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( $-0.7 \pm 1.0\%$ ,  $P < 0.001$ ), and also had a tendency to have the biggest changes in systolic blood pressure ( $-14.7 \pm 18.7$  mmHg;  $P = 0.071$ ).

#### Conclusion

Patients with  $\geq 60$  years old benefit the most from bariatric surgery regarding parameters with cardiometabolic impact, presenting heavier reductions in fasting glucose as well as A1c, and a tendency towards a higher decrease in systolic blood pressure. No clinically-significant differences in lipid profile were observed between groups.

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### AEP379

#### TGF $\beta$ 1 downregulates hepatic SHBG production by decreasing HNF-4 $\alpha$ levels via SMAD and STAT3 pathways

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Low plasmax hormone-binding globulin (SHBG) levels are present in fatty liver disease, which represents a spectrum of diseases ranging from hepatocellular steatosis through steatohepatitis to fibrosis and irreversible cirrhosis. We are interested in studying the molecular mechanisms by which plasma SHBG levels are reduced in fatty liver disease. In this regard, we have previously shown that fat accumulation in the liver reduces SHBG production by reducing hepatic nuclear factor 4 alpha (HNF-4 $\alpha$ ), a main regulator of SHBG expression. Transforming growth factor  $\beta$ 1 (TGF $\beta$ 1) plays an important role in the pathogenesis of liver fibrosis, being involved in activation of hepatic stellate cells, stimulation of collagen gene transcription, and modulation of matrix metalloproteinase expression. The aim of the present study was to evaluate the role of TGF $\beta$ 1 in regulating hepatic SHBG production. For this purpose, *in vitro* and *in vivo* studies were performed using human HepG2 cells and human SHBG transgenic mice. Our results showed that TGF $\beta$ 1 treatment reduces SHBG production (mRNA and protein) in HepG2 cells. In addition, human SHBG transgenic mice treated with TGF $\beta$ 1 showed a significant reduction SHBG expression as well as in plasma SHBG levels. Mechanistically TGF $\beta$ 1 downregulates HNF-4 $\alpha$  levels via SMAD and STAT3 pathways through TGF $\beta$ 1 receptor I. Taking together, we found for the first time that TGF $\beta$ 1 regulates hepatic SHBG production. These results may explain why patients with fibrotic livers show low plasma SHBG levels.

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### AEP380

#### Cryptogenic cirrhosis and metabolic syndrome: What relationship?

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#### Introduction

Cryptogenic cirrhosis is cirrhosis that remains of unknown etiology after an exhaustive investigation. The relationship of cryptogenic cirrhosis with metabolic syndrome is strongly suggested. The aim of this work was to study the clinical-progressive profile of cryptogenic cirrhosis and to determine the frequency of the metabolic syndrome.

#### Patients and methods

This is a study collecting all cirrhotic patients over a period of 4 years in our gastroenterology department. The patients were divided into 2 groups (group 1 (G1): cryptogenic cirrhosis and group 2 (G2): cirrhosis of known etiology).

#### Results

A total of 71 patients were collected with an average age of 62 years. Viral origin was the most common cause (40.2%). Cirrhosis was classified as cryptogenic in 26.7% of cases ( $n = 19$ ). Metabolic syndrome was more common in group 1 with a significant difference (68.4% vs 30.4%,  $P = 0.006$ ). In group 1 patients, at least one criterion of the metabolic syndrome was found: diabetes (68.4%), hypertension (21.05%), dyslipidemia (10.5%) and overweight (73.6%). Biologically, more than half of the patients in group 1 (57.8%) had disturbances in the liver balance: cytolysis in 21.05%, cholestasis in 15.8% and an isolated increase in GGT in 21.05% of cases. By comparing the 2 groups, no significant difference was found in terms of sex, age and complications of cirrhosis except hepatocellular carcinoma which was more frequent in patients in group 2 with a significant difference ( $P = 0.05$ ).

#### Conclusion

Cryptogenic cirrhosis and metabolic syndrome are strongly associated. This association argues in favor of the role of non-alcoholic steatohepatitis

as the main cause of cryptogenetic cirrhosis. This association also does not expose to a greater risk of complications.

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### AEP381

#### Association of osteoprotegerin and metabolic status in children with obesity

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#### Objective

Determination of changes in metabolic status and osteoprotegerin concentrations in obese children.

#### Methods

We examined 221 children in the University Hospital (Minsk) from 2017 to 2019 yrs. Their anthropometric parameters (height, weight, body mass index (BMI)) were determined. Blood levels of osteoprotegerin (OPG), insulin were determined. In the biochemical blood test, the parameters of uric acid, insulin were evaluated. All children were divided into 2 groups: group 1 children with morbid obesity – 159 patients (98 boys(B)/61 girls(G)) (BMI more than 99th percentile for sex and age) (BMI  $32.95 \pm 4.61$  kg/m<sup>2</sup>, age  $14.16 \pm 2.28$  years); group 2 – 62 patients (B/G = 31/31) with alimentary obesity (BMI-95-99th percentile for sex and age) (BMI  $27.86 \pm 2.04$  kg/m<sup>2</sup>, age  $14.77 \pm 2.05$  years). The control group consisted of 84 patients (B/G = 45/39) with normal body weight (BMI  $19.86 \pm 2.24$  kg/m<sup>2</sup>, age  $14.32 \pm 2.11$  years).

**Results**  
In the subgroups of boys with obesity, there were significant differences in the concentration of uric acid in comparison with the control (alimentary obesity  $426.55 \pm 62.25$  mmol/l vs  $242.58 \pm 49.90$  mmol/l ( $P = 0.01$ )), morbid obesity  $324.10 \pm 59.33$  mmol/l vs  $242.58 \pm 49.90$  mmol/l ( $P = 0.01$ )). Girls with obesity have a significant increase in uric acid level in comparison with the control group (alimentary obesity  $328.10 \pm 51.43$  mmol/l vs  $213.0 \pm 39.64$  mmol/l ( $P = 0.0001$ )), morbid obesity  $409.04 \pm 84.23$  mmol/l vs  $213.0 \pm 39.64$  mmol/l ( $P = 0.0001$ )). In boys with obesity higher concentrations of OPG were detected relative to the control group (alimentary obesity  $259.98 \pm 108.07$  ng/ml vs  $225.12 \pm 55.88$  ng/ml ( $P = 0.09$ )), morbid  $322.22 \pm 82.14$  ng/ml vs  $225.12 \pm 55.88$  ng/ml ( $P = 0.001$ )). In girls with obesity higher concentrations of OPG were detected relative to the control group (alimentary obesity  $326.84 \pm 104.02$  ng/ml vs  $254.39 \pm 78.29$  ng/ml ( $P = 0.046$ )), morbid  $347.33 \pm 93.50$  ng/ml vs  $254.39 \pm 78.29$  ng/ml ( $P = 0.03$ )). In boys with obesity higher concentrations of insulin were detected relative to the control group (alimentary obesity  $18.9 \pm 12.7$   $\mu$ U/ml vs  $9.1 \pm 4.2$   $\mu$ U/ml ( $P = 0.0001$ )), morbid  $28.71 \pm 7.36$   $\mu$ U/ml vs  $9.1 \pm 4.2$   $\mu$ U/ml ( $P = 0.001$ )). In girls with obesity, the concentration of insulin relative to the control group was (alimentary obesity  $20.28 \pm 6.25$   $\mu$ U/ml vs  $14.10 \pm 6.80$   $\mu$ U/ml ( $P = 0.001$ )), morbid obesity  $23.32 \pm 9.65$   $\mu$ U/ml vs  $14.10 \pm 6.80$   $\mu$ U/ml ( $P = 0.001$ )).

#### Conclusion

Children with obesity have an increase in insulin and OPG rates.

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### AEP382

#### Different effects of 5 $\alpha$ -dihydrotestosterone treatment on hepatic and visceral adipose tissue inflammation in animal model of polycystic ovary syndrome

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Polycystic ovary syndrome (PCOS) is a complex reproductive disorder that is usually associated with metabolic disturbances such as obesity, dyslipidemia and insulin resistance. In this study, female rats treated with nonaromatizable 5 $\alpha$  dihydrotestosterone (DHT) were used as an animal model of PCOS. The aim of this study was to assess the presence of inflammation

in liver and visceral adipose tissue (VAT), which accompanies metabolic disturbances in animal model of PCOS. Female (21 days old) Wistar rats were treated subcutaneously with DHT pellets, while control animals received placebo pellets. Glucose, triglycerides, free fatty acids (FFA) were determined in blood plasma, while corticosterone was analyzed both in plasma and liver. Expression of genes and proteins involved in lipid metabolism, such as sterol regulatory element binding protein 1 (SREBP-1), fatty acid synthase (FAS) and acetyl-CoA carboxylase (ACC), lipin-1, adipose tissue triglyceride lipase (ATGL) and hormone-sensitive lipase (HSL), were analyzed in the VAT of treated rats. Tissue inflammation evaluated by nuclear factor kappa B (NFκB) protein level and intracellular distribution, as well as by TNFα, IL6 and IL1β mRNA levels. Glucocorticoid signaling was examined at the level of 11 beta hydroxysteroid dehydrogenase type 1 (11βHSD1) and 5α-reductase, as well as by glucocorticoid receptor (GR) level and its subcellular distribution. The results showed that DHT treatment induced increase of lipogenic factors (SREBP-1, lipin-1, FAS and PEPCK), while the level of lipolytic enzyme HSL was decreased in VAT. These molecular alterations were accompanied by adipocyte hypertrophy, visceral obesity and elevated plasma FFA and triglyceride concentrations. Those changes in lipid metabolism were possible trigger for low-grade inflammation observed in the VAT and characterized by NFκB activation and increased IL6 and IL1β mRNA levels. In spite of increased VAT proinflammatory mediators, the level of proinflammatory cytokines, IL6 and IL1β, was decreased in the liver of DHT-treated rats, while the activation of NFκB remained unchanged. The state of suppressed inflammation in the liver could be an outcome of stimulated glucocorticoid signaling, as judged by increased hepatic corticosterone level and GR activation. The augmentation of hepatic glucocorticoids could be a net result of increased expression of 11βHSD1 and decreased expression of 5β-reductase mRNA. In conclusion, the results showed that abdominal obesity and dyslipidemia in the animal model of PCOS were accompanied with hypertrophic adipocytes, lipid accumulation and low-grade inflammation in the VAT. However, these metabolic disturbances did not result in hepatic inflammation due to increased tissue levels of glucocorticoids.

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### AEP383

#### Association of AdipoQ gene & its expression in adipose tissue with postprandial hypertriglyceridemia and glucose intolerance

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#### Background

AdipoQ gene is located at the 3rd chromosome and encodes for adiponectin which is involved in fat metabolism and insulin sensitivity and plays a protective role in diabetes and atherogenesis. AdipoQ gene variants have been reported to be associated with hypertriglyceridemia and risk of diabetes in different studies. We aimed to study whether polymorphism of rs2241766 polymorphic form of AdipoQ gene & its expression in adipose tissue is associated with post prandial hypertriglyceridemia and glucose intolerance.

#### Methodology

Polymorphism study of AdipoQ gene rs2241766 was carried out by using PCR-RFLP method in 200 age and sex matched subjects who were recruited in three groups (NGT ( $n=67$ ), Prediabetes ( $n=66$ ) and T2DM ( $n=67$ )) with varying glucose tolerance following 75 gm OGTT. Gene expression studies using Real Time PCR were also done in subcutaneous and omental adipose tissue in 10 subjects from each group who were scheduled to undergo abdominal surgery. A Standardized oral fat challenge test was performed in all the study subjects to determine their post prandial Tg responses besides measurement of anthropometric (BMI, Waist) and Glycaemic (Fasting, Postprandial plasma glucose and HbA1c) indices and fasting serum insulin. Results

The mean age of study subjects were  $40.25 \pm 8.27$  years and their mean BMI was  $28.12 \pm 4.89$  kg/m<sup>2</sup>. There were 93 males and 107 females. TT genotype of rs2241766 polymorphic form of AdipoQ gene showed significantly lower 2 hr PPTg ( $190 \pm 84$  vs  $244 \pm 98$  mg/dl  $P=0.008$ ), Triglyceride area under curve ( $2445 \pm 1139$  vs  $2993 \pm 1436$  mg dl<sup>-1</sup> 2 hr<sup>-1</sup>  $P=0.05$ ), fasting-plasma glucose ( $99 \pm 13$  vs  $111 \pm 35$  mg/dl  $P<0.001$ ) and postprandial plasma glucose ( $140 \pm 46$  vs  $173 \pm 79$  mg/dl  $P=0.002$ ) as compared to GG+GT genotypes. Distribution of TT genotype of rs 2241766 polymorphic form of AdipoQ gene was found to be significantly lower in the T2DM subjects (71% vs 79%  $P=0.01$ ) as compared to NGT subjects. AdipoQ gene expression was 3.1 fold lower in prediabetes group ( $P<0.01$ ) and 2.6 fold lower in T2DM group ( $P=0.003$ ) as compared to NGT group in subcutaneous

adipose tissue which correlated significantly with postprandial plasma glucose levels ( $r=-0.48$   $P<0.04$ )

#### Conclusion

The findings of the present study indicate that TT allele of rs2241766 polymorphic form of AdipoQ gene is associated with lower postprandial triglyceride levels and a lesser degree of glucose intolerance in Asian Indian subjects. Glucose intolerant subjects also display lower AdipoQ gene expression in adipose tissue.

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### AEP384

#### Evaluation of macrovascular complications with Ankle-Brachial index in Type 2 diabetes

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#### Aim

In our study; we aimed to evaluate the relationship between Ankle-brachial index (ABI) values and diabetes duration, and macrovascular complications in patients with DM with demographic data, anthropometric measurements, as well as clinical and laboratory data.

#### Materials and methods

Our study is a cross-sectional study of 226 randomly selected DM patients who were admitted to the diabetes outpatient clinic of our hospital between April 2019 and October 2019. All patients' data, such as age, sex, height, weight, waist and hip circumference, wrist and ankle circumference, family history, DM duration, chronic disease history, claudication, neuropathy, diabetic foot ulcer history, as well as routine blood and urine examinations at control were recorded. In addition, ABI was also measured in patients. Patients with ABI values of  $\leq 0.90$  were classified as PAD-positive, and those with  $> 0.90$  were classified as PAD-negative. The data, measurements and metabolic parameters we obtained, were evaluated to determine whether they are risk factors for ABI value, their association with ABI value, and differences according to ABI value.

#### Results

A total of 226 patients (138 female, 88 male) with a mean age of  $52.8 \pm 10.3$  years were included in the study. ABI was positive in 29.2% of the patients. There was no significant difference in ABI value between age, sex, family history, body mass index (BMI), waist circumference and duration of diabetes. A significant correlation was found between ABI and coronary artery disease ( $P=0.004$ ), diabetic foot ulcer ( $P=0.000$ ), smoking ( $P=0.000$ ) and wrist circumference ( $P=0.026$ ). No significant correlation was found between ABI value and glycolated hemoglobin A1c (HbA1c), low density lipoprotein (LDL) cholesterol, triglyceride, non-high density lipoprotein (non-HDL) cholesterol levels and atherogenic index. Logistic regression analysis revealed that diabetic foot, coronary artery disease, BMI, duration of diabetes and HbA1c had a negative impact on ABI.

#### Conclusion

In our study, we obtained positive ABI values consistent with PAD in 29.2% of patients, suggesting that each patient with DM should be evaluated for PAD. Among patients with diabetes, those with a history of diabetic foot ulcers and/or cardiovascular disease, obesity, long duration of diabetes and patients with increased HbA1c levels under treatment, are considered as priority groups for peripheral arterial disease.

Keywords: peripheral artery disease, diabetes mellitus, ankle-brachial index.

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### AEP385

#### Maternally inherited diabetes and deafness presenting as diabetic ketoacidosis

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#### Background

A 29 year old female presented to the Emergency department with headache, vomiting and loose stools for 24 hours. She was not known to have diabetes. She had a background of bilateral sensorineural deafness for 6 years and