management. DKA may lead to glucose and lipid metabolism dysregulation which can result in hypertriglyceridemia leading to AP. On the other hand, hypertriglyceridemia may induce AP which may decompensate diabetes and lead to DKA. In both scenarios, this triad results in an uncommon clinical presentation with up to 80% mortality rate. Most frequently reported in children, this entity accounts for only a handful of cases reported in the literature.

Case of an obese, non-alcoholic 57 year old male without history of systemic illness who visits the emergency room due to mid-upper abdominal pain for the past day. Pain radiates to the back, worsens upon lying flat, and is associated with bloating and nausea. He denies previous similar episodes, vomiting, fever or bowel habit changes. Laboratory workup revealed lipemic sample with hyperglycemia, metabolic acidosis, positive serum ketones, and normal amylase and lipase. Lipid panel revealed hypertriglyceridemia at 6,260 mg/dL (35-150). Glycated hemoglobin at 14.7%. Abdominal computed tomography showed peripancreatic inflammation consistent with pancreatitis. Given clinical and imaging criteria the diagnosis of severe hypertriglyceridemia induced AP and DKA were made. The patient was admitted to ICU and treated with intravenous insulin drip and supportive management. Resolution of DKA and successful decrease in triglycerides to less than 500 mg/dL was achieved by the third day of admission. After six days, the patient was discharged home with insulin and lipid lowering regimens.

This case demonstrates an extremely rare initial presentation of diabetes mellitus. This triad is the result of a toxic chain of events that may be lethal if not promptly identified. This case makes an exemplary lesson as to always take under consideration atypical etiologies to potentially life threatening conditions and also remarks that while uncommon, pancreatitis with normal pancreatic enzymes is a possible phenomenon. Even though negative assays have been associated with hypertriglyceridemia induced AP, only a few cases with negative lipase have been described. While no definite explanation has been yet discovered, negative lipase may be explained by early acinar cell apoptosis in AP. More research efforts are necessary in order to improve early diagnosis, treatment, and mortality rate for this rare but potentially deadly triad and to better understand the mechanisms underlying AP and the role that digestive enzymes play.

**Adrenal**

**ADRENAL CASE REPORTS II**

**Pseudohypoaldosteronism Presenting with Salt Wasting Crisis**  
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**SUN-184**  
A case of Pseudohypoaldosteronism presenting with salt wasting crisis  
Pseudohypoaldosteronism (PHA) is due to end organ resistance to mineralocorticoids. It is usually inherited in an autosomal recessive or autosomal dominant pattern, and rarely can a result of the mutation de novo. Zennaro MC, Hubert EL, Fernandes-Rosa FL. Aldosterone resistance: structural and functional considerations and new perspectives. Mol Cell Endocrinol. 2012;350:206-15.10.1016/j.mce.2011.04.023[Crossref], [PubMed], [Web of Science ®] It can be sub-classified into two forms PHA type 1 A involving the kidneys or PHA-1 B which effects
and showed that the patient had hyperkalemia and hypertension. Case: Our patient is a 5 week old male who admitted for significant electrolyte abnormalities. He was followed by PCP for failure to thrive. The child was referred to ED with increased difficulty in feeding, lethargy and episodes of emesis. In the ED, the child was in a compensated shock and had a low normal BP: 76/35, HR: 169/min and fast breathing R/R: 80/minute and afebrile. P.E. showed signs of dehydration. Lab work showed: Na: 110 mEq/L, K: 9.3 mEq/L, low Chloride and Ca: 11.1 mEq/L. Endocrinology recommended IVF supplementation with 2 x NS bolus followed by IVFs at 1.5 times maintenance (D5 + NS), along with administration of Florinef 0.2 mg suspecting CAH. Renin, 17-OHP, random cortisol level and thyroid hormone levels were ordered. Results showed: TSH of 5.30 mcIU/mL and Free T4 of 2.2 ng/dL. Cortisol: 20.5 mcg/dL. He was subsequently admitted to PICU. Septic work-up was negative. He became hemodynamically stable after hydration and did not require the stress dose of hydrocortisone. Repeat Na: 133 mEq/L, K: 4.7 mEq/L, Cl: 102 mEq/L and Glucose: 90 mg/dL. ACTH stimulation test for CAH evaluation was performed, stimulated 17OH: 88 ng/dl, cortisol: 27.1 mcg/d and normal DOC. Elevated Aldosterone: 632 ng/dl (5-80) and renin level: 351 (6.5-86). A diagnosis of PHA was made and Florinef was stopped and the child was started on NaCl supplementation which normalized the electrolytes. Genetic testing was negative for NR3C2, CUL3, KLHL3, SCNN1A, SCNN1B, SCNN1G, WNK4, WNK1, CUL3, KLHL3, SCNN1A, SCNN1B, SCNN1G, WNK4 and showed that the patient is a heterozygous for a variant of unknown significance, c.6276T>A (p.Ser2092Arg) in the WNK1 gene. However, the patient did not have hypertension and urine electrolytes were also normal did not show signs of PHA 2. Conclusion: PHA can present with severe salt wasting crisis. It can be diagnosed clinically. The relationship of mutation and phenotype can be elusive. Course was uncomplicated and he was discharged from the PICU in 6 days. Sodium doses were titrated based on serum levels with eventual dose of 22.5 mEq/kg/day and sodium level was 139 mEq/L.

Bone and Mineral Metabolism
NEW INSIGHTS INTO PTH AND CALCIUM RECEPTOR SIGNALING

Is Urinary Calcium the Only Predictor of Nephrolithiasis in Patients with Asymptomatic Primary Hyperparathyroidism?

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The 4th International Workshop for the management of asymptomatic PHPT included, among the criteria for parathyroidectomy, the presence of hypercalciuria (dUCA> 400 mg/day) and increased biochemical stone risk profile. The aim of the present study was to evaluate the biochemical stone risk profile in 176 consecutive patients (143 females and 33 males) with asymptomatic PHPT. We recorded clinical and biochemical data, including 24 hours urinary measurements of the following parameters: volume and pH, creatinine, calcium, magnesium, sodium, potassium, ammonium, uric acid, oxalate, citrate, phosphate, inorganic sulphate and chloride and kidney ultrasound. In our cohort dUCA> 400mg/day showed a low sensitivity and positive predictive value (PPV) for nephrolithiasis with high specificity (46.2, 32.7, 73.0% respectively), while hypercalciuria by 4 mg/kg/bw (d-UCA>4mg/kg) had a high sensitivity, with low PPV and specificity (79.5, 27.7, 40.1%) Daily hypomagnesuria (d-HypoMg), but not any other urinary parameter, was an independent predictor of nephrolithiasis in the univariate (OR 2.97 CI 1.27-7.09 P=0.014) and multivariate analyses adjusting for age, sex, BMI, and eGFR (OR 5.17 CI 1.17-8.42 P=0.02). d-HypoMg was relatively lower in the regression analysis with urinary calcium in patients with nephrolithiasis compared with those without. The mean ratio between (dUCA) and (dUMg) was higher in patients with nephrolithiasis compared with those without (4.6±2.0 vs 3.3±4.1, P<0.001). In the univariate and multivariate analyses the dUCA/dUMg ratio was a significant predictor of nephrolithiasis [OR 4.9 (2.3-10.5); P<0.001; OR 5.3 (2.4-11.6), P<0.001, respectively]. The AUC using the dUCA/dUMg ratio as variables was 0.69 (CI 0.60-0.79; P<0.0001). The best cut-off value, set at the highest Youden index, was equal to 4.0, with a sensitivity of 59.0% and a specificity of 77.4%. In patients with hypercalciuria (>400 mg/24-hour) dUMg was positively correlated with dUCA in those without nephrolithiasis (r=0.50, β=0.2, P=0.002) but not in those with nephrolithiasis (r=0.05, β= 0.014; P=0.8). In patients without hypercalciuria we found that hypomagnesuria remained a predictor of nephrolithiasis using either 400 mg/dio (P=0.002, OR 5.12 (1.84-14.24) or 4 mg/kg bw (P=0.014, OR 6.24 (1.45-26.8). Moreover, the OR for nephrolithiasis improved using the combination of d-HypoMg with d-UCA>4mg/kg (OR 8.12, CI 1.92-34.18, P=0.004), but not with dUCA> 400mg/day. The current urinary calcium threshold of >400 mg/24-hour has a low sensitivity in detecting nephrolithiasis; our data suggest that sensitivity, specificity and positive predictive value could be improved including dUMg, dUCA/dUMg ratio and the combination of d-HypoMg with d-UCA>4mg/kg in the stone risk evaluation.

Diabetes Mellitus and Glucose Metabolism

TYPE 2 DIABETES MELLITUS

Does Short Term Intensive Insulin Therapy in Newly Diagnosed Type 2 Diabetes Mellitus Delay Eventual Insulin Dependence

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In patients with type 2 diabetes mellitus (T2DM), dysfunction of β-cells starts years before the diagnosis of T2DM and rapidly worsens after overt hyperglycemia. Use of short-term intensive insulin therapy (STIIIT) at the time of diagnosis of overt hyperglycemia has shown clinical recovery