traumatic brain injuries (multiple falls and a ski accident) complained of years of insatiable hunger leading to hyperphagia and over 20 pounds weight gain. With tremendous will-power, she avoided additional weight gain by adopting a strict meal-plan and increasing her water intake (10–12 liters a day) to relieve her hunger. She sipped so much water, that her sodium remained 123 - 133 mmol/L (ref: 135–146) with dilute urine. Initial tests revealed: IGF-1 315 ng/ml (52–328), FSH 77.5 mIU/ml (23–116.3), LH 24.3 mIU/ml (14.2–52.3), prolactin 17.1 ng.ml (10–54.7), estradiol 17 (<31), TSH 1.09mIU/ml (0.45–4.5), FT4 1.0 ng/dl(0.8–1.8), all within the normal limit for her age. Semaglutide 0.25mg/week was started and increased to 0.5mg/week. Within the first six months of treatment, she experienced 22 pounds of weight loss, hunger relief, less water sipping behavior, and more enjoyment of food. Her sodium rose to 137 mmol/L. **Case 2:** A 40-year-old female, s/p craniectomy and aneurysm clipping due to intracranial hemorrhage complicated by an ischemic stroke developed sudden, documented, 45-pound weight gain over thirteen months despite aggressive lifestyle modification attempts. Initial labs revealed: TSH 1.33 mIU/ml (0.45–4.5), FT4 1.22 ng/dl (0.8–1.8), midnight salivary cortisol 0.03 mcg/dl (<0.09), ruling out hypothyroidism and Cushing syndrome. Liraglutide 1.8mg/day was started and has resulted to date in 26 pounds (11.8% of maximum weight) by 9 months with an associated decrease in subjective hunger. **Conclusion:** Hyperphagia can be seen in brain injury, in response to some medications, and some genetic conditions, like Prader-Willi. The exact mechanisms are not clear am may be multifactorial. In the case of brain injury, proposed mechanisms include insatiable hunger due to ventromedial hypothalamic or brain stem dysfunction, or disinhibition and poor impulse control due to frontal lobe injury. GLP-1's may act on the causal mechanism for increased hunger, or it may result in clinical improvement through a parallel pathway. More studies are warranted to investigate the application of GLP-1's to hyperphagia.

Adipose Tissue, Appetite, and Obesity

NOVEL INSIGHTS FROM THE CLINIC INTO THE DEVELOPMENT OF METABOLIC DISEASE: CASE REPORTS

Association of NOS3 and TNF Genetic Polymorphisms With the Predisposition to Elevated Cholesterol, Retrospective Study

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Background: Endothelial nitric oxide synthetase (eNOS) encoded by NOS3 gene has an important role in modulating vascular endothelial function. TNFα gene is responsible for coding TNFα protein that plays a significant role in regulating body inflammation and lipid metabolism. Many studies reported an association between NOS3 and TNFα genetic polymorphisms and elevated total cholesterol (TC) level, low-density lipoprotein (LDL), triglyceride (TG). In this study, we investigated the association of NOS3 (G>T) rs1799983 and TNFα -308G>A rs1800629 genetic polymorphisms with TC level.

Methods: A random sample of 250 subjects with an elevated TC level (defined by TC level ≥ 200mg/dL) compared with 500 healthy subjects. Sample obtained from Palestinian adults who consented to genetic and biochemical testing. Subjects genotyped for NOS3 SNP (G > T) rs1799983 and TNFα -308G>A rs1800629 using ARMS PCR. TC level was obtained for all subjects. Logistic regression analysis adjusted for age and body mass index (BMI) was performed to test for association between NOS3 and TNFα genetic polymorphisms and TC level.

Results: NOS3 T allele was significantly more frequent in the elevated TC group, (odds ratio = 1.8, 95% CI =1.02–3.18) with likelihood ratio statistically significant (P = 0.004). Homozygous TNFα variant was more frequent in the elevated cholesterol group without a statistically significant association (P = 0.54).

Discussion: Many studies reported an association between NOS3 and TNFα genetic polymorphisms and elevated TC levels. Homozygous NOS3 variant was associated with a 1.8-fold increase in the risk of high TC after adjustment for age and BMI. TNFα polymorphism didn't show a statistically

significant association with having elevated TC levels. With the increasing popularity and availability of genetic testing, NOS3 can serve as a screening tool to identify people with high risk for elevated TC. Further studies are required to understand the exact role of NOS3 genetic polymorphism in cholesterol metabolism.

Conclusion: NOS3 genetic polymorphism had a statistically significant relationship with TC levels. These results support the association between NOS3 polymorphism and elevated TC.

Adipose Tissue, Appetite, and Obesity

NOVEL INSIGHTS FROM THE CLINIC INTO THE DEVELOPMENT OF METABOLIC DISEASE: CASE REPORTS

Effects of Glucagon-Like-Peptide-1 Analogue Treatment in Genetic Obesity

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Introduction: Obesity is highly prevalent, comes with serious health burden and is difficult to treat. In a minority, there is a genetic cause for the obesity. In these patients, therapy-resistant obesity is often observed despite intensive lifestyle treatment. Moreover, it is still unclear whether bariatric surgery is less successful in genetic obesity. Liraglutide is a Glucagon-Like-Peptide-1 (GLP-1) receptor agonist or GLP-1 analogue, showing positive effects on metabolic parameters, satiety and weight loss

in lifestyle-induced obesity. We present our experiences of GLP-1 analogue treatment in patients with genetic obesity disorders. **Methods:** Adults with overweight or severe obesity and a molecularly proven genetic cause were treated with liraglutide 3,0 mg daily, in addition to ongoing intensive supportive lifestyle treatment. Anthropometrics, metabolic parameters, resting energy expenditure (REE), side effects, and subjectively reported satiety and quality of life were assessed. **Results:** Two patients with a heterozygous pathogenic melanocortin 4 recepter variant and two patients with 16p11.2 deletion syndrome, ranging in age between 21 and 32 years and in BMI between 28.1 and 55.7 kg/m^2 at baseline, were treated. At end of follow-up, ranging between 33 weeks and 12 years, a mean change in BMI and waist circumference was observed of -5.7 ± 3.8 kg/m^2 and -15.2 ± 21.1 cm, respectively. All patients reported better quality of life, three of them also reported improved satiety. Moreover, improvement of metabolic parameters was seen. No clear effect on REE was observed. Two patients experienced mild side effects, e.g. nausea and stomach pain, for a brief period.
Conclusion: We here show beneficial effects of GLP-1 analogues on weight, metabolic parameters, and quality of life in four patients with genetic obesity. Satiety improved in three of the four patients. All patient achieved at least the clinically relevant 5–10% weight loss. Our findings suggest that GLP-1 analogue treatment might be an effective treatment option, in addition to a healthy lifestyle, for patients with genetic obesity.

Adipose Tissue, Appetite, and Obesity
NOVEL INSIGHTS FROM THE CLINIC INTO THE DEVELOPMENT OF METABOLIC DISEASE: CASE REPORTS

Metabolic Improvements After Gastric Sleeve Surgery in a Patient With Familial Partial Lipodystrophy

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Familial Partial Lipodystrophy (FPLD) is a rare genetic disorder characterized by loss of subcutaneous adipose tissue mainly from peripheral areas but preservation, or increase, of fat in the face, neck, and trunk. This abnormal fat redistribution leads to a characteristic phenotype and severe metabolic derangements that are difficult to manage. FPLD often present with severe insulin resistance causing type 2 diabetes mellitus (DM2), acanthosis nigricans, hypertriglyceridemia (HTG), and non-alcoholic steatohepatitis (NASH). We present a case of FPLD with severe HTG and HTG induced pancreatitis requiring plasmapheresis, with dramatic metabolic improvements after gastric sleeve surgery. **Case presentation**: Our patient is a 40-year-old Caucasian male who was diagnosed with DM2 and HTG at age 18 when he presented with pancreatitis. He reported eruptive xanthomas with triglyceride (TG) >3000 mg/dl on the initial presentation. He has central obesity with disproportionately thin extremities and NASH. He has a strong family history of HTG and premature coronary artery disease. He was in a leptin trial; however, he was not included in an extended arm due to deterioration of his metabolic profile, specifically NASH. Despite aggressive therapy with dietary changes, fenofibrate, statin, omega-3, and niacin, he had multiple episodes of pancreatitis with TG levels >5000 mg/dl on many occasions. As a result, he was started on biweekly plasmapheresis that was later changed to weekly. His insulin requirement increased to 450 units daily on U-500. A decision was made for him to proceed with bariatric surgery with his history of insulin-resistant DM2 and morbid obesity. He lost 54 lbs in one year with sleeve gastrectomy and his insulin requirement decreased to 120 units daily. Above all, he had only a single incomplete session of plasmapheresis since his bariatric surgery. He has not required plasmapheresis for over a year so far and his TG levels are consistently <500 mg/dl while only on rosuvastatin 40 mg, with the most recent TG level of 182 mg/dl. **Discussion:** Bariatric surgery has shown tremendous results in terms of reversal of diabetes and other metabolic derangements. These metabolic benefits are attributed mainly to weight loss in restrictive surgeries and proposed increased GLP-1 levels with Roux-en-Y Gastric Bypass Surgery (RYGB). There are a few case reports of FPLD patients with positive outcomes in terms of metabolic profile with RYGB. In our patient, bariatric surgery was decided due to his DM2 and morbid obesity. He had an unexpected dramatic improvement in the metabolic control of his lipodystrophy. To our knowledge, this is the first case of a FPLD patient with severe HTG requiring plasmapheresis with striking metabolic improvements after sleeve gastrectomy. Gastric sleeve surgery may be an important adjunct or alternative treatment option to the current standard of therapy in patients with FPLD.

Adipose Tissue, Appetite, and Obesity
NOVEL INSIGHTS FROM THE CLINIC INTO THE DEVELOPMENT OF METABOLIC DISEASE: CASE REPORTS

Morbid Obesity Does Not Impact Mortality, Rate of Endoscopy in Patients With Biliary Acute Pancreatitis: A US Nationwide Analysis

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Introduction: Morbid obesity (MO) is associated with increased mortality in various conditions including acute pancreatitis. Interventions are challenging in patients with MO due to higher prevalence of comorbidities that may affect airway and cardiopulmonary management. Biliary acute pancreatitis (BAP) is the most common etiology for acute pancreatitis in the US. Population-based studies on the effect of obesity on biliary acute pancreatitis are lacking. This study aimed to assess the impact of MO on outcomes of patients with BAP.
Methods: Data was obtained from the Nationwide Inpatient Sample database for 2016 and 2017. Hospital discharges of patients 18 years and over with a principal diagnosis of BAP were included. This cohort was divided based on BMI into nonobese patients (BMI <30) and morbidly obese (MO) patients (BMI >/=40.0). Patients with BMI between 30.0–39.9 were excluded. Primary outcome was inpatient