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

mortality. Secondary outcomes included rate of endoscopic procedures, length of hospital stay (LOS), total hospital charges (THC), discharge diagnoses of hypocalcemia, septic shock, acute renal failure (AKI) and acute respiratory failure (ARF). Multivariate regression analysis was used to adjust for patients' sociodemographic factors, Charlson comorbidity index as well as hospital characteristics as confounders.

Results: A total of 128995 hospitalizations were principally for BAP, with 75.7% and 12.0% of these patients classified as nonobese and MO respectively. There was a significantly higher proportion of females (66.1 vs 54.5%, p<0.001) and lower mean age (50.1 vs 58.7 years, p<0.001) in patients with MO. There was no significant difference in adjusted odds of mortality (aOR=1.34, 95% CI: 0.88 - 2.03, p=0.174), or rate of endoscopy (aOR 1.00 95% CI: 0.91 - 1.11, p=0.958), in MO compared with patients who were nonobese. However, MO patients had increased mean LOS of 0.8 days (95% CI: 0.5 - 1.0, p<0.001), increased mean THC of $10760 (95% CI: 7077 - 14442, p<0.001), increased odds of hypocalcemia (aOR=1.60, 95% CI: 1.22 - 2.09, p=0.001), septic shock (aOR=2.13, 95% CI: 1.39 - 3.25, p<0.001), and AKI (aOR=1.48, 95% CI: 1.30 - 1.68, p<0.001).

Conclusion: Even though we did not find any significative difference in mortality, patients with MO appear to have and increased LOS and THC, as well as more complications like septic shock, AKI, and hypocalcemia. This calls for a greater recognition of this association for further research studies and to recognize this potential association during clinical practice.

Adipose Tissue, Appetite, and Obesity

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

Novel Heterozygous LMNA Variants Causing Familial Partial Lipodystrophy, Dunnigan Variety

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Familial partial lipodystrophy (FPLD) is a rare, mostly autosomal dominant disorder characterized by selective loss of subcutaneous fat from the extremities. Patients with FPLD are predisposed to insulin resistance, dyslipidemia, diabetes mellitus, cardiac abnormalities (coronary heart disease [CHD], cardiomyopathy and conduction system disorders) and hepatic steatosis. FPLD2 (the Dunnigan variety) is the most common subtype which is caused by heterozygous variants in the lamin A/C (*LMNA*) gene. Over 50 *LMNA* causal variants have been reported in patients with FPLD2, with p.R482W and p.R482Q comprising ~75% of the families. We report 5 novel *LMNA* variants (c.722T>C, p.L241P; c.848A>G, p.N283S; c.1396A>G, p.N466D; c.1543A>G, p.K515E; c.1744C>A, p.R582S) in 5 families, where a female proband presented to us with moderately-severe FPLD, from among a total cohort of 264 FPLD2 families, with 259 families harboring other known pathogenic *LMNA* variants. The p.L241P variant was found in a 62-year-old female with a body mass index (BMI) of 28 kg/m^2. She had hypertriglyceridemia. She is adopted and has two offsprings, who have not yet been examined and genotyped. The p.N283S variant was found in two males and two females from the same family (Age 40–74 y; BMI 18–45 kg/m^2). Of these, only the 74-year-old female proband had clinical lipodystrophy, diabetes and hypertriglyceridemia. The other three subjects did not have lipodystrophy. Thus, this variant did not segregate with the phenotype of lipodystrophy in this family likely due to low penetrance or reduced clinical expressivity. The p.N466D variant was found in a 53-year-old female (BMI 26 kg/m^2) who had diabetes and hypertriglyceridemia. The p.K515E variant was found in 4 females and 1 male who belonged to the same family (Age 29–62 y; BMI 19–26 kg/m^2). All of them had lipodystrophy and hypertriglyceridemia and three of them had diabetes. The p.R582S variant was found in 3 males and one female who belonged to the same family (Age 19–76 y; BMI 16–30 kg/m^2). All of them had lipodystrophy but only two of them had diabetes and hypertriglyceridemia. Eight of them had hypertension, three had CHD, one of them had acute pancreatitis and another one had a stroke. None of these patients had cardiomyopathy, cardiac conduction system defects or myopathy. In conclusion, we report genotype-phenotype relationship of 5 novel *LMNA* variants in patients presenting with FPLD2, with variable prevalence of diabetes, hypertriglyceridemia hypertension and CAD. None of these variants are associated with cardiomyopathy or myopathy or progeroid features. Our report adds to the allelic and clinical heterogeneity associated with *LMNA* variants.

Adipose Tissue, Appetite, and Obesity

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

Obesity Is Associated With Higher odds of Hepatorenal syndrome in Patients Admitted With Alcoholic Hepatitis: Analysis of the National Inpatient Sample (2016–2017)

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Introduction: Obesity is a significant independent risk factor for the development of liver disease. There is some available data suggesting worse outcomes of alcoholic hepatitis (AH) in obese patients however, national sample data supporting these findings are scarce. The aim of our study was to study the severity of AH in patients with concurrent obesity thus we analyzed data from the national inpatient sample.

Methods: We queried the National Inpatient Sample (NIS) 2016 and 2017 database. The NIS was searched for hospitalization of adult patients with alcoholic hepatitis as a principal diagnosis with and without Obesity (BMI = 30 and above) as a secondary diagnosis using ICD-10 codes. The primary outcome was inpatient mortality while the secondary outcomes were severe sepsis with shock, hospital length of stay (LOS), NSTEMI, hepatorenal syndrome