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Honduras. We revised information for 1690 patients divided in three categories according to their glycemic values at admission. They were classified as ‘140 mg/dl category 1, 140-180 mg/dl category 2 and ≥180 mg/dl and category 3; we excluded 352 patients with no recorded glycemic value at admission.

**Results:** A total of 1357 patients were included, 644 for category 1 with 240 (37.3%) deaths out of which 58 (24.2%) were known for diabetes and 182 (75.8%) with no previous diabetes diagnosis. In category 2 out of 261 included patients there were 111 (42.5%) deaths with 40(36.0%) and 71 (64.0%) previously known and not known for diabetes respectively. In category 3 out of 452 patients 194 (42.9%) deaths were confirmed with 142 (73.2%) in previously known for diabetes and 52 (26.8%) not previously known for diabetes.

**Discussion/Conclusion:** As reported previously in other parts of the world hyperglycemia proved to be a risk factor associated with death in patients known or not previously for diabetes. Even though it is not the only factor to be taken into consideration it has proven undoubtedly to be something that needs to be addressed upon admission for COVID 19 patients. A basal glucose measurement upon admission is fundamental in every patient and easily accessible even in underprivileged countries like ours. We found an ascending trend in increased mortality as glucose values at admission grow higher; this was true even for patients with no previous diabetes diagnosis. The registered mortality in this group of patients was about 35 to 47% higher compared to normoglycemic patients in the lowest glycemic tertile as has been found in previous publications. In the highest glycemic tertile (category 3) the mortality in diabetic patients found was 73% vrs 26% in non-diabetics that presented with extreme hyperglycemia which also suggests that chronic hyperglycemia is an important determinant.

Hyperglycemia alters pulmonary function due to the non enzymatic glycosilation of proteins of the lung generating alveolar capillary microangiopathy causing the accumulation of collagen in the extra cellular matrix thus generating restrictive lung disease.

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Abstract #1185180

**Case of Maturity Onset Diabetes of Young-Type V**

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**Introduction:** Maturity Onset Diabetes of Young (MODY) is a rare cause of diabetes and accounts for 1-5% of cases and has an autosomal dominant mode of inheritance. Diagnostic criteria set forth for MODY include 1. Onset before 25 years of age in one family member 2. Presence of DM in two consecutive generations 3. Absence of β-cell autoantibodies 4. Sustained endogenous insulin secretion.

**Case Description:** A male university student, aged 21 years, presented with uncontrolled DM. He was incidentally diagnosed with a case of T2D three years ago while undergoing pre-operative assessment for a right inguinal hernia repair and orchidopexy. He had a history of decreased shaving frequency (every 15 days) and no morning erections. There was a strong family history of DM, both from maternal and paternal sides.

He was obese with a BMI of 31.8 kg/m² and had normal vital signs. On genital examination, the right testicle was undescended, while the left was present in the scrotum with normal size and consistency. There was no evidence of gynecomastia.

His first HbA1C from three years ago was 12.9% and subsequent levels were 6.4%, 10.0%, and 6.3%. C-peptide was normal (3.8 mg/ml), insulin level was also normal (13.0 mU/L), and a high HOMA-IR of 3.21. His LH and FSH were 5.82 mU/ml and 2.06 mU/ml respectively. Serial testosterone levels were checked; showed fluctuating levels between 260.0 - 479.0 ng/dl. TSH and prolactin were normal; he also had hyperuricemia (7.8mg/dl) and hypomagnesemia (1.5 mg/dl). Liver function tests revealed raised ALT (51 U/L) and GGT (91U/L). His semen analysis was done twice and showed severe asthenospermia. His right testis measured 2.2x1.1cm, was located in the right inguinal canal, while the left scrotal testis was 4.1x3.1 cm, on ultrasound. It also showed distended epididymis and vas deferens. CT scan abdomen revealed fatty liver, normal kidneys but absent body and tail of pancreas representing partial agenesis of the pancreas. Genetic testing could not be done due to resource constraints.

Lifestyle Modification was advised along with sitagliptin, metformin, and glimepiride. He was referred to a urologist for removal of undescended testis and biopsy of scrotal testis. Counseling was done for fertility issues.

**Discussion:** At diagnosis, MODY cannot be distinguished easily from T1D & T2D based on clinical characteristics alone. T1D mostly differs from MODY in terms of disease etiology, as the pathogenesis of MODY does not involve pancreatic β-cell autoimmunity. Patients usually maintain β-cell function, & their diabetes is well-controlled with no or low-dose insulin for at least 5 years after diagnosis, as in our case. To date, at least 14 different gene mutations are associated with MODY. Among them, HNF-1B (previously called MODY 5), is associated with developmental renal disease, especially cysts, genitourinary malformations, gout, and pancreatic insufficiency. Additionally, these patients can have elevated liver enzymes, hyperuricemia, and hypomagnesemia. On the basis of these features, a diagnosis of MODY 5 was made in our patient.

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Abstract #1185190

**New Onset Autoimmune Diabetes Associated with Acute SARS-COV-2 Infection**

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**Introduction:** Infection with SARS-CoV-2 has been shown to cause complications affecting nearly all organ systems of the human body. Here, we outline a case of SARS-COV-2 associated with new onset of autoimmune diabetes.

**Case Description:** A 62-year-old female with past medical history of class III obesity, primary hypothyroidism, obstructive sleep apnea, and endometrial cancer established care with a multidisciplinary bariatric team in March 2021. This team included a dietitian and psychologist to promote healthful lifestyle intervention with the intent to undergo bariatric surgery in December 2021. At a follow up visit in September 2021 her HbA1c was 6.7% (normal < 5.7 %) and she was diagnosed with type 2 diabetes treated with healthful lifestyle. After lifestyle modification the patient
successfully lost 40 pounds. In December 2021, she presented to the ED (Emergency Department) complaining of fatigue and neuropathy. She was found to be hyperglycemic with glucose 369 mg/dL (normal 70-100 mg/dL). β-hydroxybutyrate was 32.1 mg/dL (normal 0.20-2.81 mg/dL) and anion gap was 10 mmol/L (normal 3-13 mmol/L). She was resuscitated with fluid and referred urgently to Endocrinology. One week later, she was seen in the office by her endocrinologist for initial consultation. She was acutely complaining of anosmia and ageusia and found to be positive for acute SARS-COV-2 infection. Bloodwork revealed an increase in HbA1c to 13.9 %, fasting glucose 303 mg/dL (normal 70-100 mg/dL), normal C-peptide 1.6 ng/dL (normal 0.5-3.3 ng/dL), elevated GAD antibody 154.3 IU/mL (normal 0-5 IU/mL), elevated anti-islet Cell antibody IgG ratio 1:64 (normal < 1: 4), elevated anti-Islet Antigen 2 antibody >120 U/mL (normal 0–7.4U/mL), and elevated anti-Zinc Transporter 8 antibody 500 U/mL (normal 0–15 U/mL). Patient was diagnosed with autoimmune diabetes associated with acute SARS-COV-2 infection and was started on basal-bolus insulin with improvement in her hyperglycemia. She did not require hospital admission or steroid treatment for SARS-COV-2 infection.

**Discussion:** Although viral infections are associated with type I diabetes related autoimmunity in children, this case study is unique regarding its mechanism in association with SARS-COV-2 infection. Potential mechanisms underlying onset of diabetes in patients with SARS-COV-2 infection are still under investigation. One potential mechanism involves pancreatic beta cell dysfunction with diminished insulin secretion due to a systemic inflammatory cascade. This case is unique in as the patient’s C-peptide was still detectable indicating intact beta cell function. Furthermore, the patient’s diabetes paradoxically worsened after a more healthful lifestyle and 40-pound weight loss. This patient’s case of autoimmune diabetes illustrates the need for further research into the mechanisms underlying the onset of diabetes after SARS-COV-2 infection.

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Abstract #1185191

**Acquired Generalized Lipodystrophy Presenting As Severe Insulin Resistance**

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**Introduction:** Roughly 100-150 cases are reported in the literature of acquired generalized lipodystrophy (AGL). This rare disorder is characterized by leptin deficiency resulting from low adipose mass and metabolic derangements such as hypertriglyceridemia and severe insulin resistance. These metabolic derangements lead to increased risk for pancreatitis, nonalcoholic fatty liver disease, and adverse cardiovascular events. The most common progression of AGL is onset of symptoms in childhood or adolescence with progression of complications into adulthood. Very few cases of this rare disease have been reported as adult-onset presenting with severe insulin resistance.

**Case Description:** A 62-year-old female presented with severe, progressive insulin resistance, persistent hyperglycemia, thin extremities and unintentional weight loss for 6 months. She denied polydipsia, neuropathy, abdominal symptoms, or any vision abnormality. History was remarkable for type 2 diabetes, hypertension and severe dyslipidemia. Vital signs stable. The patient was taking three oral anti-diabetic agents including metformin, dapagliflozin and dulaglutide. In addition to triple therapy, this patient was on 60 units of short-acting insulin total for mealtime coverage and 45 units twice a day of long acting insulin. On exam, she was thin appearing and in no distress. Labs revealed C-Peptide 4.58 ng/mL and insulin 1.5 uU/mL. Patient had a HgA1c 18.5 %, GAD antibody negative, islet islet cell antibody negative, and TSH 1.414 uU/mL. Although the patient was initially being labelled as noncompliant, they repeatedly endorsed taking all medications as prescribed without improvement of objective labs. Due to escalating insulin requirements and progressive metabolic derangement, AGL was suspected as the underlying disease due to physical exam findings and persistently escalating insulin requirements. In order to confirm the diagnosis, leptin level ordered with result of 2.2 ng/mL, which is low. In the absence of antibody positivity, the likely diagnosis was idiopathic AGL. After trial of lifestyle modifications, patient followed up with AGL specialist for metreleptin initiation, which is a recombinant human leptin analog. Subcaneous administration of this in AGL patients with hypoleptinemia has been shown to improve blood glucose levels, triglycerides, and complications such as steatosis and coronary artery disease.

**Discussion:** This case represents an exceedingly rare diagnosis of AGL associated with severe insulin resistance in an adult. In this case, hypoleptinemia confirmed the clinical findings of AGL. Although there are few cases diagnosed each year, it is important to consider this diagnosis where clinical findings such as thin extremities and lipodystrophic fat distribution correlate with severe insulin resistance. If further workup was not performed for this patient, they would be labelled as noncompliant and not receive the necessary treatment for their disease. Research is still required on the efficacy of metreleptin in reducing morbidity and mortality in the adult population due to the exceedingly rare diagnosis in adulthood.

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Abstract #1185206

**Latent autoimmune diabetes of adulthood: A challenge to diagnose**

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**Introduction:** Latent autoimmune diabetes of adulthood (LADA) is often a challenging diagnosis as the age of onset and the initial period of insulin-independence point towards type II diabetes (T2DM). Its reported prevalence is about 2%-12% of all cases of adult diabetes. Similar to pediatric-onset diabetes the autoimmune response is generated by glumatic acid decarboxylase 65 (GAD65) antibodies or islet cell antibodies. Herein we describe the case of a middle-aged female with sudden onset diabetes who was diagnosed with LADA.

**Case Description:** A 53-year-old female with Hashimoto’s thyroiditis and benign thyroid nodule presented with labwork from her primary care physician which revealed an HbA1c of 6.5%. She was complaining of increased exhaustion but otherwise denied polyuria, polydipsia, increased thirst, and increased hunger. Her vital signs and physical exam revealed no abnormalities. Her BMI was 23.46 in/lb. She was counseled in lifestyle modification in an attempt to control her diabetes without medication. When she returned for follow-up blood work her HbA1c had increased to 7.2% despite having lost more than five pounds and strict diet control. At S53