Primary chronic adrenal insufficiency (CAI, Addison’s disease) is a pathological condition associated with a decrease in the production of glucocorticoids and mineralocorticoids by the cortical layer of the adrenal glands [1]. The main cause of the disease is autoimmune adrenalitis (about 90 % of patients), other etiologic factors include infectious, iatrogenic and genetic [2, 3]. The main clinical manifestations of the disease include electrolyte disorders (hyponatremia and hyperkalemia, hypoglycemia, hypoparathyroidism and CIA, is described by D. Waiteker [4]. Often in this condition, there may be pernicious anemia, alopecia, vitiligo, bronchial asthma, glomerulonephritis, chronic autoimmune hepatitis.

It is believed that the disease develops gradually with the consequent emergence of hypoparathyroidism, CAI, later it can manifest primary hypogonadism. It is advisable to perform a diagnostic search after detecting one autoimmune disease of possible other syndrome components. APS type 2 was described by M. Schmidt in 1926, it included chronic autoimmune thyroiditis (AIT) and CAI [5]. Another variant of the APS type 2 is Carpenter’s syndrome (a combination of CAI, type 1 diabetes mellitus (DM) and AIT, described in 1964 by C. Carpenter) [6]. In 1981 N. Neufeld defined APS type 2 as a combination of autoimmune polyglandular syndrome type 2 in a patient aged 47 years is described (the onset of the disease with the development of type 1 diabetes mellitus and the subsequent chronic adrenal insufficiency) against the background of concomitant pathology (peptic ulcer, arterial hypertension, adrenal incidentaloma).

Keywords: Carpenter syndrome; diabetes mellitus; adrenal insufficiency

From anamnesis

DM type 1 has been diagnosed since 1992, from the onset of the disease on insulin therapy. Periodically he noted the development of hypoglycemic conditions. According to the patient, over the past year, there has been an increase in the development of hypoglycemic conditions, twice hypoglycemic coma, which required medical intervention. At time of hospitalization he took insulin therapy in intensify regimen: Insuman Rapid in the morning 2 IU, in the noon 2 IU, in the evening 2 IU, at 22.00 Insuman Basal — 6 IU. The dose of insulin has significantly decreased over the last year. Glycemia fluctuations from 2.9 to 29.2 mmol/l. 2 IU, in the evening 2 IU, at 22.00 Insuman Basal — 6 IU.

A clinical case of observation of a patient with Carpenter’s syndrome, which had clinical features and presented certain difficulties in diagnosis, is shown. Patient P., 47 years old, hospitalized in the endocrinology department of the KI “RK Endocrine Dispensary” ZRC on 18th May, 2017 with complaints of pronounced general weakness, fatigue, frequent hypoglycemic conditions, a decrease in body weight of 15 kg over the past 6 months, nausea, vomiting, epigastric pain, right hypochondrium and pyloroduodenal zone.
Objective examination
The general condition of moderate severity, he’s conscious, the skin is clean, earthy grey colour, BMI is 19.6, the location of fatty tissue is even, breathing rate — 16 per min, the percussion is clear over the lungs, breathing is vesicular, rales are absent, cardiac activity is rhythmic of the heart, blood pressure 110/80 mm Hg, the abdomen is mild, moderately painful in epigastrium and in the pyloroduodenal zone palpatory. The liver and spleen are not enlarged. Stool and diuretics are normal. The pastosity of the shins and feet is marked, pulsation over the peripheral arteries of the lower limbs is reduced.

Laboratory data
Blood count (19.05.2017): hemoglobin — 124 g/l, RBC — 3,7 · 10²³/l, WBC — 4,1 · 10⁹/l, ESR — 19 mm/hr, eosinophils — 1 %, stab — 0 %, segmented — 65 %, lymphocytes — 31 %, monocytes — 3 %.

Biochemistry (19.05.2017): total cholesterol — 3.5 mM/l, triglycerides — 1.42 mM/L, VLDL cholesterol — 0.56 mM/L, LDL cholesterol — 2.28 mM/L, urea — 5.2 mM/L, creatinine — 116.6 μM/l, GFR — 58, total bilirubin — 11.0 μM/l, direct bilirubin — 2.8 μM/l, thymol test — 0.99, AST — 0.27, ALT — 0.54, total protein — 56.1 g/l, potassium — 3.97 mM/l, sodium — 132 mM/l, bicarbonate — 23.05.2017 — 10,4–5,8–6,0–8,0 mM/l;
— 20.05.2017 — 4,1–9,2–11,1–5,6 mM/l;
— 23.05.2017 — 10,4–5,8–6,0–8,0 mM/l;
— 26.05.2017 — 7,4–14,0–6,2–12,7 mM/l.

Glycemic profile (08:00–11:00–16:00–20:00): 20.05.2017 — 4,1–9,2–11,1–5,6 mM/l;
23.05.2017 — 10,4–5,8–6,0–8,0 mM/l;
26.05.2017 — 7,4–14,0–6,2–12,7 mM/l.

Urinalysis: 19.05.2017: gravity — 1006, WBC — 0–2 in area vision, protein, ketones absent, singular flat epithelium.
22.05.2017: urinalysis by Nechiporenko: WBC — 500/mm³, RBC absent, protein absent.
24.05.2017: daily glucosuria absent, daily proteinuria absent.
27.05.2017: microalbuminuria — 89 mg/day.

Intrumental data
ECG (19.05.2017). HR — 80 beats per min, the voltage is lowered, sinus rhythm, electrical axis of the heart is not rejected.

X-ray of the gastrointestinal tract (29.05.2017): ulcer of duodenal bulb, chronic pancreatitis. Examination of barium passage: the whole weight of barium in the distal ileum, cecum and ascending colon.

Fiber-optic gastrooduodenoscopy (24.05.2017): Atrophic gastropathy, erosive bulbitis, scar deformation of duodenal bulbs.

CT of abdominal organs (25.05.2017): CT signs of abdominal and retroperitoneal adenopathy, nodular hyperplasia of the left adrenal gland (uneven contours due to the presence in its body node spherical shape with sharp, smooth contours, relatively homogeneous structure with a diameter of 6 mm), simple cysts of left kidney.

Narrow specialist consultations

Neurologist (18.05.2012): dismetabolic encephalopathy 1, cerebroasthenic syndrome, diabetic distal symmetric polyneuropathy of the lower limbs, sensory-motor form (NSS 3, NDS 3), chronic course.

Ophthalmologist (02.06.2017): the optic nerve disc is pale pink, the borders are clear, the arteries are narrowed, and moderate angiokeratosis. Salus 1. Microaneurysms of venous vessels, microhemorrhages. In the macular area without features. Conclusion: Nonproliferative diabetic retinopathy of both eyes.

Gastroenterologist (22.05.2017): Peptic ulcer, inactive phase, chronic pyloric ulcer in the stage of scarring, chronic pancreatitis in the stage of unstable remission. Recommended: X-ray of the stomach with repeated examination. 25.05.2017: Peptic ulcer, active phase, chronic erosive gastroduodenitis in the acute stage, associated with H.pylori.

Angiosurgeon (06.06.2017): diabetic angiopathy of the arteries of the lower extremities.

Oncourologist (26.05.2017): hyperplasia of the left adrenal gland, oncuropathology is absent.

Comments
Picture of gastric dyspepsia (nausea and vomiting) were associated with the clinic of peptic ulcer (the history of the disease was taken into account, the diagnosis of the active phase of the disease was confirmed after additional examination and consultation of the gastroenterologist). During treatment in the hospital, there was a lack of compensation for the level of the peak of hypoglycemia, despite the ongoing correction of insulin doses. At the same time, the decrease in the level of arterial pressure at the peak of hypoglycemia (up to 80/40 mm Hg) attracted attention. After additional examination, the development of primary insufficiency of the adrenal cortex was revealed. Mass in the left adrenal gland is most likely a hormonal non-active benign tumor (incidentaloma), or has a relationship to the systemic pathological process associated with lymphadenopathy in the abdominal cavity, which remained diagnostically unclear.

Diagnosis: Diabetes mellitus type 1, severe form labile course with a tendency to hypoglycemic states, decompensation. Diabetic nonproliferative retinopathy of both eyes. Diabetic peripheral distal symmetrical polyneuropathy (NSS 3, NDS 3), sensory-motor form, chronic course. Diabetic angiopathy of the lower extremities, II degree. Diabetic nephropathy III degree. CKD III. Chronic adrenal insufficiency, moderate severity, decompensation. Dysmetabolic encephalopathy, I degree, cerebroasthenic syndrome. Peptic ulcer, active phase, chronic ulcer of the duodenal bulb, associated with H.pylori, with increased acid-producing function of the stomach. Arterial hypertension, I degree, 2 stage, high cardiovascular risk, hypertensive angiopathy of the retina. Nodular hyperplasia of the left adrenal gland, hormone-inactive. Intraabdominal and retroperitoneal lymphadenopathy of the unexplained genesis.

Treatment
The patient continued to receive insulin therapy with correction of insulin doses under the control of the gly-
гландулярного синдрома типа 2 у пациента 47 лет (дебют заболевания с сахарным диабетом, хронической надпочечниковой недостаточностью) на фоне сопутствующей патологии (язвенная болезнь, гипертоническая болезнь, инсисталома коры надпочечников). Ключевые слова: синдром Карпентера; сахарный диабет; недостаточность коры надпочечников.

Климичний випадок /Clinical Case/

1. Черникова В.В.1, Солов’юк О.О.2, Солов’юк О.А.2 1 Коммунальна установа «Обласний клінічний ендокринологічний диспансер» Запорізької обласної ради, м. Запоріжжя, Україна 2 Запорізький державний медичний університет, м. Запоріжжя, Україна

Аутоіммунний полігландулярний синдром 2-го типу (клінічний випадок синдрому Карпентера)

Резюме. Синдром Карпентера — рідкісне автіоімунне захворювання, що асоціюється з порушенням функції низки органів ендокринної системи (цукровий діабет, хронічна надирково недостатність та/або автоіммунний тиреоїдит). Описаний випадок атипового перебігу автіоіммунного полігландулярного синдрому типу 2 у пацієнта 47 років (дебют заболевания с сахарным диабетом типа 1 с последующим при- соединением хронической надирковой недостаточности на фоне сопутствующей патологии (язвенная болезнь, гипертоническая боль, инсисталома коры надирковых залоз). Ключевые слова: синдром Карпентера; сахарный диабет; недостаточность коры надирковых залоз.

Резюме. Синдром Карпентера — редкое аутоиммунное заболевание, которое ассоциировано с нарушением функции ряда органов эндокринной системы ( сахарный диабет, хроническая надирковая недостатность и/или аутоиммунный тиреоидит). Описан случай атипичного течения аутоиммунного полигландулярного синдрома типа 2 у пациента 47 лет (дебют заболевания с развитием сахарного диабета типа 1 с последующим присоединением хронической надирковой недостаточности на фоне сопутствующей патологии (язвенная боль, гипертоническая боль, инсисталома коры надирковых залоз). Ключевые слова: синдром Карпентера; сахарный диабет; недостаточность коры надирковых залоз.