Introduction: Virilization in a postmenopausal woman requires evaluation for an androgen-secreting tumor. The differential diagnosis includes adrenal carcinomas and adenomas and Sertoli-Leydig cell tumors, granulosa-theca cell tumors, and hilus-cell tumors of the ovaries. We present a case of virilization in a postmenopausal woman caused by a Sertoli-Leydig cell tumor (SLCT) in which evaluation was complicated by the pattern of androgen elevation, bilateral adrenal nodules, and absence of an adrenal mass. Case: A 64-year-old female was referred for evaluation of hyperandrogenism. Hirsutism, temporal hairline regression, and unusually deep voice were noted on examination. Two total testosterone levels obtained one month apart were 146 ng/dL (2.45), and measurements of dehydroepiandrosterone sulfate (DHEAS) and androstenedione were 299 mcg/dL (12.133) and 1.84 ng/mL (0.130-0.820), respectively. Abdominal CT revealed bilateral adrenal nodules - 2 cm and -5 Hounsfield units (HU) on the left, and 1.5 cm and 5 HU on the right - but no ovarian masses. Transvaginal ultrasonography also failed to identify a discrete ovarian mass but showed endometrial hyperplasia. Virilization, magnitude of testosterone elevation, and results of imaging were felt to be most strongly indicative of ovarian hyperthecosis, and the patient underwent laparoscopic bilateral salpingo-oophorectomy and hysterectomy. The right ovary was 2.3 cm in largest diameter and approximately 90% replaced by an orange-red mass that showed Sertoli and Leydig cells on microscopy, immunohistochemical staining for the sex cord proteins inhibin and calretinin, and presence of the Leydig cell marker melan A. It was classified as well differentiated. Additional CT imaging and robotic assisted laparoscopy confirmed a stage IA tumor. One month after surgery, hyperandrogenemia had completely resolved (total testosterone <10 ng/dL, androstenedione 0.379 ng/mL, and DHEAS 99 mcg/dL), and changes of virilization had mostly regressed at an eight months appointment. Discussion: SLCTs are a type of sex-cord stromal ovarian tumor. They constitute < 0.5% of ovarian tumors but account for approximately 75% of testosterone-secreting ovarian masses. This patient’s case was unusual for multiple reasons: 1. Age - most SLCTs are diagnosed in the second or third decade, 2. Imaging - CT and ultrasonography usually show a solid or solid and cystic adnexal mass, and co-existing adrenal nodules are rare, likely due to typical young age of presentation, and 3. Pattern of androgen elevation - DHEAS was more than two-fold elevated, and usually <10% of DHEA and DHEAS are produced by the ovaries. However, DHEAS fell significantly after oophorectomy. SLCTs are a potential etiology of virilization in postmenopausal women even in the absence of a detectable adnexal mass and when biochemistries and imaging raise the possibility of an adrenal source of androgen.
Introduction: Diabetic ketoacidosis (DKA) is a life-threatening medical emergency requiring urgent treatment. Euglycemic DKA may occur in patients with both type 1 and type 2 Diabetes Mellitus (DM), as well as pregnancy. The absence of marked hyperglycemia can result in delayed diagnosis and treatment, resulting in potential adverse outcomes. Diabetes is a major comorbidity associated with severe hospital course and high fatality rate among patients with COVID-19 infection. We report our experience in a patient with gestational diabetes mellitus who developed euglycemic DKA and COVID-19 infection in her third trimester of pregnancy. Clinical Case: A 30-year-old lady at 29 weeks gestation presented with two-day history of vomiting, diarrhea and abdominal pain. She reported good fetal movements. She had been diagnosed with Gestational Diabetes Mellitus (GDM) at 20 weeks gestational age, receiving treatment with multiple daily injections of insulin. 5 days earlier, she had tested positive for COVID-19 infection. She was asymptomatic; testing was performed as she had been in contact with a confirmed case. On examination she was afebrile and vitally stable, but dehydrated. Her initial laboratory investigations showed ketonemia with normal glucose level and normal anion gap. She was treated as a case of starvation ketosis and dehydration, with intravenous fluids and electrolyte replacement. However, 3 days later, the patient complained of worsening nausea and vomiting with dry cough and she developed hypotension. Chest X-ray showed bilateral mid and lower zone pulmonary infiltrates. She was treated as COVID-19 pneumonia, received 2 units of COVID-19 convalescent plasma and broad-spectrum intravenous antibiotics. Repeated investigations showed worsening ketosis with high anion gap metabolic acidosis, consistent with a diagnosis of euglycemic DKA. Insulin infusion was initiated, isotonic saline with electrolyte replacement was also continued. She symptomatically improved over the next two days, with resolution of ketonemia and acidosis. The patient was discharged and she was well at her outpatient follow up visit. She underwent emergency Cesarean Section at 37 weeks gestational age, due to non-reassuring electronic fetal monitoring. She delivered a healthy female infant weighing 2445 grams. Conclusion: Pregnancy is a high-risk period for DKA particularly when associated with other stressors that were identified in our patient – GDM, restricted calorie intake and COVID-19 infection. Diabetes is a risk factor for developing severe forms of COVID-19 and on the other hand, COVID-19 infection is associated with poor glycemic control and higher risk of hyperglycemic emergencies including ketoacidosis in diabetic patients. Prompt recognition of euglycemic DKA is critical in pregnancy, as this condition is associated with high fetal mortality rates.

Reproductive Endocrinology

REPRODUCTIVE HEALTH CASE REPORTS

Hypomagnesemia and Hypokalemia: The Possible Role of Vasopressinase in This Rare Presentation of Gestational Diabetes Insipidus

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Background: Gestational diabetes insipidus (GDI) is a rare but recognized complication of pregnancy. We are reporting the case of a 33-year-old patient who presented in her 3rd trimester with GDI, severe asymptomatic hypokalemia & hypomagnesemia. This presentation of GDI has not been reported yet to our knowledge.

Clinical Case: A 33-year-old G2P1 patient who presented to the hospital due to the discovery of new onset severe asymptomatic hypokalemia on routine labs. Further evaluation revealed K of 2.8 mEq/L, Mg of 1.4 mg/dL & Na of 142mEq/L. She endorsed polyuria & polydipsia and a 24-hour urine collection of 9.35L confirmed diabetes insipidus. Labs also revealed a serum osmolality of 286mOsm/kg & urine K of 15 mEq/L. Her urine osmolality was inappropriately low at 138mOsm/kg. She received K & Mg supplementation. In pregnant patients suspected of having GDI who have normal serum Na, water restriction test can be done for further evaluation. It must performed with close monitoring because dehydration can lead to uteroplacental insufficiency. Given her history of previous fetal demise & report of headaches with attempts of water restriction, we opted not to do the test. She was given subcutaneous 1 mcg of desmopressin overnight with her thirst & polyuria improving only briefly. Due to her suboptimal response, we began her on 10 mcg of intranasal spray of desmopressin once in the evening. Her symptoms resolved on this regimen after a few days of observation. Her Mg & eventually K levels reached and remained at normal levels after repletion. The rest of her pregnancy went on without complications and she delivered a healthy male infant via scheduled cesarean section at 36 weeks gestation. She discontinued intranasal desmopressin after 2 weeks and has remained asymptomatic. Labs at follow up 4 weeks later remained normal. It is known that vasopressinase which is made by placental trophoblasts during pregnancy degrades endogenous ADH1. We are hypothesizing that since ADH has been shown to be effective in conserving magnesium in rats by stimulating its uptake in the distal convoluted tubule cells2, a similar mechanism could be present in humans. We believe that the loss of endogenous ADH, leads to the depletion of Mg and consequently hypokalemia in susceptible patients.

Conclusion: To our knowledge this is the first case presenting the possible role of vasopressinase in the development of hypomagnesemia & hypokalemia in a patient with GDI.

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