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Skinfold Thickness			
Bicep	13.6.(12.7–18.8)	14.3.(14.7–17.4)	.689
Tricep	21.4.(20.2–26.2)	21.3.(21.6–24.3)	.852
Subscapular	16.8.(15.2–22.6)	18.3.(19.3–22.9)	.669
Supra-iliac	32.1.(25.7–36.9)	26.2.(26.6–30.6)	.350
Supraspinale	21.4.(16.9–25.8)	17.9.(18.5–22.1)	.173
Abdominal	27.4.(23.0–31.4)	26.2.(25.7–29.4)	.363
Thigh	35.8.(35.7–40.7)	34.2.(31.6–44.4)	.893
Calf	22.2.(17.1–26.2)	19.9.(20.0–23.3)	.611
Total SFT	163.8.(148.4–202.9)	160.7.(163.4–186.1)	.392
Appendicular SFT	95.2.(83.3–114.0)	91.9.(92.5–105.1)	.979
Trunkal SFT	93.8.(82.1–115.3)	86.8.(90.6–104.6)	.979
% body fat	38.1.(36.2–42.3)	38.0.(38.3–40.9)	1.00
Girths MUAC	30.0.(28.4–32.3)	30.2.(29.6–31.2)	.936
MAMC	23.0.(21.8–24.4)	22.7.(22.7–23.7)	.979
Waist	80.2.(78.1–88.3)	80.3.(81.1–85.1)	.957
Hip	94.8.(91.9–104.1)	95.0.(94.7–98.8)	1.000

*=statistically significant at $P \leq 0.05$

Conclusion

Parameters of body composition in early gestation do not predict neonates born large for gestational age.

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AEP433

Associations between FSH levels and indices of total and regional obesity in women after menopause

Eleni Armeni¹, Stavroula A. Paschou¹, Areti Augoulea¹, Stefanos Stergiotis¹, Panagiota Chatzivasilioi¹, Dimitrios Rizos², George Kaparos³, Konstantinos Panoulis¹, Anastasia Palaiologou¹ & Irene Lambrinoudaki¹
¹Second Department of Obstetrics and Gynecology, Aretaieio Hospital, Medical School, National and Kapodistrian University of Athens, Athens, Greece; ²Hormonal and Biochemical Laboratory, Aretaieio Hospital, Medical School, National and Kapodistrian University of Athens, Athens, Greece

Introduction

Recent evidence reports a controversial extra-gonadal role of follicle stimulating hormone (FSH). Conflicting data support that the association between FSH and obesity might be maintained by a direct or even indirect effect to the adipose tissue. The aim of this study was to evaluate the associations between FSH concentrations and various obesity indices in women after menopause.

Patients and methods

This cross-sectional study included 420 postmenopausal women (age 55.6 ± 6.5 years, 8.01 ± 6.7 years since menopause) with low insulin resistance (inclusion criteria: years since menopause > 1 , FSH > 25 IU/ml, HOMA-IR < 5). We recorded anthropometric parameters. Indices of regional adiposity were sonographically assessed, including subcutaneous fat and preperitoneal fat. Blood samples were obtained for biochemical and hormonal evaluation.

Results

Mean values of BMI were 25.8 ± 4.0 kg/m². Waist circumference and BMI presented a stepwise decrease with increasing quartiles of FSH (Waist, FSH Q1 vs Q2 vs Q3 vs Q4: 93.2 ± 2.4 vs 87.6 ± 4.4 vs 85.4 ± 1.8 vs 80.89 ± 2.8 ; BMI, FSH Q1 vs Q2 vs Q3 vs Q4: 27.6 ± 5.2 vs 26 ± 4.8 vs 25.8 ± 7.1 vs 23.9 ± 2.9 ; ANOVA p-value for linear trend < 0.001 , both cases). Similarly, subcutaneous and preperitoneal fat measures decreased linearly with increasing quartiles of FSH (ANOVA P-value for linear trend < 0.001). Stepwise linear regression analysis showed that preperitoneal fat is inversely associated with FSH, independently of circulating estrogen (b coefficient = -0.130 , P -value = 0.029) and traditional cardiovascular risk factors. The association between FSH and subcutaneous fat was not evident following adjustment for circulating estrogens, implying a possible mediation effect of the latter on this association.

Conclusions

FSH is inversely associated with indices of total and regional adiposity in women after menopause. The exact mechanism of this interaction remains to be elucidated in future studies.

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AEP434

Thyroid stimulating hormone, insulin resistance and leptin in patients with obesity after bariatric surgery

Anna R. Volkova, Galina Semikova & Michael Fishman
 Pavlov First State Medical University of Street Petersburg, Saint Petersburg, Russian Federation

Background

The function of the thyroid gland effects on obesity and comorbidities. It has been proven for bariatric surgery to be the most effective in obesity treatment.

Aim

To evaluate the dynamics of body weight, thyroid status, leptin and insulin resistance in obese patients after bariatric surgery.

Materials and methods

78 obese patients were observed after bariatric surgery (sleeve gastrectomy – 46, gastric bypass – 32). Body mass index (BMI), thyroid stimulating hormone (TSH), free T4, fasting plasma leptin, insulin and glucose were estimated; the insulin resistance index HOMA-IR was calculated. The dynamics of body weight was estimated by BMI and the excess BMI loss (% EBMIL). After 3 years of follow-up, 50 patients were examined.

Results

Subclinical hypothyroidism (SH) was detected in 37.2% of patients with high degrees of obesity. A correlation was found between BMI and TSH level ($R=0.5$; $P=0.01$). HOMA-IR was increased in most patients with obesity of the II and III degree (5.2 ± 2.3 ng/ml). In the SH group, the leptin level was significantly higher than in the group with a normal TSH level: 44.1 ± 7.4 ng/ml and 33.0 ± 4.7 ng/ml ($P=0.004$). Among patients with initial SH, spontaneous reduction of TSH levels occurred in 42.7% patients 3 years after surgery.

Conclusions

Postoperatively, the decrease of BMI was associated with the decrease of TSH, leptin and HOMA-IR. The data obtained may reflect the effect of adipose tissue on the functional state of the thyroid gland in patients with high degrees of obesity after bariatric surgery. This seems to be extremely important for maintaining body weight.

Keywords: obesity, thyroid stimulating hormone, leptin, insulin resistance, bariatric surgery.

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AEP435

Fructose-induced alterations of hepatic lipid metabolism are modulated by chronic stress in male rats

Natasa Velickovic¹, Danijela Vojnovic Milutinovic¹, Jelena Brkljadic¹, Ana Teofilovic¹, Biljana Bursac¹, Marina Nikolic¹, Ljupka Gligororovska¹, Sanja Kovacevic¹, Ana Djordjevic¹, Frédéric Preitner², Luc Tappy³ & Gordana Matic¹

¹Institute for Biological Research 'Siniša Stanković', Department of Biochemistry, Belgrade, Serbia; ²Center for Integrative Genomics, University of Lausanne, Lausanne, Switzerland; ³University of Lausanne, Department of Physiology, Lausanne, Switzerland

Overconsumption of fructose-enriched beverages and everyday stress are both involved in the pathogenesis of metabolic disorders through their effects on hepatic lipid metabolism. The aim of this study was to investigate whether high-fructose diet and chronic stress synergistically perturb lipid metabolism in rat liver. Therefore, we analyzed the effects of 9-week 20% liquid fructose diet and 4-week chronic unpredictable stress, separately and in combination, on dyslipidemia, VLDL-TG kinetics, intrahepatic triglycerides (IHTG), liver *de novo* palmitate (DNPalm) content and fatty acid (FA) composition. In parallel, hepatic fractional *de novo* lipogenesis (FDNL) by stable isotope tracer protocol, as well as expression of lipid metabolism regulators were also analyzed. Results showed that high-fructose diet led to hypertriglyceridemia, increased plasma VLDL-TGs and free FA (FFA), and increased visceral adiposity. Fructose diet also augmented the

level of palmitate, palmitoleate and oleate in the liver, the latter being result of increased desaturase activity. In addition, newly synthesized palmitate (DNPalm content) was increased in the liver of fructose-fed animals, most likely as a result of stimulated fDNL. Chronic stress alone did not exert such effects, but when combined with fructose, stress decreased FFA level, ameliorated fructose-induced TG accumulation, and augmented the release of VLDL-TGs. Stress also enhanced the effects of high-fructose diet on fDNL, which was accompanied with increased expression of key regulators of lipid metabolism, that resulting in stimulated export of newly synthesized palmitate in the form of VLDL-TGs. These results imply that high-fructose diet affects hepatic lipid metabolism by stimulating fDNL and increasing *de novo* synthesized palmitate, which is partially accumulated in the liver and in part released into circulation in the form of VLDL-TGs. On the other hand, stress in combination with high-fructose diet potentiated hepatic fDNL, but it decreased temporary TG storage and redirected newly synthesized palmitate into VLDL-TGs. Thus, the combination of high-fructose diet and chronic stress, as hallmarks of modern lifestyle, exerts more detrimental influence on lipid homeostasis than the individual factors, judged by stimulated fDNL and increased export of VLDL-TGs to non-hepatic tissues.

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AEP436

Autoimmune polyglandular syndrome type ii presenting as an endocrine emergency

Malika Ichiche, Iconaru Laura & Karmali Rafik
CHU Brugmann, Endocrinology, Laeken, Belgium

Introduction

Autoimmune polyglandular syndrome (APS) is a group of polyendocrinopathies characterized by multiple glands insufficiencies associated with other autoimmune diseases resulting from immune mediated destruction. We describe a patient with type 2 APS presenting first with diabetes type 1 followed later by adrenal insufficiency and Hashimoto disease.

Case report

A 23-year-old male, known with diabetes type 1, presented with a very low blood pressure. He complained of nausea, vomiting, general weakness, easy fatigability, postural dizziness and gradual darkening of the skin since 3 months.

Physical examination revealed both general hyperpigmentation and vitiligo. Laboratory studies showed significant hyponatremia, hyperkalemia. Morning cortisol was very low 28 nmol/l (22.1–353) and elevated ACTH 1557 ng/ml (7.2–63.3), with a high level of adrenal autoantibodies, which confirm the diagnosis of autoimmune primary adrenal insufficiency. He was started on replacement therapy with physiological doses of prednisolone and fludrocortisone resulting in marked improvement in his symptoms. Further evaluation revealed also an auto-immune hypothyroidism which required Levothyroxine supplementation.

The analysis for mutations in the AIRE gene was negative, without excluding genetics polymorphisms. Screening for other auto-immune diseases associated with APS 2 was negative.

Discussion

Both APS 1 and 2 are associated with type 1 diabetes. The type 2 syndrome is much more prevalent than the type 1 syndrome and primary adrenal insufficiency is its principal manifestation. Adrenal insufficiency is the initial manifestation in about 50 percent of patients, occurs simultaneously with autoimmune thyroid disease or diabetes mellitus in about 20 percent, and follows them in about 30 percent.

Our patient had type 1 diabetes and presented with adrenal insufficiency with an adrenal crisis. He was diagnosed as a case of APS type 2 consistent of Addison's disease, type 1 diabetes, autoimmune thyroid disease and vitiligo. We could not detect mutations in the AIRE gene. However it is possible that certain mutations are not detectable with the used technique.

Conclusion

In type 1 diabetes patients and their relatives a search for APS is crucial given predilection to other concomitant autoimmune diseases. Furthermore regular surveillance in these patients is crucial to screen for these other autoimmune disorders even decades after the initial diagnosis.

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AEP437

Hepatic steatosis indices as a predictors of vitamin D3 deficiency in patients with non-alcoholic fatty liver disease and type 2 diabetes

Udwan Mahmoud¹, Nazarii Kobylak¹, Dmytro Kyriienko² & Iuliia Komisarenko¹

¹Bogomolets National Medical University, Kyiv, Ukraine; ²Kyiv City Clinical Endocrinology Center, General Endocrine Pathology, Kyiv, Ukraine

Background

Recently, vitamin D3 deficiency is considered one of the factors associated with the development of non-alcoholic fatty liver disease (NAFLD). The aim was to evaluate steatosis indices and metabolic parameters in NAFLD depending on D3 status.

Materials and methods

According to the recommendations of the European Society of Endocrinology, all patients were divided into 3 groups: group 1 – with an optimal level of vitamin D3 (30 ng/ml); group 2 – D3 insufficiency (21–29 ng/ml) and group 3 – D3 deficiency (<20 ng/ml).

Results

The study included 126 T2D patients with NAFLD diagnosed with US. The highest hepatic steatosis (HSI) and fatty liver (FLI) index values were diagnosed in D₃ deficiency as compared to optimal group (HSI – 43.34±6.59 vs 39.67±4.37; *P*=0.032 and FLI – 79.21±19.61 vs 64.96±17.72; *P*=0.007). Triglyceride and glucose index (TyG) also insignificantly growth parallel to D3 status worsened (*P*=0.175). In multivariate logistic regression analysis according to the results obtained, regardless of the transaminases activity HSI (Nagelkerke *R*²=0.215) and FLI (Nagelkerke *R*²=0.163) were associated with vitamin D₃ deficiency. According to other logistic models, HSI and TyG indices (Nagelkerke *R*²=0.358) as well as body mass index (BMI) and T2D duration (Nagelkerke *R*²=0.328) were independent predictors associated with D3 deficiency in this cohort of patients.

Conclusions

hepatic steatosis indices (HSI, FLI and TyG) independently from anthropometric parameters and transaminase activity associated with D3 deficiency in NAFLD patients.

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AEP438

Clinical differences between patients with new-onset diabetes and those without it in a cohort of individuals with pancreatic cancer

Carlos Silva Vaca, David Males, ALBA MARTIN GONZALEZ, Elena García, Guillermo Martínez & Miguel León
Hospital 12 De Octubre, Endocrinology and Nutrition, Madrid, Spain

Background

Pancreatic Cancer (PC) is uncommon; however, it is one of the most deadly cancer types of all. New-onset diabetes (NOD) is associated with a higher risk of pancreatic cancer than general population, particularly if late-onset. An association between new-onset diabetes and increased mortality in patients with pancreatic cancer has been suggested but clinical relevant differences between those individuals with new-onset diabetes and those without are not well established in this population.

Aims

We aim to identify if any clinical differences between individuals with new-onset diabetes and those without exist within a cohort of patients with pancreatic cancer.

Materials and methods

The data was obtained from an institutional registry of 236 patients with pancreatic cancer at '12 de Octubre' University Hospital in Madrid, Spain during the period of time from 2013 to 2017. The patients' imaging studies and hospital records were reviewed. Diabetes Mellitus (DM) was defined by known medical history, or abnormal fasting blood glucose and HbA1c levels according to the American Diabetes Association 2019 criteria within four years of the cancer diagnosis. New-onset diabetes was defined by an arbitrary duration cutoff of ≤3 years since diagnosis. SPSS25.0 software package was used to perform the statistical analysis.

Results

A total of 222 patients fulfilled the inclusion criteria and were included in the final analysis. Patients were predominantly white (215, 96.8%) males (55%) with a median (Interquartile range, IQR) age of 69 (15) years. The median age of the patients was 69 years; 62 (27.9%) patients were 76 years of age or older and had pathologically confirmed pancreatic cancer. Almost one third of patients (27.3%) presented NOD criteria before PC diagnosis,