(weight<90kg-1mg; ≥90kg-1.5mg) stimulation test was done in follow-up to assess GH and cortisol axes.
Results: Among 30 (out of 50) patients who turned up for follow-up, GH, cortisol and TSH deficiency was noted in 50% (n-15), 13.3% (n-4) and 3.2% (n-1) of subjects, respectively. Pan-hypopituitarism characterised by deficiency of at least two hormonal axes was seen in 16.7% (n-5) patients.
Conclusion: Significant delayed hypopituitarism especially GH deficiency was seen among patients of heart block.
Implications: This novel finding if reproduced in subsequent studies will result in unearthing of a significant etiology of hypopituitarism which could potentially become the most common cause of acquired adult onset hypopituitarism considering the huge burden of patients with heart block. Heightened cardiovascular risk due to hypopituitarism in these patients may be reversed by active screening and replacement of deficient hormones. **References:** (1) Simsek, Kaya, Tanriverdi et al. (2014) Evaluation of long-term pituitary functions in patients with severe ventricular arrhythmia: a pilot study. J Endocrinol Invest 37:1057-1064Keywords: Cortisol, GH deficiency, Heart block, Hypopituitarism, Pituitary **Disclosure:** Authors have nothing to disclose

Adrenal

ADRENAL CASE REPORTS I

Allgrove Syndrome: How to Suspect a Problem?

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SAT-219
Background: Allgrove syndrome (triple A syndrome) is a rare autosomal recessive multisystem disease characterized by adrenal insufficiency, alacrimia and achalasia. It is caused by a mutation in the AAAS gene (12q13) encoding the protein ALADIN (1). This syndrome is often associated with neurological dysfunction disorders, amyotrophy, in such cases, it is named 4A and 5A syndrome, but sometimes there is also 2A syndrome. Prevalence:<1/1000000. The first description was in 1978. **Clinical case:** A 18-year patient A. complained of fatigue, weakness, darkening of the skin. From anamnesis of life: born from the first pregnancy without complications, weight 3200g. Parents often turned to the pediatrician with complaints: lethargy, frequent regurgitation, ARVI up to 6–7 times a year. Slow weight gain, dyspeptic syndrome (nausea, vomiting) was noted objectively. At the age of 3, the boy entered the surgical department with acute abdomen, fever, vomiting. Achalasia was revealed, reconstructive surgery was carried out. In the diagnostic search for the causes of body weight loss he was directed to the endocrinologist. There were an increase in ACTH 470 pg/ml (0,0-46 pg/ml), cortisol 0.05 μg/DL. Diagnosis: primary chronic adrenal insufficiency; the dose of hydrocortisone 10 mg/day did not change with age. An in-depth anamnesis found: the patient never cried with tears. Objectively: asthenic body type, BMI 16.5 kg/m2, hyperpigmentation of the palms, armpits; weakness in the proximal muscles of the limbs. Laboratory studies: ACTH 95 PG/ml, cortisol 0.1 μg/DL (3.7–19.4 μg/DL). The secretion of mineralocorticoids was evaluated: plasma aldosterone and renin levels were within the reference values. Ophthalmologist: injected conjunctiva, sclera. Schirmer's test: mild alacrimia. It allowed to make the diagnosis: "Primary chronic adrenal insufficiency. Condition after surgery for achalasia (1997). Alacrimia. Allgrove Syndrome." The dose of hydrocortisone was increased to 17.5 mg/day. In 2019, the patient complained of a sharp deterioration of health, darkening of the skin. The dose of hydrocortisone was increased to 25 mg/day (15 mg at 8.00, 10 mg in the afternoon). The ophthalmologist noted an increase in the severity of alacrimia, artificial tear drops was recommended. The diagnosis was confirmed by pathogenic mutation c.43C>T of the AAAS gene. **Discussion:** Despite the full clinical picture, the right diagnosis was made only after 14 years. We shown the difficulty of diagnosis is due to the lack of awareness of clinicians about the disease, the importance of interdisciplinary interaction, as well as the need for follow-up of such patients. **Reference:** (1) Handschug K, Sperling S, Yoon SJ, et al. Triple A syndrome is caused by mutations in AAAS, a new WD-repeat protein gene. Human Molecular Genetics. 2001;10:283–290.

Diabetes Mellitus and Glucose Metabolism

DIABETES COMPLICATIONS II

Emphysematous Gastritis and Diabetes

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MON-688
Background: Emphysematous gastritis (EG) is a rare and severe form of gastritis of infectious origin. Diabetes is an important underlying risk factor as it leads to a systemic predisposition to infections. Other risk factors include long term steroid use, nonsteroidal anti-inflammatory (NSAID) use, alcohol use, corrosive ingestion, and pancreatitis, all of which disrupt gastric mucosa. First described in the 1800s, it is characterized by the presence of air in the stomach wall and differentials for these cases include gastric emphysema and cystic pneumatosis both of which are non-infectious in origin.
Clinical Case: A 57-year-old male with type 2 diabetes presented with a one-day history of abdominal pain, non-bloody diarrhea, and vomiting. One day prior to presentation, he developed diarrhea which was followed by episodes of projectile vomiting reported as orange-tinged with mucus. On the day of admission, he was afebrile, tachycardic in 120s with stable blood pressure. Laboratory evaluation was significant for leukocytosis at 18.8 k/uL (4.3–11.3 k/uL) and lactic acidosis 2.37 mmol/L (0.7–2.1 mmol/L). Abdominal examination was notable for soft abdomen with diffuse tenderness to deep palpation without rebound or guarding. Further workup with Computed Tomography (CT) was concerning for emphysematous gastritis with air in the gastric vein, splenic vein, and portal vein. Given hemodynamic stability and benign abdominal examination, medical management was initiated. He was