Clinical case of gestational diabetes insipidus

Abstract. Diabetes insipidus is a rare endocrinological disease and occurs in 2–4 per 100,000 pregnancies [1, 2]. Diagnosis of gestational diabetes insipidus is very difficult because it develops against the background of physiological mechanisms that accompany pregnancy: thirst threshold decreases leading to polydipsia and plasma osmolarity decreases causing hypotonic polyuria. Understanding of pathophysiology of the disorder is very important for further management of these vulnerable patients. 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 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. 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

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. The patient 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 was 115 bpm, weight 71 kg (body mass index 26.9 kg/m²). The patient was at high
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 μEq/l, free thyroxine 14.7 μmol/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 2.1 l, 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 adrenocortical apparatus 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 thyroxine. 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 and diabetes mellitus 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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Клінічний випадок гестаційного нецукрового діабету

Abstract. Diabetes mellitus during pregnancy is a potentially dangerous disease for both the mother and the newborn. The treatment of this disease is required in cases where glycosuria, polydipsia, or nocturia is observed. The woman, 32 years old, at the 36th gestational week, was admitted to the endocrinologist due to polyuria (6.5 l/day), nocturia — up to 5 times, associated with the development of signs of pre-eclampsia. Laboratory data: analysis of urine by Zimnitsky: daily volume — 6.8 l, specific gravity — 1.010; 1.005; 1.012; 1.014; 1.010. Complete blood count, urine analysis, content of sodium and potassium in the blood, liver function, level of thyroid-stimulating hormone, free thyroid, antibodies to thyroid peroxidase and morning free cortisol. The patient was admitted to the group of high risk of pregnancy complications. During the physical examination, a decrease in skin turgor, arterial pressure 110/85 mm Hg, heart rate 115 beats/min, body weight 71 kg (BMI 26.9 kg/m²). The patient was on a diet of 4 g, 8 divided doses: 1,012; 1,008; 1,010; 1,005; 1,012; 1,014; 1,010. During the 12th week, increased risk of hypoglycemia, decreased and osmolality of blood, decreased, and osmolality of blood, decreased, and osmolality of blood, decreased, and osmolality of blood, decreased, and osmolality of blood, decreased, and osmolality of blood, decreased.

Key words: non-sugar diabetes; pregnancy; gestational diabetes.