Introduction

Diabetes insipidus is a rare endocrinological disease and occurs in 2–4 per 100,000 pregnancies [1, 2]. It develops at the end of the second-third trimester of pregnancy and remits spontaneously 4–6 weeks after birth [3]. However, some physiological mechanisms, which accompany pregnancy, can complicate the timely diagnosis of this disease that poses a threat to the mother and foetus [4].

Therefore, a clear understanding of the mechanisms of development, knowledge of the clinical picture and proper diagnosis and treatment are extremely important.

Case presentation

A 32-year-old patient at 36 weeks of gestation, primigravida, was referred to an endocrinologist with complaints of polyuria (6.5 l/day), nocturia — up to 5 times, severe polydipsia. At 12 weeks of gestation, there was a risk of abortion for prevention of which the patient received progesterone 100 mg intravaginally twice a day until 34 weeks. She has a history of subacute thyroiditis, with no family history of endocrine pathology. Physical examination revealed a decrease in skin turgor, blood pressure 110/85 mm Hg. Heart rate 115 bpm, weight 71 kg (body mass index 26.9 kg/m²). The patient was at high risk of developing preecampsia. Laboratory data: analysis of urine according to Zimnitsky: volume per day — 6.8 l, specific gravity in portions: 1.012; 1.008; 1.010; 1.005; 1.012; 1.014; 1.010. Total blood count, total urine test, serum sodium and potassium, liver function tests, level of thyroid-stimulating hormone, free thyroxine, thyroid peroxidase antibodies and morning free cortisol level were normal. The patient was administered desmopressin 10 μg intranasally twice daily. Six weeks after delivery, desmopressin was stopped and she had no further evidence of polyuria, polydipsia or nocturia.

Keywords: diabetes insipidus; pregnancy; gestational diabetes insipidus
risk of developing preeclampsia. Laboratory data: analysis of urine according to Zimnitsky: volume per day — 6.8 l, specific gravity in portions: 1.012; 1.008; 1.010; 1.005; 1.012; 1.014; 1.010. Serum sodium was 141 mmol/l (135–145), potassium 3.8 mmol/l (3.5–5.0), urea 2.4 mmol/l (2.5–8.3), creatinine 42 μmol/l (44–110), serum morning cortisol 7.6 μg/dl (6.2–19.4), thyroid-stimulating hormone 2.75 mEq/l, free thyroxine 14.7 pmol/l (10.5–20.0), thyroid peroxidase antibodies 32 IU/ml, glucose 4.2 mmol/l and HbA1c 5.1 %. Bilirubin, alanine aminotransferase, alkaline phosphatase, albumin, uric acid, creatinine and general blood and urine tests were within normal limits. Thyroid ultrasound was without any remarkable changes.

The patient was treated with desmopressin 10 μg intranasally twice daily.

In this treatment, her daily diuresis decreased to 2.5 l, urine analysis according to Zimnitsky: specific gravity in portions: 1.016; 1.012; 1.025; 1.018; 1.020; 1.022; 1.018.

The pregnancy ended at 41 weeks by caesarean section due to the lack of progressive cervical dilatation, a boy was born with a 1-min Apgar score of 6.

The patient stopped taking vasopressin 4 weeks after delivery. Daily diuresis at 6 weeks postpartum was 2.11, serum potassium, serum sodium were within normal ranges.

**Discussion**

In pregnant women, some physiological processes make diagnosis of diabetes insipidus very difficult: thirst threshold decreases leading to polydipsia and plasma osmolarity decreases causing hypotonic polyuria [5].

Few mechanisms lead to the development of gestational diabetes insipidus but all are caused by decreased vasopressinase activity. Decreased vasopressin level leads to aggravation of hypotonic polyuria [6].

Vasopressinase is produced by trophoblasts and leads to a significant reduction of vasopressin level since the end of the second trimester and aggravates with the development of pregnancy. That’s why this disorder is diagnosed at this period. Some cases of preeclampsia that are characterised by acute fatty liver of pregnancy or other liver diseases may lead to decreased degradation of vasopressinase in liver leading to same disorder.

Hypertrophy or hyperplasia of the adenohypophysis may develop during pregnancy, which may compress the posterior pituitary with decreased release of vasopressin.

During pregnancy, there is an increase of hormones that are antagonists of vasopressin: corticosteroids, progesterone and thyroid hormone. Concomitant diseases, which are characterised by increased production of these hormones, have to be excluded.

Understanding of pathophysiology of the disorder is very important for further management of these vulnerable patients. Differential diagnosis with central and nephrogenic diabetes insipidus is mandatory.

It is made by analysing clinical history, evaluating the concentrating ability of kidneys by Zimnitsky test, fasting blood sugar, HbA1c, sodium, potassium, calcium level in the blood, glomerular filtration rate, creatinine, aspartate aminotransferase, alanine aminotransferase, total bilirubin, cortisol, thyroid-stimulating hormone and thyroxin concentrations, ultra-

sound examination of the liver and kidneys and by the response to desmopressin [6].

Patients should be administered a high amount of free water orally and in severe cases intravenously because of their inability to concentrate urine that may lead to dehydration and hypernatremia. In mild cases, diet with increased amount of water and decreased sodium chloride intake is sufficient, but more commonly the prescription of L-deamino-arginine vasopressin (desmopressin) is necessary. Desmopressin is a vasopressin analogue with changed amino-terminal that makes it resistant to vasopressinase. It has no maternal or fetal side effects, so can be used for the treatment of gestational diabetes insipidus [3].

**Conclusions**

Diagnosis of gestational diabetes insipidus is very difficult because develops against the background of physiological mechanisms that accompany pregnancy.

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**References**


Клінічний випадок /Clinical Case/

Резюме. Нецукровий діабет належить до рідкісних ендокринологічних захворювань і трапляється у 2—4 пацієнтів на 100 000 вагітностей. Діагностика гестаційного нецукрового діабету дозволяє складна, оскільки він розвивається на тлі фізіологічних процесів, які супроводжують вагітність: поріг спраги знижується, що призводить до полідипсії, зменшується гіпотензічна поліурія. Розуміння патологічної фізіології захворювання дуже важливе для подальшого ведення цих пацієнтів. Наводиться опис клінічного випадку.

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Клінічний випадок гестаційного нецукрового діабету

Клинический случай гестационного несахарного диабета

Резюме. Несахарный диабет принадлежит к редким эндокринологическим заболеваниям и встречается у 2—4 пациентов на 100 000 беременностей. Диагностика гестационного несахарного диабета сложна, поскольку он развивается на фоне физиологических процессов, сопровождающих беременность: порог жажды снижается, что приводит к полидипсии, уменьшается гипотоническая полиурия. Понимание патологической физиологии заболевания очень важно для дальнейшего ведения этих пациентов. Представляется описание клинического случая.

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