

Title: Gestational Diabetes Insipidus, a Rare Presentation in the Postpartum Period: A Case Report

Authors: Margaret Ligon, DO¹, Himani Patel², Jace DeGarmo², Anthony Royek, MD¹

Affiliations: 1. Memorial Health University Medical Center, Department of Obstetrics and Gynecology, 2. Mercer University School of Medicine

Background/Synopsis:

There are many physiologic changes to the renal and urologic system during pregnancy which can interfere with prompt recognition and treatment of certain diagnoses. These changes include decreased serum osmolality, decreased plasma sodium levels, and thus decreased hypothalamic threshold for antidiuretic hormone production in order to maintain normal urine concentrating ability. It is also known that the placentally-secreted enzyme vasopressinase degrades ADH and if the breakdown of ADH by this enzyme overwhelms hypothalamic and pituitary production the result is decreased serum ADH with resultant inability of the kidneys to properly concentrate urine. This phenomenon, first identified in 1984, was termed gestational diabetes insipidus (DI), which is notably different from both central and nephrogenic causes of DI as it is due to vasopressinase excess rather than decreased production and/or response to secreted ADH. Almost all reported cases have been noted in the late 3rd trimester, and often these cases are diagnosed during labor and delivery as patients are anesthetized and on restricted oral intake which interferes with the ability to maintain serum osmolality from excessive PO hydration. Rarely, these cases are seen in the postpartum period as most resolve with delivery.

Objective/Purpose:

DI is a rare complication of pregnancy, affecting approximately 1 in 30,000 pregnancies (Ananthakrishnan 2009). This case offers the opportunity to review normal physiology of pregnancy and recognize/treat this serious, life-threatening metabolic derangement in a multidisciplinary manner.

Case Report:

A 41-year-old multigravida woman (G6P5005) at 35 weeks and 4 days of gestation was transferred to a tertiary care center due to concerns for acute fatty liver of pregnancy. Her pregnancy was complicated by insulin-dependent type 2 diabetes mellitus, advanced maternal age, and grand multiparity. She reported three days of worsening nausea, vomiting, and abdominal pain. Initial labs revealed AST/ALT levels >600 U/L, serum creatinine 1.70 mg/dL, and normal platelets. Abdominal ultrasound showed hepatic steatosis and cholelithiasis without signs of acute cholecystitis.

On arrival, she remained normotensive but symptomatic with persistent vomiting and upper abdominal tenderness. Laboratory evaluation confirmed transaminitis, mild thrombocytosis, and worsening renal function. The fetus was in transverse lie. Given her clinical presentation and abnormal labs, a presumptive diagnosis of severe preeclampsia was made, and a primary low transverse cesarean section was performed on hospital day 1.

Postoperatively, she initially recovered well. However, on postoperative day 1, she developed worsening nausea, emesis, and reported new right upper quadrant pain and generalized weakness. Despite IV fluids, her urinary output exceeded 10 liters over 24 hours. Her serum sodium rapidly rose from 143 to 175 mmol/L in under 12 hours. She developed altered mental status with confusion and asymmetric pupils. The emergency response team was contacted, and the patient was transferred to the ICU.

In the ICU, she was diagnosed with hypernatremia secondary to central diabetes insipidus. She was managed with IV D5W at increasing rates and ultimately transitioned to a desmopressin infusion. Nephrology and endocrinology were consulted. Brain MRI revealed an enlarged pituitary gland with T2 hyperintensity involving the infundibulum and surrounding structures, consistent with inflammation or ischemia. Hormonal workup remained within normal limits, and there was no evidence of panhypopituitarism or Sheehan's syndrome.

With aggressive medical support, her sodium levels normalized, urine output stabilized, and mental status returned to baseline. She was later transitioned to subcutaneous desmopressin, which was tapered prior to discharge. Her renal function and liver enzymes improved, and she was ultimately discharged in stable condition with endocrinology follow-up.

Conclusion:

This case highlights the importance of recognizing postpartum DI, particularly in patients with hypertensive disorders of pregnancy and hepatic dysfunction.

References:

Ananthakrishnan S. Diabetes insipidus in pregnancy: etiology, evaluation, and management. *Endocr Pract.* 2009;15(4):377-382. doi:10.4158/EP09090.RA

Goldrich A, Yuan J, Stohl H. Postpartum gestational diabetes insipidus related to preeclampsia: A case report. *Case Rep Womens Health.* 2023;37:e00487. Published 2023 Feb 14. doi:10.1016/j.crwh.2023.e00487

Krege J, Katz VL, Bowes WA Jr. Transient diabetes insipidus of pregnancy. *Obstet Gynecol Surv.* 1989;44(11):789-795.

Lindheimer, M. D. (2005). Polyuria and Pregnancy: Its Cause, Its Danger. *Obstetrics & Gynecology*, 105 (5), 1171-1172. doi: 10.1097/01.AOG.0000162538.95869.05.

Brewster, U. C. & Hayslett, J. P. (2005). Diabetes Insipidus in the Third Trimester of Pregnancy. *Obstetrics & Gynecology*, 105 (5), 1173-1176. doi: 10.1097/01.AOG.0000161811.02155.68.

Sherer DM, Cutler J, Santoso P, Angus S, Abulafia O. Severe hypernatremia after cesarean delivery secondary to transient diabetes insipidus of pregnancy. *Obstet Gynecol.* 2003 Nov;102(5 Pt 2):1166-8. doi: 10.1016/s0029-7844(03)00704-x. PMID: 14607044.