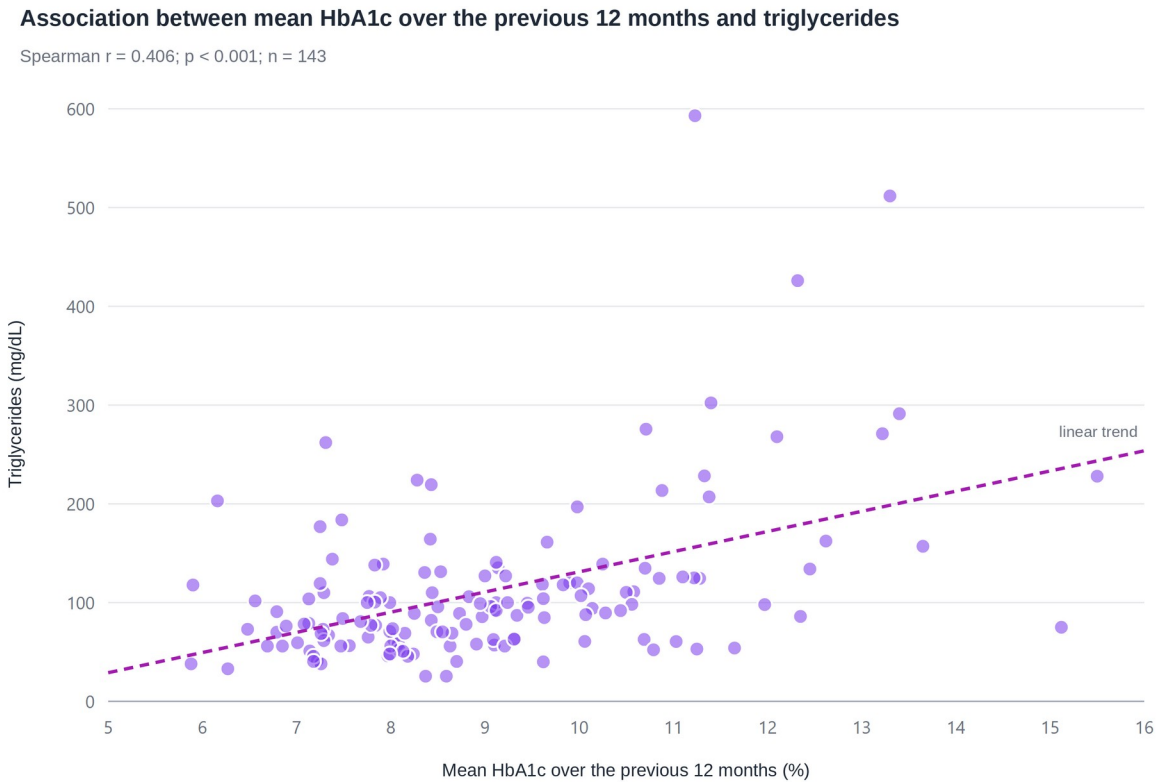

Figure 2. Association between mean HbA1c over the previous 12 months and triglycerides (Spearman r =

0.406; p < 0.001; n = 143). The dashed line shows the linear trend.

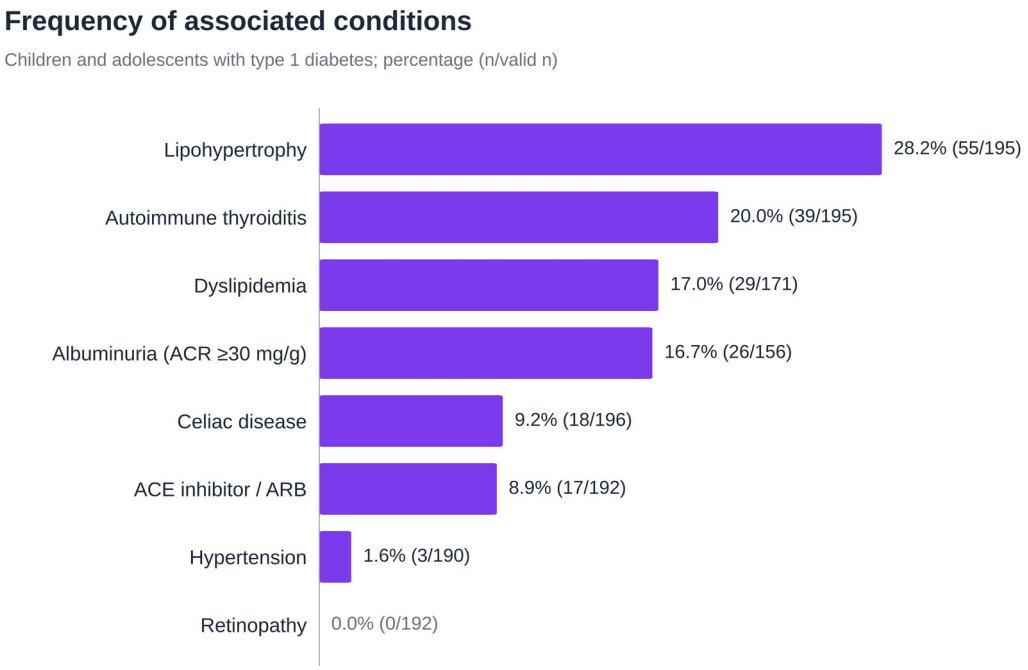

Figure 3. Frequency of associated conditions in children and adolescents with type 1 diabetes, % (n/valid n).

BİLDİRİ № 04 · ABSTRACT 04 DOI: 10.5281/zenodo.23659736

The Effect of Continuous Glucose Monitoring on Health-Related Quality of Life and Glycemic Control in Children with Type 1 Diabetes

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Keywords: Continuous glucose monitoring; Type 1 diabetes; Quality of life; Glycemic control

Introduction: Continuous glucose monitoring (CGM) systems have become an integral part of type 1 diabetes mellitus (T1DM) management by improving glycemic monitoring and reducing diabetes burden. Since January 2025, reimbursement of CGM systems by the Social Security Institution of Türkiye has enabled their wider use in children with T1DM. However, data on their effects on health-related quality of life (HRQoL) in children remain limited.

Objective: To evaluate the effects of CGM use on health-related quality of life and glycemic control in children with T1DM after six months of sensor use.

Methods: A total of 102 children with T1DM aged 5–18 years and followed at the Department of Pediatric Endocrinology, Akdeniz University, were included. Parents completed the Pediatric Quality of Life Inventory (PedsQL 4.0) before CGM initiation and after six months of use. HbA1c values before CGM and at month six were compared. Paired statistical analyses were applied, and $p<0.05$ was considered statistically significant.

Results: After six months of CGM use, the total PedsQL score increased significantly from 72.4±13.8 to 77.6±11.8 (mean difference +5.2; 95% CI 3.9–6.5; $p<0.001$). Significant improvements were observed in all domains: physical health (73.1±17.3 vs. 77.3±15.1), emotional functioning (65.3±16.9 vs. 71.4±16.2), social functioning (87.4±16.6 vs. 92.0±13.2), school functioning (63.6±19.0 vs. 70.2±19.5) and psychosocial health (72.1±14.0 vs. 77.8±12.7) (all $p<0.001$). Effect sizes were moderate; the greatest improvement was seen in emotional functioning and total health-related quality of life. Mean HbA1c decreased from 8.9±2.4% to 8.1±1.5% (mean reduction 0.75%; $p=0.006$).

Conclusion: CGM use was associated with significant improvements in both health-related quality of life and glycemic control in children with T1DM. The most pronounced benefits were observed in emotional functioning and overall health-related quality of life, supporting the routine use of CGM in pediatric diabetes care.

BİLDİRİ № 05 · ABSTRACT 05 DOI: 10.5281/zenodo.23659740

Comparative Analysis of Type 1 Diabetes Mellitus in Children

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Keywords: Type 1 diabetes mellitus; Children; Incidence; Retrospective analysis

Introduction: Diabetes mellitus (DM) is a chronic metabolic disease caused by reduced insulin secretion or impaired insulin action. According to the World Health Organization (WHO) and the International Diabetes Federation (IDF), approximately 1.1–1.2 million children and adolescents worldwide were living with type 1 diabetes mellitus (T1DM) in 2019–2020. The annual incidence of T1DM among children and adolescents increases by approximately 2–5%. Currently, approximately 8.75 million people of all ages worldwide live with T1DM. The increasing global prevalence of diabetes, particularly the growing number of cases among children and adolescents, highlights the medical and social significance of this disease.

Methods: The study was conducted in the Endocrinology Department of Children's Clinical Hospital No 6 in Baku, Azerbaijan. Statistical data were collected through a retrospective analysis of the medical records of children aged 0–18 years examined and treated between 2019 and 2020. Diagnoses were established according to the relevant WHO criteria, and appropriate examinations and treatment were provided.

Results: In 2019, 933 children with T1DM were hospitalized, including 199 (21.3%) newly diagnosed cases. Girls accounted for 500 (53.6%) patients and boys for 433 (46.4%). Of these, 276 (29.6%) were from Baku and 657 (70.4%) from other regions of Azerbaijan. In 2020, 609 children were hospitalized, including 255 (41.8%) newly diagnosed cases. Girls accounted for 313 (51.4%) patients and boys for 296 (48.6%). A total of 242 (39.8%) patients were from Baku and 367 (60.2%) from other regions. T1DM was identified in two children from the same family in three cases.

Conclusion: Newly diagnosed T1DM cases in creased from 199 in 2019 to 255 in 2020, representing a 28.1% increase. Investigating childhood T1DM prevalence, epidemiological patterns, age-related and gender-related characteristics, and disease trends is essential for improving early diagnosis and preventing complications.

BİLDİRİ № 06 · ABSTRACT 06 DOI: 10.5281/zenodo.23659742

Real-World Continuous Glucose Monitoring with the Anytime 5 System: On-Target GMI May Mask Hypoglycaemia in Type 1 Diabetes

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Keywords: Continuous glucose monitoring; Type 1 diabetes; Hypoglycaemia; Glucose management indicator

Introduction: Continuous glucose monitoring (CGM) has become a cornerstone of modern diabetes management. Time in range (TIR), time below range (TBR) and the coefficient of variation (CV) reveal risks not captured by HbA1c or its CGM-derived equivalent, the glucose management indicator (GMI). The Anytime 5 CGM system (Yuwell, People's Republic of China) has recently been introduced in Azerbaijan, but real-world CGM data from the country remain limited.

Objective: To evaluate glycaemic profiles of patients monitored with the Anytime 5 system and to identify discordance between GMI and hypoglycaemia exposure in type 1 diabetes.

Methods: We retrospectively analysed CGM data uploaded to the Anytime View cloud platform between June and October 2026. For each patient, the most recent sensor with ≥70% active wear time was included. Metrics were assessed against the International Consensus on Time in Range targets: TIR 70–180 mg/dL >70%, TBR <70 mg/dL <4%, TBR <54 mg/dL <1% and CV ≤36%. Data are presented as median (range).

Results: Of 14 registered patients, 11 had analysable data. In type 1 diabetes (n=5; 3 adolescents, 2 adults), median TIR was 59% (45–83%), GMI 6.5% (6.3–8.1%), TBR 9% (0–22%) and CV 40% (31.8–55.6%); only 2 patients achieved the TIR target. All 4 patients with GMI ≤7.0% had TBR ≥4%. Three patients exceeded the level 2 hypoglycaemia target (time <54 mg/dL >1%; maximum 10%), and all 3 patients with TBR ≥9% had a CV >36%. In the most severe case, a GMI of 6.3% coexisted with a TBR of 22% and 38 hypoglycaemic events (mean duration 100 minutes) over 14 days. Hypoglycaemia was predominantly morning-related, with morning TBR of 20–54% in 3 patients. One patient had persistent hyperglycaemia without hypoglycaemia (GMI 8.1%, TAR 55%). In other groups, one patient with type 2 diabetes had a TIR of 77% and a TBR of 1%; one patient without a recorded diagnosis showed diabetic-range glycaemia (GMI 8.2%, TIR 42%); and four normoglycaemic or prediabetic individuals had mean glucose of 92–127 mg/dL and CV of 12.6–18.9%. Sensor continuity was a frequent problem: 7 of 11 patients had no sensor data in the preceding 4 weeks.

Conclusion: In type 1 diabetes, an on-target GMI may mask clinically significant, predominantly morning hypoglycaemia, and high glycaemic variability signals hypoglycaemia risk. CGM reports should therefore always be interpreted using TBR and CV alongside GMI. Structured follow-up to ensure uninterrupted sensor wear is essential to maximise the clinical benefit of CGM. These preliminary observations warrant confirmation in larger cohorts.

BİLDİRİ № 07 · ABSTRACT 07 DOI: 10.5281/zenodo.23659744

HbA1c Changes Following Reimbursed Sibionics Use in Türkiye and Associations with CGM Metrics Across Different Reporting Periods

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Keywords: Type 1 diabetes; Continuous glucose monitoring; Sibionics; HbA1c

Background: Expanded access to reimbursed continuous glucose monitoring (CGM) in Türkiye from January 2025 provided an opportunity to evaluate glycaemic outcomes in routine clinical practice. This study assessed changes in HbA1c following Sibionics use and associations between laboratory HbA1c and CGM metrics over the preceding three months, one month, and 14 days.

Methods: This single-centre retrospective study included 51 individuals with type 1 diabetes whose device was recorded exclusively as Sibionics. Mean HbA1c recorded before January 2025 was compared with the latest available HbA1c after sensor initiation using the Wilcoxon signed-rank test. In 46 participants with complete data across all three reporting periods, associations of the corresponding laboratory HbA1c with glucose management indicator (GMI), mean sensor glucose, time in range (TIR), and time in tight range (TITR) were assessed using Spearman correlation. Mean absolute HbA1c–GMI differences were also calculated.

Results: At a median of 12.6 months after sensor initiation, mean HbA1c decreased from 8.87 ± 2.09% to 7.81 ± 1.08%, representing a mean reduction of 1.07 percentage points ($p<0.001$).

HbA1c correlated positively with three-month, one-month, and 14-day GMI ($\rho=0.831$, 0.810, and 0.735, respectively) and mean sensor glucose ($\rho=0.849$, 0.807, and 0.739, respectively). Negative correlations were observed with TIR ($\rho=-0.828$, −0.745, and −0.700, respectively) and TITR ($\rho=-0.827$, −0.761, and −0.723, respectively; all $p<0.001$). Three-month TIR and TITR showed nearly identical correlations with HbA1c. Mean absolute HbA1c–GMI differences were 0.54, 0.48, and 0.53 percentage points, respectively.

Conclusions: Sibionics use following expanded reimbursement access was associated with a significant reduction in HbA1c. Three-month CGM metrics, including TIR and TITR, showed numerically stronger correlations with laboratory HbA1c than one-month and 14-day metrics. These findings suggest that reviewing three-month CGM summaries alongside the most recent 14-day data may provide a more comprehensive assessment of glycaemic control during outpatient follow-up.

BÖLÜM 2 · SECTION 2

Obezite ve Metabolik Bozukluklar

Obesity and Metabolic Disorders

5 bildiri · 5 abstracts

BİLDİRİ № 08 · ABSTRACT 08 DOI: 10.5281/zenodo.23659746

Childhood Obesity and Urine Chemistry: Uncovering Early Clues to Stone Formation

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Keywords: Childhood obesity; Urine pH; Urinary calcium excretion; Nephrolithiasis risk

Background: Emerging evidence in adults suggests an association between obesity and nephrolithiasis, potentially mediated by alterations in urinary composition, including reduced urine pH. However, data in pediatric populations remain limited.

Objectives: This study aimed to investigate the association between body mass index (BMI) and urinary biochemical parameters, particularly urinary calcium excretion and urine pH, in school-aged children.

Methods: In this cross-sectional study, 541 children aged 6–18 years from a school-based screening program (2019–2022) were included. Participants were categorized according to BMI standard deviation scores (BMI-SDS). Anthropometric measurements, blood pressure, and spot urine samples were obtained. Urinary pH, density, calcium, creatinine, and calcium-to-creatinine ratio were analyzed. Statistical analyses included nonparametric tests, Spearman correlation and multivariable linear regression models adjusted for age, sex and urine density.

Results: Among participants, 62.4% had normal BMI, 26.4% were overweight, and 10.5% were obese. No significant differences were observed across BMI categories for urinary pH, density, calcium, creatinine or calcium-to-creatinine ratio (all $p>0.05$), with negligible effect sizes. BMI-SDS was not associated with any urinary parameter. A weak inverse correlation was identified between weight-SDS and urine pH ($r=-0.088$, $p=0.041$). In multivariable analyses, BMI was not an independent predictor of urinary outcomes, whereas age emerged as the most consistent determinant.

Conclusion: BMI-defined adiposity was not associated with urinary biochemical parameters in this pediatric cohort. The observed weak association between higher body weight and lower urine pH appears to be of limited clinical significance. These findings suggest that urinary metabolic profiles in children may be driven predominantly by physiological factors rather than adiposity. Longitudinal studies are warranted to clarify potential long-term implications.

BİLDİRİ № 09 · ABSTRACT 09 DOI: 10.5281/zenodo.23659748

Can Mid-Upper Arm Circumference Predict Insulin Resistance and Dyslipidemia in Children and Adolescents with Obesity?

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Keywords: Childhood obesity; Mid-upper arm circumference; Insulin resistance; Dyslipidemia

Background and Objective

Childhood obesity is associated with cardiometabolic complications, including insulin resistance and dyslipidemia, highlighting the importance of early risk identification. This study aimed to investigate the associations of mid-upper arm circumference (MUAC) with insulin resistance and dyslipidemia in children and adolescents with obesity and to determine its incremental discriminatory value beyond anthropometric measures.

Methods

A total of 169 children and adolescents with obesity were included. Demographic characteristics, pubertal stage, anthropometric measurements, and biochemical parameters were evaluated. Insulin resistance was defined using sex- and puberty-specific HOMA-IR cutoffs, while dyslipidemia was defined as elevated triglycerides and/or reduced HDL cholesterol. Associations between anthropometric indices and metabolic parameters were assessed using Spearman correlation analysis. Discriminatory performance was evaluated using ROC analysis, and independent associations were examined using logistic regression adjusted for age, sex, Tanner stage, and BMI-SDS. The incremental contributions of MUAC and waist circumference were also investigated.

Results

Among 169 participants, 104 (61.5%) were girls. Insulin resistance was identified in 112 (66.3%) and dyslipidemia in 100 (59.2%). MUAC was positively correlated with HOMA-IR ($r = 0.368$, $p < 0.001$) and the triglyceride-to-HDL cholesterol ratio ($r = 0.164$, $p = 0.033$). Hepatic steatosis was present in 86 of 157 participants (54.8%) with available data. Both MUAC and waist circumference were significantly higher in participants with hepatic steatosis (both $p < 0.001$). After adjustment, MUAC was not independently associated with insulin resistance ($p = 0.955$) or dyslipidemia ($p = 0.472$). Adding MUAC did not significantly improve discrimination, whereas waist circumference significantly improved for both outcomes ($p = 0.038$ and $p = 0.039$, respectively).

Conclusion

Although MUAC correlated with markers of insulin resistance and dyslipidemia, it provided no independent incremental value for identifying metabolic abnormalities. Waist circumference may be a more informative anthropometric measure for cardiometabolic risk assessment in children and

adolescents with obesity.

Table 1. Demographic and anthropometric characteristics of the study population

Variable	Total (n=169)	Girls (n=104)	Boys (n=65)	p
Age (years)	13.07 (9.97–15.41)	13.97 (10.36–15.89)	12.40 (9.50–14.61)	**0.038**
Height SDS	0.64 ± 1.23	0.60 ± 1.23	0.69 ± 1.23	0.633
Weight (kg)	77.84 ± 23.88	79.03 ± 22.90	75.93 ± 25.44	0.425
BMI (kg/m²)	30.64 (27.78–34.99)	31.06 (27.59–35.48)	30.38 (28.53–31.76)	0.173
BMI-SDS	2.68 (2.31–3.32)	2.85 (2.44–3.40)	2.51 (2.20–2.88)	**<0.001**
MUAC (cm)	31.67 ± 4.55	32.14 ± 4.74	30.93 ± 4.15	0.083
Waist circumference (cm)	91.00 (82.00–99.50)	88.00 (81.53–98.00)	95.00 (88.00–101.00)	**0.015**
Hip circumference (cm)	105.84 ± 15.47	107.15 ± 15.58	103.74 ± 15.19	0.162
Waist-to-hip ratio	0.87 (0.82–0.93)	0.85 (0.79–0.89)	0.91 (0.87–0.96)	**<0.001**
Waist-to-height ratio	0.58 (0.54–0.63)	0.57 (0.53–0.62)	0.60 (0.56–0.64)	**0.002**

MUAC, mid-upper arm circumference. Data are presented as mean ± standard deviation or median (25th–75th percentile). Distributions were assessed using the Shapiro–Wilk test; Welch's t-test or Mann–Whitney U test was applied as appropriate. P values were not adjusted for multiple comparisons.

Table 2. Biochemical and metabolic characteristics of participants by sex

Parameter	Total (n=169)	Girls (n=104)	Boys (n=65)	p
Fasting glucose (mg/dL)	89 (86–93)	89 (85–93)	91 (88–93)	0.143
Fasting insulin (µIU/mL)	20.30 (13.81–28.09)	22.12 (15.55–29.50)	16.42 (12.81–25.70)	**0.012**
HOMA-IR	4.46 (3.07–6.33)	4.77 (3.52–6.60)	3.99 (2.75–5.60)	**0.018**
HbA1c (%)	5.28 (5.00–5.50)	5.20 (4.95–5.50)	5.40 (5.10–5.60)	**0.031**
Uric acid (mg/dL)	6.00 (5.09–6.85)	5.90 (5.10–6.80)	6.20 (5.00–6.95)	0.809
Triglycerides (mg/dL)	115 (87.5–173)	110.5 (86–160)	127 (90–186)	0.156
HDL-C (mg/dL)	42 (38–48)	44 (38–50.25)	41 (35–47)	0.074
LDL-C (mg/dL)	85.32 ± 23.20	89.31 ± 22.61	78.99 ± 22.87	**0.005**
Total cholesterol (mg/dL)	155.88 ± 27.50	159.09 ± 27.07	150.73 ± 27.60	0.056
TG/HDL-C ratio	2.70 (1.98–4.25)	2.50 (1.95–3.98)	3.06 (2.13–4.82)	0.101

TG, triglycerides; HDL-C, high-density lipoprotein cholesterol, MUAC, mid-upper arm circumference. Data are presented as mean ± standard deviation or median (25th–75th percentile).

Table 3. Correlations of anthropometric measures with HOMA-IR and the TG/HDL-C ratio

Anthropometric measure	HOMA-IR (r)	p	TG/HDL-C (r)	p
BMI-SDS	0.293	**<0.001**	0.095	0.220
MUAC	0.368	**<0.001**	0.164	**0.033**
Waist circumference	0.371	**<0.001**	0.244	**0.001**
Waist-to-hip ratio	−0.059	0.442	0.191	**0.013**
Waist-to-height ratio	0.193	**0.012**	0.190	**0.014**

n = 169. Spearman correlation analysis was used. r, Spearman correlation coefficient, MUAC, mid-upper arm circumference, TG, triglycerides; HDL-C, high-density lipoprotein cholesterol

Table 4. Adjusted Associations of Anthropometric Measures with Metabolic Abnormalities and Hepatic Steatosis

Anthropometric measure	Insulin resistance aOR (95% CI)	p-value	Elevated TG / Low HDL-C[1] aOR (95% CI)	p-value	Hepatic steatosis aOR (95% CI)	p-value
MUAC	1.00 (0.88–1.15)	0.955	1.05 (0.92–1.19)	0.472	1.08 (0.93–1.26)	0.305
Waist circumference	1.05 (1.00–1.10)	**0.044**	1.05 (1.00–1.09)	**0.044**	1.07 (1.02–1.12)	**0.011**
Waist-to-hip ratio	1.34 (0.82–2.19)	0.250	1.42 (0.91–2.22)	0.122	1.53 (0.93–2.53)	0.097
Waist-to-height ratio	1.24 (0.56–2.74)	0.589	1.47 (0.72–3.00)	0.294	2.61 (1.13–6.05)	**0.025**

Adjusted odds ratios (aORs) were estimated using separate multivariable logistic regression models for each anthropometric measure, adjusted for age, sex, Tanner stage (categorical), and body mass index standard deviation score (BMI-SDS).

Odds ratios correspond to a 1-cm increase in MUAC and waist circumference and a 0.1-unit increase in waist-to-hip and waist-to-height ratios.

The analyses included 169 participants for insulin resistance and lipid abnormalities and 157 participants for hepatic steatosis.

[1] Elevated triglycerides (TG) and/or low high-density lipoprotein cholesterol (HDL-C). Elevated TG was defined as ≥100 mg/dL in children younger than 10 years and ≥130 mg/dL in those aged 10–19 years. Low HDL-C was defined as <40 mg/dL. Insulin resistance was defined using sex- and puberty-specific HOMA-IR thresholds: >2.22 for girls and >2.67 for boys at Tanner stage 1; >3.82 for girls and >5.22 for boys at Tanner stages 2–5.

Abbreviations: aOR, adjusted odds ratio; CI, confidence interval; MUAC, mid-upper arm circumference; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol

BİLDİRİ № 10 · ABSTRACT 10 DOI: 10.5281/zenodo.23659750

Micronutrient Deficiencies in Obese Children and Adolescents and Their Relationship with Glucose Metabolism

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Keywords: Obesity; Vitamin D; Transferrin saturation; Insulin resistance

Introduction and Aim: Micronutrient deficiencies may occur in obese children; however, their relationship with glucose metabolism remains controversial. This study aimed to evaluate the prevalence of vitamin D, vitamin B12 and iron deficiency in obese children and adolescents and their association with insulin resistance and prediabetes.

Methods: Anthropometric measurements, pubertal stage, transferrin saturation (TSAT), 25(OH)D, vitamin B12, HbA1c, fasting glucose and insulin levels of 152 patients with a body mass index (BMI) ≥95th percentile who presented to the Pediatric Endocrinology Outpatient Clinic of Düzce Atatürk State Hospital between October 2025 and August 2026 were retrospectively reviewed. Insulin resistance was defined using puberty- and sex-specific HOMA-IR cutoffs.

Results: Of the patients, 56.6% were female and 88.2% were pubertal (age: 13.9±2.6 years). Vitamin D insufficiency (<20 ng/mL) was found in 83.6%, vitamin D deficiency (<12 ng/mL) in 40.8%, vitamin B12 deficiency (≤200 pg/mL) in 19.7% and iron deficiency (TSAT <16%) in 32.2%. Multiple deficiencies were present in 38.2%. The prevalence of insulin resistance was 59.2% and that of prediabetes 52.0%. Female sex (OR 2.39; 95% CI 1.08–5.26) and BMI SDS (OR 2.06 per 1-unit increase; 95% CI 1.15–3.71) were season-independent risk factors for vitamin D deficiency. After adjustment for age, sex, puberty and BMI SDS, TSAT was lower in patients with insulin resistance (20.3% vs 25.7%; p=0.016), prediabetes (19.5% vs 25.2%; p=0.008) and impaired fasting glucose (16.1% vs 22.6%; p=0.036). HOMA-IR was independently and inversely associated with TSAT (β=−0.31; p<0.001). Vitamin D and vitamin B12 deficiencies were not associated with insulin resistance. Among patients with prediabetes, 84% were diagnosed based on HbA1c, and TSAT was inversely associated with HbA1c.

Conclusion: The prevalence of vitamin D deficiency increased with rising BMI SDS. As reported in the literature, this may be explained by the lifestyle and dietary habits of obese individuals and by altered vitamin D metabolism in obesity. The other finding, lower TSAT in individuals with insulin resistance, may be explained by increased inflammation and hepcidin in obesity. The association of low TSAT with prediabetes independent of obesity may result. from the effect of iron deficiency on HbA1c levels; therefore, a diagnosis of prediabetes should be confirmed by OGTT.

BİLDİRİ № 11 · ABSTRACT 11 DOI: 10.5281/zenodo.23659752

MASLD in Adolescents with Obesity: Joint Evaluation of Central Adiposity, Dyslipidemia, and Genetic Susceptibility

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Keywords: MASLD; Obesity; Dyslipidemia; PNPLA3

Background/Objectives

This study evaluated associations of metabolic dysfunction-associated steatotic liver disease (MASLD) with waist circumference standard deviation score (SDS), dyslipidemia, HOMA-IR, and PNPLA3/TM6SF2 risk allele carriage in adolescents with obesity.

Methods

This single-center cross-sectional study included 40 non-obese controls and participants with obesity without (n=24) or with MASLD (n=76). Classification used ultrasonographic steatosis and metabolic criteria. Genotyping used PCR-RFLP. Dyslipidemia was defined as triglycerides ≥130, HDL cholesterol <40, or LDL cholesterol ≥130 mg/dL. Carrier frequencies were compared using Fisher–Freeman–Halton tests. Exploratory Firth regression included waist circumference SDS, dyslipidemia, HOMA-IR, and both variants in 74 participants with obesity and complete data. Separate Holm corrections addressed carrier comparisons and model coefficients.

Results

Age did not differ significantly across groups. Participants with obesity and MASLD had higher median waist circumference SDS (3.48 vs 3.20), AST (20.40 vs 18.50 U/L), ALT (21.30 vs 17.25 U/L), and GGT (17 vs 12 U/L) than those without MASLD (all nominal $p<0.05$); BMI SDS and HOMA-IR did not differ significantly. PNPLA3 genotyping was unsuccessful in 31 participants. PNPLA3 carriage was 41.9%/42.9%/61.4% and TM6SF2 carriage 12.5%/8.7%/19.7% in controls/obesity without MASLD/obesity with MASLD (Holm-adjusted $p=0.286/0.454$). Dyslipidemia frequencies in the obese groups were 12.5%/55.3%. Adjusted ORs (95% CI): dyslipidemia 12.32 (2.82–87.95), waist SDS 3.28 (1.15–11.89), HOMA-IR 0.73 (0.53–0.95), PNPLA3 2.99 (0.82–12.26), TM6SF2 9.07 (1.36–123.30). Only dyslipidemia remained significant after Holm correction ($p=0.002$).

Conclusions

Dyslipidemia was associated with MASLD. Risk allele carriage was numerically higher with MASLD, but group differences were nonsignificant. The inverse HOMA-IR association did not survive Holm correction. Small sample size, wide confidence intervals, unsuccessful genotyping, and PNPLA3 Hardy–Weinberg disequilibrium warrant cautious interpretation.

BİLDİRİ № 12 · ABSTRACT 12 DOI: 10.5281/zenodo.23659754

Endocrine Comorbidity in Children with Bronchial Asthma

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Keywords: Bronchial asthma; Obesity; Endocrine comorbidity; Children

Background: The presence of endocrine disorders in childhood among children with bronchial asthma poses a serious medical and social problem. This, in turn, requires improvements in specialized medical care at various stages of treatment, taking into account not only clinical but also medical and social characteristics of the children.

Aim: To investigate the prevalence of endocrine disorders in children with bronchial asthma.

Materials and Methods: The study included 983 children aged 1 to 18 years with bronchial asthma (BA). Data collection involved extracting information from medical records and outpatient charts, as well as administering questionnaires to parents. The main inclusion criteria were a confirmed diagnosis of bronchial asthma in the child, age under 18 years, and informed consent from the parents. Comorbid conditions other than bronchial asthma included allergic rhinitis and grade 1–2 obesity. Data on concomitant comorbid conditions were analyzed.

Results: Comorbid conditions other than bronchial asthma included allergic rhinitis; endocrine disorders were represented by grade 1–2 obesity. A history of obesity was noted in 30 children with asthma. No endocrine disorders were found in children with mild asthma. Obesity was detected in 8 (2.1 ± 0.7%) cases among children with moderate asthma, in 12 (5.9 ± 1.7%) cases among children with severe asthma, and in 10 (2.5 ± 0.8%) cases among children with moderate asthma and concomitant allergic rhinitis ($p < 0.05$). Overall, endocrine comorbidities were significantly more common in children with severe asthma compared to those with mild or moderate asthma ($p < 0.05$). This is due to the predominance of older children in the group with severe asthma, who are more likely to have comorbid conditions.

Conclusion: Identifying concomitant endocrine disorders in children with bronchial asthma is important for a comprehensive assessment of patients' health. Timely diagnosis of endocrine disorders can facilitate the selection of individualized monitoring and treatment strategies. As a risk factor, obesity contributes to the development of bronchial asthma; moreover, it has a direct impact on the severity of the disease and its management. The information obtained can serve as a basis for developing medical and organizational measures to improve specialized medical care for bronchial asthma in children.

BÖLÜM 3 · SECTION 3

Tiroid Hastalıkları

Thyroid Disorders

3 bildiri · 3 abstracts

BİLDİRİ № 13 · ABSTRACT 13 DOI: 10.5281/zenodo.23659756

An Overlooked Finding in Children Presenting with Elevated TSH: Iodine Deficiency

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Keywords: Elevated TSH; Urinary iodine; Iodine deficiency; Thyroid function tests

Objective

Although the iodization of salt has largely eliminated severe iodine deficiency, mild iodine deficiency can still occur in children. However, it remains unclear whether mild iodine deficiency affects thyroid function and how well a single spot urine sample captures this relationship. In this study, we aimed to evaluate the relationship between urinary iodine level and thyroid function tests, body mass index, age, and sex in children referred to our clinic for elevated TSH. We also aimed to compare urinary iodine levels across clinical groups and to determine whether children with low iodine levels could be distinguished from other children based on clinical and laboratory findings.

Materials and Methods

We included children referred to the Pediatric Endocrinology Clinic of Ankara Etlik City Hospital between August 2023 and August 2026 for elevated TSH on thyroid function testing. Children younger than 3 years of age, because of the possibility of congenital hypothyroidism, and those with Down syndrome or central hypothyroidism, were excluded. Analyses were performed on a total of 990 children.

TSH, free T4, thyroid autoantibodies (anti-TPO and anti-Thyroglobulin), urinary iodine, and urinary creatinine were measured in the same laboratory in all children; free T3 was not routinely measured early in the study period and was therefore available in only 394 children (39.8%), with the relevant comparisons based on this subgroup. Children were classified into three mutually exclusive clinical groups based on antibody status and thyroid function test (TFT) results: euthyroid (antibody-negative, normal TFT), Hashimoto's thyroiditis (antibody-positive; TFT may be normal or abnormal), and subclinical hypothyroidism (antibody-negative, abnormal TFT). We categorized urinary iodine level as both the spot measurement and the creatinine-corrected value (µg iodine/g creatinine), using the same numerical cut-offs (<100, 100–299, ≥300); we have not separately validated these cut-offs for the ratio and used them solely for comparability. All analyses (Tables 1–6) were based on these classifications. Because urinary creatinine excretion is naturally lower in younger children, the creatinine-corrected iodine value may appear higher simply because of age, even without a true difference in iodine status; the relevant comparisons were therefore repeated taking the effect of age into account.

The Mann–Whitney U or Kruskal–Wallis test was used as appropriate for the distribution of the data and the number of groups compared. Categorical variables were compared with the chi-square test. To isolate the effect of age, the relevant comparisons were also repeated using linear regression (ANCOVA) models with age included as a covariate. We considered $p < 0.05$ statistically significant.

Results

Of the 990 children included, 42.0% (n=416) were euthyroid, 15.6% (n=154) had Hashimoto's

thyroiditis, and 42.4% (n=420) had subclinical hypothyroidism. Among children with Hashimoto's thyroiditis, 39% remained euthyroid, while the rest had abnormal thyroid function tests. Among 836 antibody-negative children, 352 (42.1%) had a spot urinary iodine level below 100 µg/L. Findings for all six comparisons are presented in Tables 1–6.

Table 1. Comparison of three groups according to urinary iodine level (N=990)

Variable	Iodine <100 (n=404)	Iodine 100–299 (n=532)	Iodine ≥300 (n=54)	p
Age (months), median [IQR]	129 [96–166]	127 [92–170]	119 [76–168]	0.510
TSH (µIU/mL), median [IQR]	4.87 [3.37–6.22]	4.71 [3.47–6.16]	4.87 [3.82–6.68]	0.282
fT4 (ng/dL), median [IQR]	1.23 [1.11–1.35]	1.23 [1.12–1.34]	1.26 [1.16–1.42]	0.123
fT3/T3, median [IQR]	4.16 [3.74–4.63]	4.16 [3.76–4.63]	4.31 [3.77–4.71]	0.906
BMI SDS, median [IQR]	0.43 [−0.66–1.35]	0.38 [−0.51–1.32]	0.40 [−0.62–1.16]	0.807
Female sex, n (%)	228 (56.4)	306 (57.5)	31 (57.4)	0.945

IQR: interquartile range (25th–75th percentile).

Table 2. Comparison of three groups according to urinary iodine/creatinine ratio (N=990)

Variable	Ratio <100 (n=447)	Ratio 100–299 (n=495)	Ratio ≥300 (n=48)	p (unadjusted)	p (age-adjusted)
Age (months), median [IQR]	154 [117–183]	110 [82–144]	68 [55–97]	<0.001	—
TSH, median [IQR]	4.59 [3.17–6.08]	4.90 [3.54–6.28]	5.85 [4.49–6.65]	0.668	0.946
fT4, median [IQR]	1.21 [1.10–1.33]	1.25 [1.14–1.35]	1.24 [1.16–1.40]	0.0046	0.339
fT3/T3, median [IQR]	3.92 [3.60–4.47]	4.33 [3.92–4.75]	4.35 [3.96–4.71]	<0.001	0.146
BMI SDS, median [IQR]	0.36 [−0.72–1.35]	0.42 [−0.46–1.33]	0.36 [−0.35–0.96]	0.595	—
Female sex, n (%)	264 (59.1)	274 (55.4)	27 (56.3)	0.514	—

Table 3. Comparison of the euthyroid, Hashimoto's thyroiditis, and subclinical hypothyroidism groups (N=990)

Variable	Euthyroid (n=416)	Hashimoto's (n=154)	Subclinical (n=420)	p (unadjusted)	p (age-adjusted)
Spot iodine (µg/L), median [IQR]	119.0 [74.0–182.0]	131.0 [78.2–190.5]	111.0 [65.7–169.0]	0.0944	—
Iodine/creatinine, median [IQR]	101.4 [67.5–153.9]	104.5 [74.8–158.7]	112.4 [77.2–176.6]	0.0108	0.173

Table 4. Comparison of low and non-low urinary iodine levels in antibody-negative children (n=836)

Variable	Iodine <100 (n=352)	Iodine ≥100 (n=484)	p
Age (months), median [IQR]	128 [96–164]	127 [92–169]	0.900
TSH, median [IQR]	4.89 [3.49–6.20]	4.61 [3.43–6.04]	0.404
fT4, median [IQR]	1.24 [1.13–1.35]	1.24 [1.13–1.35]	0.556
BMI SDS, median [IQR]	0.45 [−0.64–1.35]	0.41 [−0.51–1.34]	0.734
Female sex, n (%)	188 (53.4)	268 (55.4)	0.622

Table 5. Comparison of euthyroid and TFT-dysfunction subgroups among children with Hashimoto's thyroiditis (n=154)

Variable	Euthyroid Hashimoto's (n=60)	Hashimoto's + TFT dysfunction (n=94)	p
Spot iodine (µg/L), median [IQR]	114.0 [60.0–185.2]	133.0 [92.8–193.5]	0.155
Iodine/creatinine, median [IQR]	99.7 [71.7–143.9]	113.0 [76.2–171.2]	0.310
Iodine <100, n (%)	25 (41.7)	27 (28.7)	0.245

Table 6. Other findings

Comparison	Result
TSH–BMI SDS correlation (n=990)	rho=0.099; p=0.0017
Antibody-positive / negative — TSH (n=990)	5.05 [3.57–7.13] / 4.71 [3.44–6.12]; p=0.0075
Antibody-positive / negative — fT4 (n=989)	1.21 [1.08–1.32] / 1.24 [1.13–1.35]; p=0.0026
Antibody-positive / negative — female sex (n=990)	70.8% / 54.5%; p=0.0003

rho: Spearman's correlation coefficient

A weak but significant positive correlation was found between TSH and BMI SDS. Antibody-positive children had higher TSH, lower fT4, and a higher proportion of female sex.

Conclusion

We found no clear relationship between spot urinary iodine level and thyroid function tests. Similarly, children with low iodine levels did not differ significantly from other children in terms of age, sex, BMI SDS, or thyroid function tests. The differences observed in analyses based on the urinary iodine/creatinine ratio disappeared once we accounted for age, indicating that this ratio can be misleading in children if age is not considered. By evaluating both the spot urinary iodine level and the iodine/creatinine ratio, and by separately examining the effect of age, our study showed that the differences we observed likely reflected age rather than a true effect of iodine status.

Notably, however, among 420 children with abnormal thyroid function tests despite negative thyroid autoantibodies, 186 (44.3%) had a urinary iodine level below 100 µg/L. The limitations of a single spot urine sample in reliably reflecting individual iodine status should nonetheless be kept in mind. Although our study did not show a relationship between low urinary iodine and thyroid dysfunction, these findings suggest that assessing iodine status may be a reasonable part of the clinical work-up in antibody-negative children with elevated TSH.

BİLDİRİ № 14 · ABSTRACT 14 DOI: 10.5281/zenodo.23659758

Clinical Characteristics and Long-Term Outcomes of Pediatric Graves' Disease: A Single-Center Experience

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Keywords: Graves' disease; Hyperthyroidism; Methimazole; Children

Background: Graves' disease is the most common cause of hyperthyroidism in children and adolescents and is an autoimmune disorder mediated by thyroid-stimulating hormone receptor antibodies (TRAb). Antithyroid drugs (ATDs), particularly methimazole, are generally used as first-line treatment. Reported remission rates with ATD therapy are lower in children than in adults, at approximately 40%, with frequent relapse and long-term remission rates of 20–25%. This study aimed to evaluate the clinical, biochemical, ultrasonographic, and treatment characteristics of pediatric patients with Graves' disease and to identify factors associated with recurrent hyperthyroidism.

Methods: This single-center, retrospective observational study included 33 pediatric patients with Graves' disease followed at a tertiary referral center. Demographic characteristics, presenting symptoms, physical examination findings, anthropometric measurements, thyroid function tests, thyroid autoantibody levels, and thyroid ultrasonography findings were reviewed, together with treatment regimens and doses. Time to euthyroidism and subsequent episodes of hyperthyroidism were assessed during follow-up. Clinical, biochemical, and ultrasonographic findings were compared between patients with and without recurrent hyperthyroidism. Continuous variables are presented as medians (minimum–maximum) or means ± standard deviations, as appropriate. The Mann–Whitney U test was used to compare continuous variables between groups, with $p < 0.05$ considered statistically significant.

Results: Of the 33 patients, 25 (75.8%) were female. The median age at diagnosis was 12.8 years (2.3–17.5). Most patients (90.9%) were symptomatic at presentation, with palpitations being the most common symptom (70%). Goiter and tachycardia were each observed in 75.8% of patients. At diagnosis, median thyroid-stimulating hormone (TSH), free thyroxine (fT4), and free triiodothyronine (fT3) levels were 0.005 (0.001–0.3) mIU/L, 2.9 (1.8–7.7) ng/dL, and 8.1 (3.6–27.0) pg/mL, respectively. The median follow-up duration was 3.4 years (1.2–8.4). All patients received methimazole. The mean initial dose was higher in patients with recurrent hyperthyroidism than in those without recurrence (0.56 ± 0.19 vs. 0.43 ± 0.20 mg/kg/day; $p = 0.039$). Propranolol was added in 81.8% of patients at a mean dose of 0.72 ± 0.30 mg/kg/day. The median time to euthyroidism was 5.5 months (3–24). No patient achieved remission, and recurrent episodes of hyperthyroidism occurred in 22 patients (66.7%) after euthyroidism had been attained. Patients with recurrent hyperthyroidism had significantly higher baseline fT4 and fT3 levels ($p = 0.040$ and $p = 0.030$, respectively; Table 1). Baseline TRAb levels were numerically higher in this group, but the difference was not statistically significant ($p = 0.636$). Thyroid ultrasonography showed increased total thyroid volume in all patients, with no significant difference between groups. Thyroid nodules were identified in five patients, two of whom had biopsy-confirmed papillary thyroid carcinoma. Six patients underwent thyroidectomy: four for treatment-resistant disease and two for thyroid carcinoma.

Conclusions: Pediatric Graves' disease is usually symptomatic at presentation. Although ATD therapy can restore euthyroidism, recurrent hyperthyroidism remains common during follow-up. Higher baseline fT4 and fT3 levels may be associated with recurrent hyperthyroidism. These findings underscore the importance of long-term follow-up and individualized monitoring strategies.

Table 1. Comparison of patients with and without recurrent hyperthyroidism

Characteristic	No recurrent hyperthyroidism (n = 11)	Recurrent hyperthyroidism (n = 22)	p value
Age at diagnosis, years	14 (5.5–16.5)	12.3 (2.3–17.5)	0.588
Female sex, n (%)	9 (81.8)	16 (72.7)	0.454
Height SDS	0.8 (−1.3–2.5)	0.14 (−1.26–3.1)	0.267
Weight SDS	−0.2 (−2.1–1.3)	0.5 (−1.5–3.5)	0.248
BMI SDS	0.5 (−3.7–2.7)	0.5 (−1.6–3.0)	0.475
TSH at diagnosis, mIU/L	0.005 (0.001–0.3)	0.005 (0.005–0.2)	0.381
fT4 at diagnosis, ng/dL	2.3 (1.9–4.0)	3.0 (1.8–7.7)	0.040
fT3 at diagnosis, pg/mL	6.8 (3.6–15.0)	8.9 (4.2–27.0)	0.030
TRAb at diagnosis, IU/L	2.7 (1.7–13.5)	3.9 (1.8–40.0)	0.636
Anti-TPO at diagnosis, IU/mL	144.5 (40–607)	163 (11–600)	0.746
Anti-Tg at diagnosis, IU/mL	95 (19.7–2357)	139 (10–4000)	0.681
Thyroid volume SDS	7.2 (0.9–29.0)	5.4 (1.8–31.5)	0.660
Initial methimazole dose, mg/kg/day	0.43 ± 0.20	0.56 ± 0.19	0.039
Time to euthyroidism, months	4.5 (3–9)	6 (3–24)	0.286

Values are presented as median (minimum–maximum), n (%), or mean ± SD, as appropriate.

Abbreviations: SDS, standard deviation score; BMI, body mass index; TSH, thyroid-stimulating hormone; fT4, free thyroxine; fT3, free triiodothyronine; TRAb, TSH receptor antibodies; anti-TPO, thyroid peroxidase antibodies; anti-Tg, thyroglobulin antibodies.

BİLDİRİ № 15 · ABSTRACT 15 DOI: 10.5281/zenodo.23659760

Evaluation of Fatty Liver in Adolescents with Hashimoto's Thyroiditis Using Ultrasound Attenuation Scores: A Prospective Study

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Keywords: Hashimoto’s thyroiditis; Fatty liver; Ultrasound attenuation; Adolescents

Aim; This study aims to evaluate the association between Metabolically associated steatotic liver disease (MASLD) and demographic characteristics, thyroid function tests, autoantibodies, metabolic parameters, and the Mean USAT (Ultrasound Attenuation Analysis) score, calculated as the average of three separate ultrasonographic measurements, in patients diagnosed with thyroiditis.

Material and Methods: A total of 95 patients (82 females, 13 males) with thyroiditis were analyzed. Based on ultrasonographic findings, patients were categorized into two groups: Without Fatty Liver (n=77) and With Fatty Liver (n=18). The "Mean USAT" variable was created by averaging the USAT1, USAT2, and USAT3 scores.

Results: The Mean USAT score was significantly higher in the fatty liver present group compared to the absent group (0.70 ± 0.10 vs. 0.51 ± 0.06, p<0.0001). Regarding thyroid parameters, free T4 (fT4) levels were significantly lower in patients with fatty liver (1.17 ± 0.26 ng/dL vs. 1.32 ± 0.27 ng/dL, p=0.036). However, no statistically significant differences were observed between the groups in terms of TSH (p=0.7779), fT3 (p=0.3121), Anti-TPO (p=0.7117), Anti-Tg (p=0.8687), or gender distribution (p=0.4507). Furthermore, a significant negative correlation was found between Mean USAT scores and fT4 levels (r=-0.295, p=0.0037). A statistically significant and positive (direct) correlation was found between the fT3 / fT4 ratio and the mean USAT score (P=0.0308) (r= +0.222).

Conclusion: In patients with thyroiditis, the presence of fatty liver is strongly associated with increased metabolic risk, insulin resistance, and higher USAT scores. Additionally, fT4 levels may represent an independent risk factor for the development of MASLD and elevated USAT values, whereas the severity of thyroid autoimmunity does not appear to have a direct impact on fatty liver status.

BÖLÜM 4 · SECTION 4

Hipofiz ve Büyüme Bozuklukları

Pituitary and Growth Disorders

2 bildiri · 2 abstracts

BİLDİRİ № 16 · ABSTRACT 16 DOI: 10.5281/zenodo.23659762

Clinical and Laboratory Characteristics of Children with Growth Hormone Deficiency in the Republic of Uzbekistan

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Keywords: Growth hormone deficiency; Short stature; IGF-1; Uzbekistan

Background and Aims

This study analyzed the clinical and hormonal characteristics of growth hormone deficiency in children presenting with short stature and delayed physical development.

Methods

The study included 51 patients (26 boys, 25 girls) aged 5.1 to 16.0 years with a confirmed diagnosis of growth hormone deficiency (GHD), who underwent examination and treatment at the Republican Specialized Scientific and Practical Medical Center of Endocrinology named after Academician Y. Kh. Turakulov during 2020–2025. The diagnosis was established based on a characteristic clinical presentation (growth retardation, height SDS below −2), results of growth hormone stimulation tests (standardized insulin or clonidine stimulation tests), serum IGF-1 levels, MRI findings of the hypothalamic–pituitary region, and hand radiography for determination of bone age. Anthropometric parameters were assessed by calculating standard deviation scores (SDS) for height and body weight according to WHO standards (WHO AnthroPlus, 2007). Statistical analysis was performed using descriptive statistical methods (M±SD, Me, min–max, percentages).

Results

Among the examined children, boys accounted for 51.0% (n=26), and girls for 49.0% (n=25). The mean age of the patients was 10.9±3.1 years (range: 5.1–16.0 years). The most frequent age at presentation was 9–13 years, which likely reflects delayed referral and diagnosis. The mean height at presentation was 120.5±14.6 cm with a height SDS of −3.4±1.2. In all patients, height SDS did not exceed −1.2, and in 62.7% of children it was below −3.0, indicating severe linear growth retardation. The mean body weight was 23.1±7.8 kg with a weight SDS of −3.8±2.3. A growth hormone (GH) stimulation test with assessment of five time points of GH secretion was performed in 40 of 51 patients. The mean peak GH level was 4.8±4.3 ng/mL. In 37 patients (72.5%), the maximal stimulated GH level did not reach the diagnostic threshold of 10 ng/mL; in 32 patients (62.7%), it did not exceed 7 ng/mL, consistent with criteria for severe growth hormone deficiency according to ISPAD and the Growth Hormone Research Society (2019). IGF-1 data were available for 32 patients. The mean IGF-1 level was 99.4±88.3 ng/mL (range: 9.9–352.0 ng/mL), with a median of 82.8 ng/mL. In the majority of patients, IGF-1 levels were reduced compared to age-adjusted reference ranges, which is consistent with decreased GH secretion and correlates with the severity of clinical manifestations.

Conclusions

The clinical analysis of 51 patients with growth hormone deficiency (GHD) demonstrated a characteristic profile: a marked reduction in height SDS (−3.4±1.2), a decreased peak GH response in the stimulation test (mean 4.8±4.3 ng/mL), and low IGF-1 levels (median 82.8 ng/mL), with

severe GHD confirmed in 72.5% of patients based on a peak GH level <10 ng/mL. A diagnostic delay of 3–4 years highlights the need for active screening for growth retardation at the primary healthcare level, as early diagnosis and timely initiation of recombinant growth hormone therapy improve growth outcomes and quality of life.

BİLDİRİ № 17 · ABSTRACT 17 DOI: 10.5281/zenodo.23659764

Evaluation of Clinical, Hormonal and Treatment Characteristics in Pediatric Prolactinoma Cases: A Single-Center Experience

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Keywords: Pediatric prolactinoma; Microadenoma; Macroadenoma; Cabergoline

Objective

Prolactinomas are rare in childhood and adolescence; however, they represent the most common pituitary adenomas in this age group. Clinical presentation, pituitary function and treatment requirements may differ according to tumor size. This study aimed to evaluate the clinical, hormonal and treatment characteristics of pediatric patients with prolactinoma and to compare patients with microadenomas and macroadenomas.

Methods

The medical records of 21 patients followed up for prolactinoma at a tertiary pediatric endocrinology center were retrospectively reviewed. Based on pituitary magnetic resonance imaging findings, patients were classified as having a microadenoma (<10 mm; n=12) or a macroadenoma (≥10 mm; n=9). Demographic characteristics, auxological parameters (body weight and height standard deviation scores [SDS]), hormonal parameters (IGF-1, TSH, free T4 [fT4], ACTH, cortisol, LH, FSH, estradiol and prolactin) and treatment characteristics were analyzed. Continuous variables were compared using Student's t-test or the Mann–Whitney U test, as appropriate, and categorical variables using Fisher's exact test. A p-value <0.05 was considered statistically significant.

Results

Twenty of the 21 patients (95.2%) were female. The mean age was 15.78 ± 1.28 years, the mean body weight SDS was 0.52 ± 2.11 and the mean height SDS was −0.34 ± 0.60. No significant differences were found between the microadenoma and macroadenoma groups in IGF-1, TSH, fT4, ACTH, LH, FSH, estradiol or prolactin levels (p>0.05). Cortisol levels were significantly lower in the macroadenoma group than in the microadenoma group (8.30 ± 4.15 vs. 12.14 ± 3.81 µg/dL; p=0.045). Two patients (9.5%) had undergone surgery, both of whom had macroadenomas (p=0.17). Levothyroxine and hydrocortisone replacement were each required in three patients (14.3%), all in the macroadenoma group (p=0.06). Cabergoline treatment duration, weekly dose and weight-adjusted dose did not differ significantly between the groups (p>0.05), and the time to prolactin normalization was similar. In patients with macroadenomas, a mean reduction in tumor diameter of 26.5 ± 11.5% was achieved within the first year of cabergoline treatment.

Conclusion

Pediatric prolactinoma showed a marked female predominance in this cohort. Cabergoline appeared effective in both groups, with comparable times to prolactin normalization and a substantial reduction in tumor size in macroadenomas. Although most hormonal parameters did not differ according to tumor size, lower cortisol levels in the macroadenoma group may suggest greater involvement of the corticotroph axis in larger tumors. This finding warrants confirmation

with dynamic testing in larger, multicenter cohorts.

Table 1. Comparison of Hormonal Parameters Between the Microadenoma and Macroadenoma Groups

Variable	Group		Total	p
	Microadenoma (n=12)	Macroadenoma (n=9)		
IGF-1 (ng/mL), mean (SD)	209.17 (60.45)	247.5 (102.1)	224.5 (76.79)	0.472†
TSH (µIU/mL), mean (SD)	2.11 (0.69)	1.89 (0.89)	1.86 (0.58)	0.523†
fT4 (ng/dL), mean (SD)	1.18 (0.18)	1.02 (0.23)	1.08 (0.25)	0.089†
ACTH (pg/mL), median (IQR)	20.9 (7.8)	21.7 (14.7)	21.5 (10.45)	0.970‡
Cortisol (µg/dL), mean (SD)	12.14 (3.81)	8.3 (4.15)	11 (4.15)	**0.045†**
LH (mIU/mL), median (IQR)	4.89 (4.85)	3.31 (12.16)	6.43 (10.99)	0.422‡
FSH (mIU/mL), mean (SD)	4.69 (2.17)	5.32 (2.17)	5.22 (2.58)	0.519†
E2 (pg/mL), median (IQR)	48 (65.1)	22.3 (39)	41 (40.75)	0.086‡
Prolactin (ng/mL), median (IQR)	132 (69.5)	124 (231.5)	109.85 (162)	0.678‡

†Student's t-test ‡ Mann-Whitney U test

Table 2. Comparison of Clinical Characteristics

Variable	Group n (%)		Total	p†
	Microadenoma	Macroadenoma		
Surgical History				
No	12 (100)	7 (77.8)	19 (90.5)	0.17
Yes	0 (0)	2 (22.2)	2 (9.5)	
Levothyroxine Use				
No	12 (100)	6 (66.7)	18 (85.7)	0.06
Yes	0 (0)	3 (33.3)	3 (14.3)	
Hydrocortisone Use				
No	12 (100)	6 (66.7)	18 (85.7)	0.06
Yes	0 (0)	3 (33.3)	3 (14.3)	

† Fisher's exact test

Table 3. Parameters Related to Cabergoline Treatment

Variable	Group				Total	p
	Microadenoma		Macroadenoma			
	n	Value	n	Value		
Duration of Cabergoline Use (Months)	10	2.8 (1.6)	4	4 (2.16)	3.14 (1.78)	0.271†
Cabergoline Dose (mg/week)	12	0.75 (0.5)	9	1 (0.25)	1 (0.5)	0.31‡
Cabergoline Dose (mg/kg)	12	0.010 (0.009)	9	0.014 (0.006)	0.01 (0.01)	0.241‡
Tumor Size Reduction Within 1 Year (%)	-	-	8	26.5 (11.5)	26.5 (11.5)	-
Time to Prolactin Normalization (Months)	12	2 (4.13)	9	4 (2.25)	2 (2.5)	0.374‡

† Variables presented as mean ± SD were analyzed using Student's t-test, and ‡ variables presented as median (IQR) were analyzed using the Mann-Whitney U test.

BÖLÜM 5 · SECTION 5

Puberte Bozuklukları

Disorders of Puberty

4 bildiri · 4 abstracts

BİLDİRİ № 18 · ABSTRACT 18 DOI: 10.5281/zenodo.23659768

Height SDS 'Catch-Down' During GnRH Analogue Treatment in Central Precocious Puberty and Achievement of Target Height

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Keywords: Central precocious puberty; GnRH analogue; Final height; Target height

Introduction / Aim

In the treatment of central precocious puberty (CPP), GnRH analogues (GnRHa) aim to preserve genetic height potential by slowing accelerated skeletal maturation. However, suppression of the hypothalamic–pituitary–gonadal axis is accompanied by a decline in growth velocity and a physiological regression in height percentile ("catch-down"). The aim of this study was to examine annual height SDS dynamics in girls receiving GnRHa treatment and to evaluate the effect of this on-treatment percentile loss on attainment of genetic target height (TH SDS) and final adult height.

Methods

Data from 137 girls who received depot GnRHa (leuprolide acetate) treatment at our clinic and reached final height were retrospectively reviewed. Annual height SDS changes were assessed in 88 patients followed uninterruptedly for at least two years. Baseline and final data were compared using the paired samples t-test; non-normally distributed first- and second-year SDS changes were analysed with the Wilcoxon signed-rank test; and the effect of height deceleration on final height was examined by Spearman correlation analysis.

Results

Mean age at diagnosis was 7.99±0.97 years (median: 8.08), weight SDS was +1.05±1.13 and height SDS was +0.80±1.17. Mean treatment duration was 32.4±11.9 months (median: 32.0 months); in patients whose treatment was completed at 11.0 years of age, final height was assessed a mean of 5.93±2.22 years after treatment discontinuation (median: 5.0 years; mean current age: 16.93±2.22 years). In the whole cohort (n=137), final height SDS showed a significant net height gain relative to predicted adult height at diagnosis (+0.71±1.30 SDS, $p<0.001$) and exceeded target height (+0.30±1.12 SDS, $p=0.004$). In the height-velocity follow-up group (n=88), height SDS percentile was maintained in the first year (+0.12±0.42 SDS), whereas a marked physiological loss of height SDS ("catch-down") was observed in the second year, when the axis was fully suppressed (−0.12±0.43 SDS, median: −0.15; Wilcoxon $Z=-4.258$, $p<0.001$). Spearman analysis showed no significant correlation between this second-year loss of height SDS and either net height gain ($r_s=0.038$, $p=0.723$) or the difference from target height ($r_s=0.055$, $p=0.620$).

Conclusion

In girls with CPP receiving GnRHa treatment, physiological "catch-down" becomes evident mainly in the second year, when the axis is fully suppressed, rather than in the first year. Long-term follow-up of approximately six years after treatment discontinuation demonstrated that this percentile regression does not reduce net height gain or success in attaining genetic target height. Growth deceleration observed in the second year is an expected, natural indicator of axis

suppression and should not be mistaken for treatment failure leading to premature discontinuation.

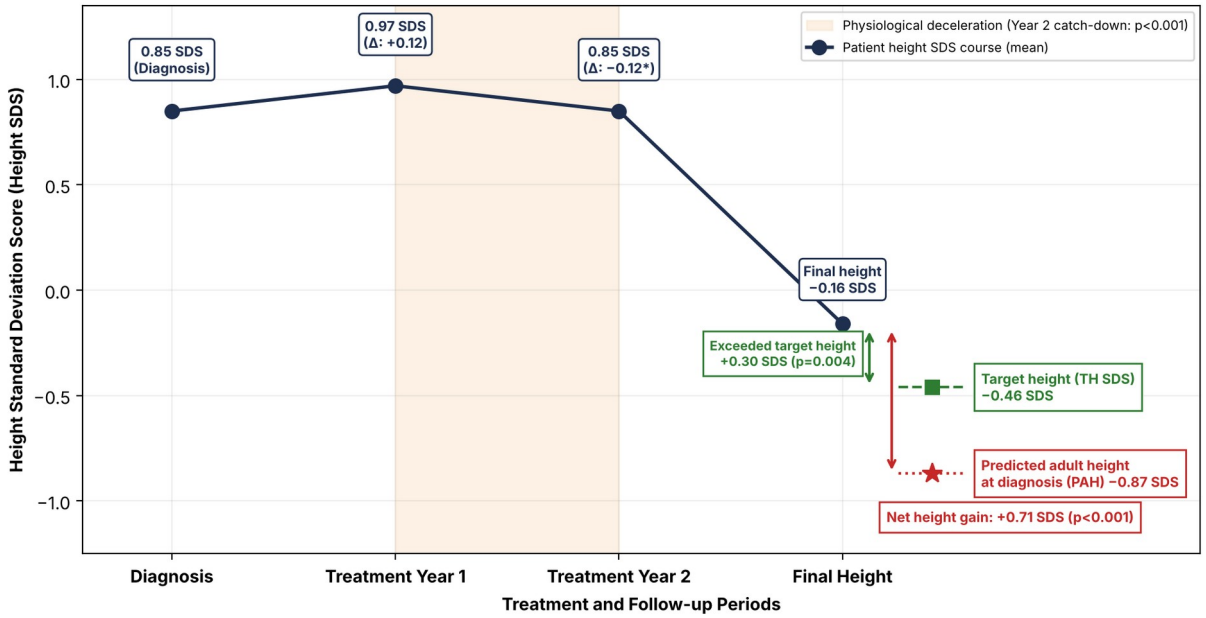

Figure 1. Course of mean height SDS from diagnosis to final height in girls with CPP treated with GnRHa. The shaded area indicates physiological deceleration in the second treatment year (catch-down, p<0.001). PAH, predicted adult height at diagnosis; TH, target height.

BİLDİRİ № 19 · ABSTRACT 19 DOI: 10.5281/zenodo.23659770

Clinical, Laboratory, and Cranial MRI Characteristics and Evaluation of Post-Treatment BMI and BMI SDS in Girls Diagnosed with Central Precocious Puberty: A Single-Center Experience

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Keywords: Central precocious puberty; GnRH analog; Body mass index; Cranial MRI

Objective

Previous studies have reported inconsistent findings regarding the effects of gonadotropin-releasing hormone (GnRH) analog therapy on body mass index (BMI) and BMI standard deviation score (BMI SDS).

This study aimed to evaluate the baseline clinical, laboratory, and cranial magnetic resonance imaging (MRI) characteristics of girls with central precocious puberty who initiated treatment from 2024 onward and to assess changes in BMI SDS from baseline to 6 and 12 months following the initiation of GnRH analog therapy.

Methods

A total of 91 girls diagnosed with central precocious puberty at the Pediatric Endocrinology Clinic and initiated on monthly GnRH analog therapy were included in the study. Treatment response at 6 and 12 months was assessed based on anthropometric measurements, BMI SDS, Tanner stage, bone age, and hormonal parameters.

Results

A total of 91 girls with central precocious puberty who received GnRH analog therapy were evaluated. According to baseline BMI SDS, 59 patients (64.8%) were classified as having normal weight, 21 (23.1%) as overweight, 10 (11.0%) as obese, and 1 (1.1%) as underweight (Table 1).

At 6 months of treatment, mean BMI SDS increased from 0.67 ± 1.03 to 0.73 ± 1.00; however, this change was not statistically significant ($p = 0.535$) (Table 2). At 12 months, mean BMI SDS increased from 0.55 ± 1.03 to 0.68 ± 0.99, corresponding to a statistically significant mean change of +0.13 SDS ($p = 0.032$) (Table 3).

Among girls who were classified as normal weight or underweight at baseline, the mean 12-month change in BMI SDS was approximately +0.23 SDS, whereas a mean change of approximately −0.06 SDS was observed among those who were overweight or obese at baseline. The difference in BMI SDS change between these groups was statistically significant ($p \approx 0.04$).

Conclusion

Monthly GnRH analog therapy resulted in effective clinical suppression of pubertal progression in the majority of patients at both 6 and 12 months. Although no statistically significant change in BMI SDS was observed at 6 months but statistically significant increase was detected at 12 months. Notably, the increase in BMI SDS was more pronounced among girls who were normal weight or underweight at baseline, whereas no corresponding increase was observed among those who were overweight or obese.

These findings suggest that changes in BMI during GnRH analog therapy may vary according to baseline weight status. Therefore, longitudinal monitoring of BMI SDS during treatment may be

particularly relevant in girls who are of normal weight at treatment initiation.

Table 1. Baseline clinical and anthropometric characteristics of the patients

Variable	Baseline value
Number of patients, n	91
Age at treatment initiation, years	7.39 ± 0.69
Weight SDS	0.77 ± 1.16
Height SDS	0.60 ± 1.19
BMI SDS	0.63 ± 1.02
Bone age, years	8.52 ± 1.43
Bone age–chronological age difference	≈ +1.1 years
Tanner stage 1, n (%)	2 (2.2%)
Tanner stage 2, n (%)	54 (59.3%)
Tanner stage 3, n (%)	25 (27.5%)
Tanner stage 4, n (%)	10 (11.0%)
Family history of early puberty, n (%)	5 (5.5%)
Patients evaluated by pelvic ultrasonography, n	89
Uterine length, mm	33.83 ± 7.58
Underweight, n (%)	1 (1.1%)
Normal weight, n (%)	59 (64.8%)
Overweight, n (%)	21 (23.1%)
Obese, n (%)	10 (11.0%)
Overweight + obese, n (%)	31 (34.1%)

Data are presented as mean ± SD or n (%).

Table 2. Clinical and anthropometric findings at baseline and month 6

Variable	Baseline	Month 6	p value
Number of patients evaluated, n	70	70	—
Body weight, kg	29.24 ± 7.55	31.56 ± 8.14	<0.001
Weight SDS	0.87 ± 1.17	0.95 ± 1.12	0.058
Height, cm	127.48 ± 8.12	130.89 ± 8.38	<0.001
Height SDS	0.75 ± 1.19*	0.86 ± 1.18*	0.001
BMI, kg/m²	17.73 ± 2.93	18.14 ± 2.97	0.015
BMI SDS	0.67 ± 1.03	0.73 ± 1.00	0.535
Tanner stage 1, n (%)	2 (2.9%)	21 (30.0%)	—
Tanner stage 2, n (%)	40 (57.1%)	19 (27.1%)	—
Tanner stage 3, n (%)	19 (27.1%)	24 (34.3%)	—
Tanner stage 4, n (%)	9 (12.9%)	6 (8.6%)	—
Regression in Tanner stage, n (%)	—	27 (38.6%)	—
Stable Tanner stage, n (%)	—	34 (48.6%)	—
Progression in Tanner stage, n (%)	—	9 (12.9%)	—
Clinical pubertal suppression, n (%)	—	61 (87.1%)	—

* The number of evaluable patients for height SDS was n = 69.

Clinical pubertal suppression was defined as regression or stabilization of Tanner stage.

Table 3. Clinical, anthropometric, bone age, and laboratory findings at baseline and month 12

Variable	Baseline	Month 12	p value
Number of patients evaluated, n	56	56	—
Body weight, kg	27.68 ± 6.91	32.73 ± 8.05	<0.001
Weight SDS	0.65 ± 1.18	0.83 ± 1.10	<0.001
Height, cm	125.53 ± 7.90	132.94 ± 7.70	<0.001
Height SDS	0.49 ± 1.18	0.69 ± 1.13	<0.001
BMI, kg/m²	17.32 ± 2.76	18.28 ± 3.02	<0.001
BMI SDS	0.55 ± 1.03	0.68 ± 0.99	0.032
Bone age, years	8.33 ± 1.25	9.63 ± 1.22	<0.001
FSH, IU/L	2.74 ± 1.43*	1.54 ± 1.05*	<0.001
LH, IU/L	0.56 ± 0.45†	0.73 ± 0.99†	0.909
Estradiol	14.07 ± 12.87†	9.70 ± 8.38†	0.075
Tanner stage 1, n (%)	2 (3.6%)	21 (37.5%)	—
Tanner stage 2, n (%)	39 (69.6%)	18 (32.1%)	—
Tanner stage 3, n (%)	11 (19.6%)	13 (23.2%)	—
Tanner stage 4, n (%)	4 (7.1%)	4 (7.1%)	—
Regression in Tanner stage, n (%)	—	25 (44.6%)	—
Stable Tanner stage, n (%)	—	23 (41.1%)	—
Progression in Tanner stage, n (%)	—	8 (14.3%)	—
Clinical pubertal suppression, n (%)	—	48 (85.7%)	—

* The number of patients included in the FSH comparison was n = 44.

† The number of patients included in the LH and estradiol comparisons was n = 54.

Clinical pubertal suppression was defined as regression or stabilization of Tanner stage.

BİLDİRİ № 20 · ABSTRACT 20

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Inhibin A and Inhibin B in Predicting the Pubertal LH Response in Girls

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Keywords: Inhibin B; Inhibin A; LHRH stimulation test; Puberty

Objectives: To evaluate basal and stimulated serum inhibin A and inhibin B for predicting the pubertal luteinizing hormone (LH) response during the luteinizing hormone-releasing hormone (LHRH) stimulation test in girls.

Methods: In this prospective study, 74 girls with Tanner stage 2 breast development were analyzed after approximately 6 months of clinical follow-up. LH, follicle-stimulating hormone (FSH), inhibin A, and inhibin B were measured at 0, 30, and 60 minutes. Pubertal response was defined as peak LH ≥5 IU/L (Group A) and, in a sensitivity analysis, peak LH ≥5 IU/L plus peak LH/peak FSH ≥0.66 (Group B). Diagnostic performance was assessed by receiver operating characteristic (ROC) analysis.

Results: Pubertal response was present in 35 girls (47.3%) in Group A and 20 (27.0%) in Group B. In Group A, basal inhibin B was higher in positive than negative cases [51.0 (30.1–82.5) vs 21.8 (9.9–32.5) ng/L; p<0.0001]. Basal and peak inhibin B showed similar performance (area under the ROC curve [AUC] 0.775 vs 0.777; paired DeLong p=0.925), with no significant basal-versus-peak difference in Group B (p=0.371). Basal inhibin B

outperformed basal inhibin A in Group A (ΔAUC=0.129; p=0.028) but not basal LH (p=0.792).

Conclusions: Inhibin B showed moderate ability to predict the pubertal LH response. Similar basal and peak performance suggests that a single basal measurement may be clinically practical, although its added value over basal LH and the exploratory thresholds require external validation.

BİLDİRİ № 21 · ABSTRACT 21 DOI: 10.5281/zenodo.23659774

IGF-1 and IGFBP-3 Cut-off Values for Predicting Pubertal Activation in 6–8-Year-Old Girls with Tanner Stage 2 Breast Development and Their Relationship with Gonadotropins, Estradiol and DHEA-S: A Retrospective Evaluation

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Keywords: IGF-1; IGFBP-3; Early puberty; DHEA-S

Objective: We aimed to determine IGF-1 and IGFBP-3 cut-off values predicting biochemical pubertal activation in 6–8-year-old girls with Tanner stage 2 breast development, and to examine the relationship of these two markers with basal gonadotropins, estradiol and DHEA-S.

Methods: Girls aged 6–8 years who presented to our outpatient clinic with breast enlargement (Tanner stage 2) between January 2024 and September 2026 were included. Basal values at each patient's first visit were used. Patients were evaluated retrospectively. Basal LH ≥ 0.3 IU/L and/or measurable estradiol (≥ 10 pg/mL) was defined as biochemical pubertal activation. A DHEA-S level of 40 µg/dL or above was considered consistent with adrenarche. Cut-off values for IGF-1 and IGFBP-3 were determined by ROC analysis and the Youden index. Age groups were compared with the Kruskal-Wallis test, and associations were assessed with Spearman correlation.

Results: All patients were girls; 61 were 6, 74 were 7 and 84 were 8 years old. Median IGF-1 increased from 109 ng/mL at age 6 to 132.5 ng/mL at age 8 ($\rho = 0.16$; $p = 0.017$). IGFBP-3 did not change with age ($p = 0.81$) but was moderately correlated with IGF-1 ($\rho = 0.46$; $p < 0.001$). Basal FSH increased with age ($p = 0.002$). Median DHEA-S was 46 µg/dL and did not differ by age. IGF-1 was positively correlated with estradiol ($\rho = 0.39$; $p < 0.001$), basal LH ($\rho = 0.29$; $p < 0.001$) and DHEA-S ($\rho = 0.25$; $p = 0.045$); these associations persisted after adjustment for age. IGFBP-3 was not associated with any of these hormones. **The optimal IGF-1 cut-off for predicting pubertal activation was 140 ng/mL (AUC 0.65; 95% CI 0.56–0.74; sensitivity 59%, specificity 74%, positive predictive value 71%). This cut-off performed better in predicting measurable estradiol (AUC 0.72; sensitivity 73%, specificity 71%, negative predictive value 86%).** IGFBP-3 failed to discriminate pubertal activation (AUC 0.47), and no clinically useful cut-off could be determined. The cut-off for the IGF-1/IGFBP-3 ratio was 47 (AUC 0.68; sensitivity 62%, specificity 69%).

Conclusion: In 6–8-year-old girls with Tanner stage 2 breast development, **an IGF-1 level of 140 ng/mL or above supports biochemical pubertal activation**; IGFBP-3 cannot be used for this purpose. IGF-1 rises with age, whereas IGFBP-3 does not change significantly. The association of IGF-1 with estradiol and basal LH suggests that early activation of the pubertal axis is reflected in the growth axis.

BÖLÜM 6 · SECTION 6

Adrenal Bez Hastalıkları

Adrenal Disorders

4 bildiri · 4 abstracts

BİLDİRİ № 22 · ABSTRACT 22 DOI: 10.5281/zenodo.23659776

Evaluation of Metabolic Health Indicators in Children Diagnosed with Congenital Adrenal Hyperplasia

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Keywords: Congenital adrenal hyperplasia; Metabolic health; Obesity; Glucocorticoid therapy

Introduction and Aim: Metabolic health problems may accompany congenital adrenal hyperplasia (CAH) and complicate disease management.Inappropriate glucocorticoid replacement dosing or uncontrolled hyperandrogenism may increase adipocytokine secretion and systemic inflammation, thereby adversely affecting metabolic health.This study aimed to evaluate metabolic health status in patients with classic and nonclassic CAH and to examine its relationship with disease control.

Methods: Patients followed with a diagnosis of classic CAH or nonclassic CAH (NCCAH) between January 2005 and January 2025 were included. Obesity, hypertension, dyslipidemia, and impaired glucose homeostasis were assessed as metabolic health indicators. Patients meeting at least two of these criteria were defined as having "impaired metabolic health."CAH-specific disease control was assessed using follow-up total testosterone levels in 46,XX patients and follow-up androstenedione levels in 46,XY patients.

Results:A total of 74 patients(46 classic CAH, 28 NCCAH; 52 46,XX and 22 46,XY) were included.Impaired metabolic health was present in 35.1% of patients (classic CAH 32.6%,NCCAH 39.3%; p=0.560). Impaired metabolic health developed at a mean age of 12.26±3.68 years, and 84% of cases occurred during puberty.Obesity was detected in 43.2% of patients. Age at diagnosis was higher in patients with impaired metabolic health(4.45±4.21 vs. 2.70±3.59 years; p=0.027).Total testosterone levels in 46,XX patients(101.89±191.61 vs. 24.31±22.29 ng/dL; p<0.001) and androstenedione levels in 46,XY patients(559.01±314.59 vs. 98.19±87.11 ng/dL; p=0.020) were higher in this group. Hydrocortisone dose did not differ between groups(14.22±3.84 vs. 14.26±3.59 mg/m²/day;p=0.930).Polycystic ovary syndrome was identified in 33.3% (11/33) of pubertal girls.

Conclusion: Significant metabolic health problems develop in both classic CAH and NCCAH patients. In our study, impaired metabolic health was associated with older age at diagnosis, obesity, pubertal stage, and hyperandrogenism. Treatment doses were not high, and no adverse effect of these doses on metabolic health was observed.The higher androgen levels in patients with impaired metabolic health underscore the importance of controlling hyperandrogenism in patients with CAH.

BİLDİRİ № 23 · ABSTRACT 23 DOI: 10.5281/zenodo.23659780

Examination and Treatment of a Patient Diagnosed with Adrenogenital Syndrome, Virilizing Form

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Keywords: Congenital adrenal hyperplasia; Adrenogenital syndrome; 17-OH progesterone; Hydrocortisone

Introduction: Congenital adrenal hyperplasia (CAH) is an autosomal recessive disorder resulting from enzyme deficiencies involved in cortisol synthesis, most commonly 21-hydroxylase deficiency. This leads to excessive production of adrenal androgens such as testosterone, progesterone, and 17-hydroxyprogesterone. Clinically, CAH manifests in three forms: salt-wasting, virilizing, and postpubertal. The virilizing form, though less life-threatening than the salt-wasting form, can significantly affect sexual development and psychosocial adjustment if not diagnosed and treated promptly. The aim of this report is to present the diagnostic evaluation and therapeutic management of a patient with the virilizing form of adrenogenital syndrome.

Case Presentation: A 4-year-old girl was brought to medical attention with aggressive behavior, interest in male-associated games and activities, increased body hair (hirsutism), and ambiguous external genitalia exhibiting both male and female characteristics. The evaluation included a complete blood count, hormonal assays (ACTH, 17-hydroxyprogesterone and free testosterone), left wrist X-ray for bone age, abdominal and pelvic ultrasound, pelvic magnetic resonance imaging (MRI) and cytogenetic analysis. Initial laboratory findings were: ACTH 85.5 pg/ml (normal range: 7.2–63.3 pg/ml); 17-OH progesterone 3955.61 ng/dl (normal range: 0.2–1.3 ng/dl); free testosterone 9043 pg/ml (normal range: 0–2.85 pg/ml). Bone age was advanced, corresponding to 10 years, with open growth plates. Pelvic MRI suggested female pseudohermaphroditism; abdominal ultrasound showed no significant pathological findings. Cytogenetic analysis confirmed a normal female karyotype (46,XX). The patient was diagnosed with the virilizing form of adrenogenital syndrome and hydrocortisone (Genkort®, 10 mg tablets) was started: ½ tablet at 07:00, ¼ tablet at 15:00 and ¼ tablet at 22:00. After six months of hydrocortisone therapy, hormonal parameters normalized (ACTH 18.9 pg/ml; 17-OH progesterone 1.03 ng/dl; free testosterone 1.71 pg/ml), allowing successful surgical correction.

Discussion: Early diagnosis and appropriate hormonal replacement therapy are crucial in managing the virilizing form of CAH. The hydrocortisone regimen effectively suppressed ACTH overproduction and normalized androgen levels, enabling corrective surgery. Post-treatment follow-up demonstrated sustained normalization of hormonal levels, with continued hydrocortisone therapy necessary for maintaining homeostasis. This case highlights the importance of a multidisciplinary approach involving pediatric endocrinologists, surgeons, radiologists, and geneticists in the management of CAH.

Conclusion: The combination of comprehensive diagnostic evaluation, appropriate hormonal therapy, and surgical correction resulted in successful management of the patient with the virilizing form of adrenogenital syndrome. Ongoing hormone replacement therapy remains essential to maintain endocrine balance and support normal growth and development.

BİLDİRİ № 24 · ABSTRACT 24

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3β-Hydroxysteroid Dehydrogenase Type 2 Deficiency: Clinical and Genetic Characterization and Prenatal Diagnosis in a High-Risk Family

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Keywords: 3β-HSD2 deficiency; HSD3B2; Congenital adrenal hyperplasia; Prenatal diagnosis

Background. 3β-Hydroxysteroid dehydrogenase type 2 (3β-HSD2) deficiency is a rare form of congenital adrenal hyperplasia (CAH) caused by biallelic pathogenic variants in the HSD3B2 gene. The absence of genital virilization in girls makes early diagnosis difficult.

Objective. To present a case of salt-wasting 3β-HSD2 deficiency in a girl with molecular genetic confirmation and subsequent prenatal diagnosis in the family.

Case Presentation. A 4-month-old girl was referred to our center for severe failure to thrive. Investigations revealed hyponatremia, hyperkalemia, elevated ACTH and low cortisol, with elevated 17-OHP, DHEA-S and 17-OH-pregnenolone. The external genitalia were female, without virilization. Replacement therapy with hydrocortisone (15 mg/m²/day) and fludrocortisone (50 µg/day) was initiated.

Sequencing of the HSD3B2 gene identified a homozygous pathogenic variant, NM_000198.4:c.1003C>T (p.Arg335Ter); both parents were heterozygous carriers. The parents are non-consanguineous. A male sibling from the second pregnancy had died on day 2 of life with dehydration; no genetic testing had been performed.

After 5 months of treatment, weight and growth improved (height SDS from −1.5 to +0.5). At 6 years 6 months, bone age was 6.5–7 years. Now aged 7 years, she has had no adrenal crises.

In the third pregnancy, first-trimester chorionic villus sampling showed that the fetus did not carry the familial HSD3B2 variant. The pregnancy is ongoing (7 months of gestation).

Conclusion. Salt-wasting adrenal insufficiency without virilization in girls warrants exclusion of 3β-HSD2 deficiency; measurement of Δ5 steroids and molecular genetic testing are key to diagnosis. Molecular confirmation enabled carrier identification in the parents, suggested a possible cause of the sibling's death and allowed targeted prenatal diagnosis. In the absence of neonatal CAH screening in Uzbekistan, such cases are diagnosed late, after severe failure to thrive or a salt-wasting crisis, supporting the implementation of screening and cascade genetic testing in high-risk families.

BİLDİRİ № 25 · ABSTRACT 25 DOI: 10.5281/zenodo.23659784

Solid Right Adrenal Mass with Antenatal Presentation: Challenges in Differential Diagnosis

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Keywords: Pheochromocytoma; Neuroblastoma; Adrenal mass; Infancy

Background. Solid adrenal masses in the first months of life are most often neuroblastomas; pheochromocytoma at this age has been reported only in isolated cases. Similar imaging features and a possible hormonally inactive course complicate non-invasive differential diagnosis. We present an antenatally detected solid right adrenal mass in an infant and assess the value of laboratory and imaging methods in differentiating these tumors.

Case Presentation. Third-trimester ultrasound screening revealed a hyperechoic fetal lesion (25×24 mm) with clear, smooth contours above the right kidney. At 1 month 25 days of age, ultrasound showed a solid, heterogeneous, hypovascular mass (35×45×28 mm) in the right adrenal region, without necrosis or calcifications, deforming the upper pole of the kidney; lymph nodes were not enlarged. Contrast-enhanced MRI demonstrated a well-defined rounded lesion (38×37×40 mm), slightly hypointense on T1 and slightly hyperintense on T2 and DWI, with heterogeneous enhancement; the major vessels were intact. Neuron-specific enolase, CEA, β-hCG and alpha-fetoprotein were within normal limits. 18F-FDG PET/CT showed low metabolic activity (SUVmax 1.88) and no distant lesions. At 2 months, plasma normetanephrine was 151.2 pg/mL (reference <190.0) and metanephrine 91.2 pg/mL (reference <90.0); aldosterone and renin were normal. Elective surgical removal of the mass with histological and immunohistochemical examination was recommended.

Discussion. Imaging suggested a hormonally inactive pheochromocytoma, but neuroblastoma could not be excluded. Near-normal plasma metanephrines and low FDG uptake exclude neither tumor. Urinary catecholamine metabolites, ^{123}I-MIBG scintigraphy and molecular genetic testing are needed to narrow the diagnosis before surgery; histological verification has not yet been performed.

Conclusion. An antenatally detected solid adrenal mass that enlarges after birth requires differentiation between neuroblastoma and pheochromocytoma. Laboratory and imaging findings alone may be inconclusive, so histological verification remains decisive, within multidisciplinary management involving pediatric endocrinologists and oncologists.

BÖLÜM 7 · SECTION 7

Kalsiyum, Kemik ve Paratiroid Bozuklukları

Calcium, Bone and Parathyroid Disorders

3 bildiri · 3 abstracts

BİLDİRİ № 26 · ABSTRACT 26 DOI: 10.5281/zenodo.23659786

A Diagnostically Challenging Case of Pseudohypoparathyroidism Type 1B: Endocrine Awareness in Bone Cysts

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Keywords: Pseudohypoparathyroidism type 1B; GNAS; Bone cysts; PTH resistance

Introduction

PHP type 1B results from methylation defects at the GNAS locus, causing PTH resistance in the renal proximal tubule, usually without the Albright hereditary osteodystrophy (AHO) phenotype. Chronic PTH elevation may increase bone turnover and rarely cause lytic-cystic bone lesions (brown tumors), which can mimic malignancy and delay diagnosis.

Case Presentation

A 10-year-old patient underwent distal femoral hemiepiphysiodesis for genu valgum at another center. Postoperative MRI showed a 12×8 mm cystic lesion in the right iliac bone, interpreted as a simple cyst. PTH was elevated (1242 pg/mL) and 25-OH vitamin D was low (9 mcg/L). During follow-up, the lesion enlarged to 58 mm, and pelvic CT revealed expansile lesions in both iliac bones and the left pubic bone, prompting referral to our clinic.

At admission (age 11.05 years), height was 139 cm (−1 SD) and BMI 20.5 kg/m² (0.8 SD). Examination revealed a Trendelenburg gait and limited internal rotation of both hips; no AHO phenotype was present. PTH was 714 pg/mL, with calcium and phosphorus at or slightly above the upper limit of normal (Table 1). Neck imaging showed no evidence of parathyroid adenoma. Bone scintigraphy revealed focal uptake in the cranium, right humerus, and left femur.BMD was normal, and no other hormone resistance was detected.

PHP type 1B was suspected and calcitriol (0.5 mcg twice daily) was initiated. MLPA analysis of GNAS and STX16 revealed hypomethylation at the GNAS-AS1, GNAS-XL, and GNAS A/B regions.Biochemical parameters improved during follow-up (Table 1), and the patient had no active complaints.

Conclusion

PHP type 1B may present without hypocalcemia, and borderline hyperphosphatemia may be overlooked, delaying diagnosis. Phosphorus levels should be evaluated alongside vitamin D deficiency in the differential diagnosis. In unexplained cystic bone lesions, PTH should be measured before advanced imaging or surgery, and elevated levels warrant endocrine evaluation

for pseudohypoparathyroidism.

Table 1. Laboratory findings and treatment during follow-up

Time point	Calcium (mg/dL)	Phosphorus (3.7–5.6 mg/dL)	ALP (120–488 U/L)	PTH (pg/mL)	25-OH vitamin D (mcg/L)	Calcitriol treatment
Outside center, before diagnosis	9.3	5.8	895	1242	9	–
At diagnosis	9.2	5.7	691	714	31.7	2× 0.5 mcg
Month 1 of treatment	9.4	6.2	690	555	–	2× 0.5 mcg
Month 3	9.5	6.3	591	578	22.5	1 + 0.5 mcg
Month 6	9.8	6.26	418	269	–	1.5 + 1 mcg
Month 9	9.9	5.7	420	163	15	1.5 + 1 mcg

BİLDİRİ № 27 · ABSTRACT 27 DOI: 10.5281/zenodo.23659788

Clinical Spectrum and Inheritance Patterns in IPPSD2 and IPPSD3: A Single-Center Experience

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Keywords: IPPSD; GNAS; Pseudohypoparathyroidism; Inheritance

Background/Introduction: IPPSD2 and IPPSD3 are caused by pathogenic variants in GNAS and epigenetic abnormalities affecting the GNAS locus, respectively. This study aimed to compare the clinical, anthropometric, and laboratory characteristics of patients with IPPSD2 (formerly PHP1A) and IPPSD3 (formerly PHP1B) and to evaluate inheritance patterns.

Methods: Ten patients with a clinical diagnosis of IPPSD were retrospectively evaluated. Age at diagnosis, anthropometric measurements, phenotypic features, and hormone resistance were assessed. Patients with pathogenic GNAS variants were classified as IPPSD2 (n=6), whereas those with methylation abnormalities at the GNAS locus were classified as IPPSD3 (n=3). One patient with negative GNAS sequencing and MLPA results who was undergoing SNP-array analysis was excluded from subgroup comparisons. Parental GNAS sequencing and MS-MLPA analyses were performed to assess inheritance patterns.

Results: Median age at diagnosis was 2.8 years in IPPSD2 and 10.9 years in IPPSD3. Median height SDS was −0.9 and 0.2, respectively. Albright hereditary osteodystrophy (AHO) features were more prominent in IPPSD2, whereas IPPSD3 patients had milder phenotypic findings. Hypothyroidism was present in all IPPSD2 patients (100%) and in one IPPSD3 patient (33%). Heterotopic ossification was observed in 50% and 33% of patients, respectively. Maternal transmission was demonstrated in two patients, while the remaining molecularly confirmed cases were consistent with de novo occurrence based on parental molecular analyses.

Conclusion: In this cohort, IPPSD2 was characterized by earlier diagnosis, lower height SDS, and more pronounced AHO features than IPPSD3. The closer-to-normal height SDS values and milder phenotype observed in IPPSD3 may reflect a less prominent clinical presentation. The predominance of apparently de novo cases highlights the diagnostic value of molecular testing even in the absence of a positive family history. Larger cohorts are needed to confirm these observations.

BİLDİRİ № 28 · ABSTRACT 28 DOI: 10.5281/zenodo.23659791

Contribution of Four-Dimensional Computed Tomography to the Localization of Parathyroid Lesions in Pediatric Primary Hyperparathyroidism

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Keywords: Primary hyperparathyroidism; Four-dimensional CT; Parathyroid localization; Children

Introduction: Primary hyperparathyroidism (PHPT) is a rare endocrine disorder in childhood, and its definitive treatment is surgery. Accurate preoperative localization is important for surgical planning, particularly in ectopic, small or multiglandular disease. Ultrasonography (US) and 99mTc-sestamibi scintigraphy/SPECT-CT are first-line localization methods but may be insufficient in some cases. Although four-dimensional computed tomography (4D-CT) provides high anatomical resolution, its use in children should be selective because it involves ionizing radiation. This study aimed to evaluate the contribution of 4D-CT to the localization of parathyroid lesions and its added value over conventional imaging in pediatric PHPT.

Methods: Patients followed at our center with a diagnosis of PHPT between January 2020 and June 2026 were retrospectively evaluated. Patients with a biochemical diagnosis of PHPT who underwent parathyroidectomy and achieved biochemical cure after surgery were included. Preoperative US, 99mTc-sestamibi scintigraphy/SPECT-CT, conventional contrast-enhanced neck CT, cervical MRI and 4D-CT findings were compared with surgical and histopathological results. The added contribution of 4D-CT over US and other imaging methods, and the ionizing radiation burden, were assessed on a per-patient basis.

Results: Twelve patients with PHPT were included. Biochemical cure was achieved in all patients after one or two surgical procedures. US detected the lesion in 9/12 (75%) patients, and 99mTc-sestamibi scintigraphy/SPECT-CT in 8 of 11 (72.7%). Conventional contrast-enhanced neck CT was performed in four patients and 4D-CT in eight selected patients. 4D-CT demonstrated at least one candidate parathyroid lesion in all eight patients in whom it was performed, with complete agreement with the final surgical localization in seven (87.5%). In two US-negative cases, 4D-CT showed the anatomical location of the lesion; in one patient it defined the ectopic location in more detail; and in four patients it confirmed previous imaging findings. In one patient with multiglandular disease, two lesions were shown but not all pathological foci could be localized, and a second surgical procedure was required.

Conclusion: 4D-CT may contribute to localization in selected pediatric PHPT cases, particularly those not localized by US, those with ectopic or anatomically difficult locations, and those with suspected small or multiglandular disease. Our findings support the use of 4D-CT, which involves ionizing radiation, as a second-line imaging method in selected cases where conventional imaging is insufficient, rather than its routine use in all pediatric PHPT patients.

BÖLÜM 8 · SECTION 8

Cinsiyet Gelişim Bozuklukları

Disorders of Sex Development

3 bildiri · 3 abstracts

BİLDİRİ № 29 · ABSTRACT 29 DOI: 10.5281/zenodo.23659793

Clinical Characteristics and Genetic Diagnoses in Patients with Undescended Testes

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Keywords: Undescended testis; Cryptorchidism; Genetic diagnosis; Disorders of sex development

Introduction/Aim: This study aimed to evaluate the demographic, clinical, biochemical, imaging and follow-up characteristics, as well as the genetic diagnoses, of patients referred to our clinic for undescended testes.

Methods: Patients with a predominantly male phenotype referred for undescended testes were retrospectively reviewed. Demographic characteristics, anatomical location of the testes, accompanying genital findings and the External Genitalia Score (EGS) were recorded. Hormonal evaluation included basal gonadotropins, gonadal steroids, anti-Müllerian hormone (AMH) and β-hCG stimulation test results. Genetic evaluation began with karyotype analysis; in patients with a clinical indication, clinical exome sequencing with a disorders of sex development (DSD) panel or whole-exome sequencing was performed.

Results: Sixty patients were included. Median age at presentation was 2 (0.5–15.72) years and median EGS was 10 (7–11.5). Bilateral undescended testes were present in 90% of patients, micropenis in 6.7% and Müllerian structures in 10%. Genetic investigation was planned for 55 (91.6%) patients with EGS <11; it was performed in 32 (53.3%), and a cryptorchidism-related genetic diagnosis was established in 22 (22/32, 68.7%). Etiologies included 5α-reductase deficiency, androgen receptor and AMH receptor defects, congenital adrenal hyperplasia, Noonan, Down, Prader-Willi and Escobar syndromes, and 45,X/46,XY mixed gonadal dysgenesis.

Conclusion: Undescended testis may be a sign of an underlying genetic etiology rather than an isolated surgical problem. While the reported rate of genetic diagnosis in cryptorchidism cohorts is 3–8%, it was 68.7% in our phenotype-selected cases. Even when the external genitalia appear near-normal, a systematic evaluation including EGS, accompanying dysmorphic and systemic findings, and routine karyotype analysis may enable early recognition of genetic causes.

BİLDİRİ № 30 · ABSTRACT 30

DOI: 10.5281/zenodo.23659795

Hereditary Disorders in Patients with Cryptorchidism: Clinical and Genetic Aspects

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Keywords: Cryptorchidism; Hereditary disorders; Karyotyping; Disorders of sex development

Background. Cryptorchidism is a multifactorial condition that may occur as an isolated congenital anomaly or as a feature of chromosomal disorders, monogenic syndromes, and differences of sex development (DSD). Identifying an underlying hereditary cause is important for diagnosis, further evaluation, and assessment of reproductive prognosis.

Objective. To assess the frequency and spectrum of hereditary disorders in patients with cryptorchidism.

Materials and Methods. We retrospectively analyzed data from 488 male patients aged 3–20 years with cryptorchidism evaluated at the Republican Specialized Scientific and Practical Medical Center of Endocrinology between 2000 and 2024. Syndromic conditions were diagnosed on the basis of phenotypic features and established clinical criteria; karyotyping [and molecular testing] was performed when clinically indicated. Proportions are presented with 95% confidence intervals (CIs, Wilson method).

Results. Hereditary disorders were identified in 45 of 488 patients (9.2%; 95% CI, 7.0–12.1), comprising four groups. Monogenic syndromes (n = 20; 44.4%): fragile X syndrome, 6; Bardet–Biedl, Noonan, Seckel, and Kallmann syndromes, 3 each; Bloom and Robinow syndromes, 1 each. Chromosomal abnormalities (n = 11; 24.4%): Klinefelter syndrome (47,XXY), 5; Down syndrome, 4; Y-chromosome abnormalities, 2. DSD (n = 8; 17.8%): partial androgen insensitivity, 3; gonadal dysgenesis, 2; undervirilization of unknown etiology, 1; unclassified DSD, 2. Imprinting disorders (n = 6; 13.3%): Prader–Willi syndrome, 4; Silver–Russell syndrome, 2. All chromosomal abnormalities were confirmed by karyotyping; diagnoses were confirmed by. Five cases (Seckel, Bloom, and Robinow syndromes) were considered probable pending molecular confirmation.

Conclusion. In a tertiary endocrinology center, hereditary disorders were identified in approximately one in 11 patients with cryptorchidism, predominantly monogenic syndromes and chromosomal abnormalities. Because karyotyping was performed selectively, the frequency of chromosomal abnormalities may have been underestimated. These findings support systematic phenotypic assessment of all patients with cryptorchidism, karyotyping in bilateral cryptorchidism or suspected DSD, in line with current recommendations, and wider access to molecular genetic testing and genetic counseling.

BİLDİRİ № 31 · ABSTRACT 31 DOI: 10.5281/zenodo.23659797

Timing of Orchidopexy in Cryptorchidism and Long-Term Testicular and Spermatogenic Outcomes

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Keywords: Cryptorchidism; Orchidopexy; Spermatogenesis; Testicular volume

Background. Cryptorchidism is one of the most common congenital anomalies of the male genitalia and is associated with impaired testicular development and reduced fertility potential. International guidelines recommend orchidopexy before 18 months of age; however, diagnosis and surgical treatment are often delayed.

Objective. To assess the age at first referral and at orchidopexy in children with cryptorchidism, temporal trends in age at surgery, and long-term testicular and spermatogenic outcomes after late orchidopexy.

Materials and Methods. We retrospectively analyzed data from 362 patients with cryptorchidism treated at our center between 2000 and 2020. Long-term outcomes were evaluated in 27 young men aged 16–21 years, 10–12 years after unilateral orchidopexy performed at 4–11 years of age; the control group comprised 10 healthy age-matched young men. Testicular volume was measured, and semen analysis was performed according to WHO (2010) criteria. Data are presented as mean ± SD.

Results. The mean age at first referral was 6.6 ± 2.5 years (median, 7 years). The mean age at orchidopexy decreased from 10.4 years in 2000 to 4.4 years in 2015 and 3.1 years in 2020.; however, only 6.5% of patients underwent surgery at 1 year of age, and orchidopexy was most frequently performed at 3 years (27.3%).

At follow-up, mean testicular volume in young men after orchidopexy was less than half that of controls (9.5 ± 3.2 vs. 20.7 ± 3.3 mL; $p<0.001$), and sperm concentration was fourfold lower (18.1 ± 8.0 vs. 75.9 ± 27.7 $\times 10^6$/mL; $p<0.001$).

Conclusion. Although the age at orchidopexy declined over the study period, most children with cryptorchidism still underwent surgery much later than recommended. Young men who underwent orchidopexy at 4–11 years of age showed markedly reduced testicular volume and sperm concentration compared with healthy peers. These findings support the need for early detection of cryptorchidism in primary care and orchidopexy before 18 months of age.

BÖLÜM 9 · SECTION 9

Endokrin Otoimmünite ve Diğer Konular

Endocrine Autoimmunity and Other Topics

2 bildiri · 2 abstracts

BİLDİRİ № 32 · ABSTRACT 32 DOI: 10.5281/zenodo.23659801

Immunological Abnormalities in Children with Autoimmune Endocrine Diseases: Association with Multiple Autoimmunity

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Keywords: Autoimmune endocrine diseases; Inborn errors of immunity; Multiple autoimmunity; Hashimoto's thyroiditis

Introduction:
Autoimmune endocrine diseases, particularly early-onset or multiglandular presentations, may be manifestations of an underlying inborn error of immunity (IEI). This study compared the frequency of documented immunological abnormalities in children with and without autoimmune endocrine diseases and examined their association with multiple autoimmunity.

Materials and Methods:
This retrospective, cross-sectional study reviewed patient records from the Pediatric Endocrinology and Pediatric Allergy and Immunology clinics of Ankara Training and Research Hospital. Of 347 records reviewed, 324 with documented immunological assessment status were included: 184 with autoimmune endocrine diseases and 140 without autoimmune disease, comprising 126 controls and 14 children with thyroid function abnormalities. Endocrinology patient were routinely referred to pediatric immunology, where clinical history guided laboratory investigations. Available complete blood counts and serum immunoglobulin levels were reviewed, together with lymphocyte subsets, IgG subclasses, and genetic test results in selected cases. Immunological abnormalities were identified from specialist-recorded assessments. Group frequencies were compared using Pearson's chi-square test

Results:
Immunological abnormalities were documented in 46 of 184 children with autoimmune endocrine diseases (25.0%) and 21 of 140 children without autoimmune disease (15.0%; p=0.028). Among children with autoimmune diseases, 151 (82.1%) had a single autoimmune condition and 33 (17.9%) had multiple autoimmune conditions. Immunological abnormalities were more frequent in those with multiple autoimmunity than in those with a single autoimmune condition (14/33,42.4% versus 32/151, 21.2%; p=0.011). Hashimoto's thyroiditis was the most common diagnosis, affecting 119 patients (64.7%), including those with coexisting autoimmune conditions, followed by type 1 diabetes mellitus.

Conclusion: In this selected clinical population, documented immunological abnormalities were more frequent in children with autoimmune endocrine diseases, particularly those with multiple autoimmunity. These findings support considering clinical history-guided immunological assessment in children with multiple autoimmune conditions.

BİLDİRİ № 33 · ABSTRACT 33 DOI: 10.5281/zenodo.23659803

Performance of a Large Language Model on Pediatric Endocrinology Test Questions Across Common Disorders

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Keywords: Large language models; Artificial intelligence; Pediatric endocrinology; Medical education

Background: Artificial intelligence (AI), particularly large language models (LLMs), is increasingly used in medical education and clinical decision support. Its reliability in pediatric endocrinology, where diagnostic criteria depend on age and growth parameters, remains insufficiently studied. We aimed to evaluate LLM accuracy on test questions covering the most prevalent pediatric endocrine disorders.

Methods: AI was used as the object and method of the study. A set of 135 multiple-choice questions was compiled from standard pediatric endocrinology guidelines and divided into five sections of 27 questions each: childhood obesity, vitamin D deficiency and rickets, thyroid disorders, childhood diabetes, and growth disorders. Sections were ranked by disease prevalence. Each question was submitted to an LLM in a separate session, using an identical prompt. Answers were compared with a reference key. Accuracy was calculated as the percentage of correct answers for each section and overall, and 95% confidence intervals were estimated.

Results: The LLM answered 116 of 135 questions correctly (85.9%; 95% CI 79.0-90.8). Accuracy by section, in order of decreasing prevalence: childhood obesity 27/27 (100%), vitamin D deficiency and rickets 24/27 (88.9%), thyroid disorders 23/27 (85.2%), childhood diabetes 23/27 (85.2%), growth disorders 19/27 (70.4%). Accuracy tended to be higher in sections covering more prevalent conditions with well-standardized guidelines, and lower in sections requiring interpretation of growth charts and age-specific thresholds. Incorrect answers most often concerned diagnostic cut-off values.

Conclusion: The LLM showed high accuracy on common pediatric endocrine topics, with a decrease in sections requiring quantitative, age-dependent interpretation. AI may serve as a supportive educational tool, but its answers require verification by a specialist. Further studies with multiple models are warranted.

BÖLÜM 10 · SECTION 10

Geç Gelen Bildiriler

Late-Breaking Abstracts

1 bildiri · 1 abstract

BİLDİRİ № 34 · ABSTRACT 34

DOI: 10.5281/zenodo.23911157

Epidemiology of Type 1 Diabetes in Children in Azerbaijan: Trends from 2017 to 2025 and Projections for 2026–2030

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Keywords: Type 1 diabetes mellitus; Children; Epidemiology; Azerbaijan

Background and Objective: Type 1 diabetes mellitus (T1DM) is one of the most common chronic endocrine disorders of childhood, and its incidence is rising worldwide. Reliable national data are essential for healthcare planning and resource allocation. This study aimed to describe trends in newly diagnosed pediatric T1DM cases in Azerbaijan in 2017–2025, to report registered prevalence in 2023–2024, and to estimate possible trends up to 2030.

Materials and Methods: A retrospective descriptive analysis was conducted using records of the Republican Pediatric Center, the national referral institution for pediatric diabetes, and data from the national registry of the Republican Endocrinology Center. Children aged 0–17 years were included. Annual hospital-based case rates per 100,000 children were calculated using an approximate constant population denominator of 2,560,000. Rates for 2026–2030 were projected using the compound annual growth rate (CAGR) for 2017–2025.

Results: The number of newly diagnosed T1DM cases recorded at the Republican Pediatric Center increased 3.6-fold, from 128 in 2017 to 463 in 2025, corresponding to estimated rates of 5.0 and 18.1 per 100,000 children, respectively (CAGR 17.4%). A transient decline was observed in 2023, followed by a marked increase in 2024–2025. The national registry included 2,415 children with T1DM in 2023 and 3,075 in 2024 (approximately 94 and 120 per 100,000 children), an increase of 27.3% in one year. If the 2017–2025 growth rate were to continue, the case rate would reach 21.2, 24.9, 29.3, 34.4 and 40.4 per 100,000 children in 2026–2030, respectively.

Conclusion: The number of children newly diagnosed with T1DM and the number of registered cases in Azerbaijan increased substantially over the study period. Part of this increase may reflect improved case ascertainment and centralized referral rather than a true rise in incidence alone. Because rates were derived from referral-center data with a fixed population denominator, and projections assume constant exponential growth, these estimates should be interpreted with caution. A population-based national pediatric diabetes registry is needed to confirm these trends and to plan pediatric endocrinology services.