

in patients with acromegaly, which should be part of the treatment of childbearing patients with any pathological condition.

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Diabetes insipidus induced by pregnancy. A case report[☆]

Diabetes insípida inducida por el embarazo. Comunicación de un caso

Diabetes insipidus (DI) is a rare endocrine disorder reported to occur in one per 25,000–30,000 people in the general population.¹ The occurrence of this syndrome in pregnancy is even rarer, and only a limited number of cases of DI have therefore been reported during pregnancy. DI may be the continuation of the syndrome discovered before pregnancy, or may first occur during pregnancy and subsequently disappear.²

We report the case of a 16-year-old female patient, a primigravida with no significant clinical history regularly attending antenatal visits. No medical problems were reported until the 38th week of pregnancy, when she was found to have high blood pressure (162/102 mmHg) associated with headache, epigastric pain, and proteinuria (650 mg/day). Laboratory test results were normal, except for a serum creatinine level of 151 mmol/L. An intravenous dose of clonidine was administered, and a magnesium sulfate infusion was started to stabilize the patient, after which a cesarean section was performed for fetal distress, delivering a live newborn weighing 3400 g.

A persistent increase in urine output (900 mL/h) and a urine specific gravity of 1005 were seen despite fluid restriction to 80 mL/h. The patient reported severe polydipsia. On further questioning, the patient reported polydipsia and polyuria three months before admission. Plasma osmolality was 289 mOsm/L, and decreased urinary osmolality

(141 mOsm/L) and normal glucose, potassium and calcium levels were found. DI was therefore diagnosed. Treatment was started with L-deamino 8-D arginine vasopressin (LDDV; 10 µg intranasal twice daily), which resulted in decreased urine output within 60 min of administration and increased urinary osmolality. Urinary osmolality increased to 249 mOsm/L 48 h after delivery, and LDDV was therefore discontinued. The patient was discharged from hospital on the fifth postpartum day with no signs of DI or additional complications. Endocrinological follow-up confirmed that there were no underlying metabolic disorders.

The placenta normally secretes small amounts of vasopressinase (a cystinaminopeptidase produced by the trophoblast), which reaches peak levels at the end of pregnancy. The factors predisposing some women to have placental vasopressinase levels high enough to cause DI are unknown. The sudden occurrence and rapid disappearance of symptoms suggest a diagnosis of DI induced by vasopressinase secreted by the placenta. Since vasopressinase levels decrease by 25% one day after delivery, rapid recovery in the postpartum period is normal.³ The risk of recurrence in the next pregnancy is unknown.

The usual signs of DI are polydipsia and polyuria. Diagnosis is not simple because of changes in water metabolism during pregnancy. The initial step is confirmation of free water diuresis to exclude diabetes mellitus. During pregnancy and in the absence of glycosuria, hypokalemia, or hypercalcemia, diabetes insipidus may be diagnosed in a patient with polydipsia and polyuria with serum osmolality higher than 285 mOsm/L associated with hyposthenuria. The exclusion of organic diseases is essential to diagnose DI.⁴

As seen in the reported case, DI is often associated with preeclampsia. Specifically, DI has been associated with acute fatty liver of pregnancy or other liver diseases (elevated transaminases), which are also manifestations of preeclampsia. A hypothesis relating DI and preeclampsia was postulated by Gordge et al.⁵ These authors suggested that

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the association may be explained if vasopressin degradation products retain their pressor activity but lose their anti-diuretic activity. Another theory proposes that decreased vasopressinase metabolism due to pre-existent liver disease results in increased vasopressinase activity and increased vasopressin degradation.⁶

Patients with DI need access to fluids containing free water. The oral intake of pregnant women is restricted in most obstetric departments, which makes intravenous fluid management essential. Because of their inability to concentrate urine, patients cannot prevent hyperosmolality without the administration of high amounts of free water.⁷ LDDV, a vasopressin analogue with a different amino terminal that makes it resistant to vasopressinase, may be administered. No maternal or fetal adverse effects of LDDV have been reported during pregnancy.⁸ Moreover, LDDV has no pressor effect, which is important because of the association of DI with preeclampsia.

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Liver abscess caused by *Klebsiella pneumoniae* in a diabetic patient[☆]

Absceso hepático por *Klebsiella pneumoniae* en un paciente diabético

Liver abscess (LA) is a pus collection located in the liver resulting from any infectious process that causes the destruction of liver parenchyma and stroma. LAs are, in order of frequency, of pyogenic (or bacterial), fungal, and amebic origin.

Pyogenic LA (PLA) may occur by contiguity, as a complication of a biliary tract or abdominal infection, by hematogenous dissemination, or may be cryptogenic (in up to 25–60% of cases). PLAs generally contain multiple microorganisms, which usually come from the gastrointestinal flora. Various recent reports have suggested an increased incidence of PLAs in the elderly, cancer patients, and immunosuppressed subjects.¹ In a recent study conducted in the United Kingdom, Mohsen et al. have found an annual incidence of 18 cases per 100,000 admissions.² In Spain, the annual incidence is 14–35 cases per 100,000 admissions.³

Escherichia coli has traditionally been considered as the main agent responsible for PLA. LAs caused by *Klebsiella pneumoniae* (*K. pneumoniae*) were initially reported in Eastern countries,⁴ but cases of such infection have been reported in recent years in Western countries, including Spain. In addition, according to some studies, its incidence is gradually increasing.⁵ The vast majority of LAs induced by *Klebsiella* spp. are caused by *K. pneumoniae*.^{6,7}

Diabetes mellitus (DM) has been shown to be the most significant risk factor for developing a LA caused by *K. pneumoniae*.⁶ We report a case of hyperglycemic decompensation in a young patient which was triggered by a LA caused by *K. pneumoniae* with hematogenous dissemination and pulmonary embolism.

This was a 46-year-old male taken to the emergency room after being found stuporous, febrile, and with hyperglycemia at his home. For the previous two days, the patient had had a dry cough, low-grade fever, self-limited vomiting, and urinary frequency. His personal history included post-pancreatectomy diabetes mellitus diagnosed 10 years before an episode of alcoholic pancreatitis. No biliary stones or other biliary or pancreatic condition was found. The patient had chronic poor glucose control with glycosylated hemoglobin levels ranging from 9% to 12%, and had been admitted to hospital six months before for diabetic ketoacidosis. There were no known chronic complications of diabetes, and the patient did not regularly attend a medical clinic. Kidney function was normal, with creatinine levels ranging from 0.5 to

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