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EPISODE OF RENAL DYSFUNCTION IN A CHILD WITH EATING DISORDER: A CASE REPORT

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Among the various organ dysfunctions seriously affected by eating disorders (EDs) is kidney damage. The kidneys are vital organs responsible for several essential functions, including the balance of the body's acid-base and mineral metabolism, the removal and excretion of substances, and the regulation of blood volume and pressure.

Eating disorders are a widespread and clinically relevant class of multiorgan disorders that occur mainly in adolescence. EDs often have serious clinical manifestations, which are determined by electrolyte imbalance, endocrine and mental disorders, renal failure, and other disorders. Common eating disorders discussed in the literature and frequently encountered in clinical practice are anorexia nervosa and bulimia nervosa. Anorexia nervosa is a type of abnormal eating behaviour that involves the consumption of very little food and may include intentional vomiting or the misuse of laxatives or diuretics.

Kidneys perform a number of vital functions to maintain homeostasis in our body. One of the main functions of the kidneys is blood filtration and the removal of metabolic products. This allows you to maintain the optimal level of fluid and electrolytes and remove toxins. Restriction of fluid intake and abuse of diuretics can cause a decrease in blood flow to the kidneys and further renal failure. The result of the latter is the development of serious violations of vital functions. Low dietary protein intake in people following a restricted diet (restrictive anorexia) leads to a decrease in glomerular filtration rate (GFR) and renal plasma flow. Treatment of acute kidney injury on the background of volume deficit due to fluid loss involves its termination and hydration orally or with the help of intravenous infusions of physiological solution depending on the severity of volume deficit and kidney damage, which was observed and described in this clinical case.

***Aim**. To analyze the peculiarities of kidney injury in a child with an eating disorder.*

***Materials and Methods.** A detailed analysis of the case history of a pediatric patient diagnosed with eating disorder complicated with an acute episode of renal dysfunction was done. Basic anamnestic, clinical, laboratory, and instrumental data were analyzed and given in the paper. A graphic presentation is done with PowerPoint.*

***Results**. We describe a 14-year-old female patient diagnosed with ED and acute kidney injury (AKI) leading to intensive care unit interventions.*

***Conclusions.** AKIs can result from a number of conditions that are common among anorexics and include dehydration, high blood pressure, toxins, inadequate nutrition and possibly altered electrolytes. Serum creatinine, blood urea nitrogen (BUN), glomerular filtration rate (GFR), and electrolytes are among the kidney function markers that need to be monitored in the early stages of AKI linked to anorexia. The right medical care, which includes normalizing blood pressure and adequate hydration, leads to the patient's recovery. Further follow-ups on kidney function in such patients are of high importance*

***Keywords**: eating disorder, kidney damage, children, monitoring*

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1. Introduction

Kidney problems are among the many organ dysfunctions that are significantly impacted by eating disorders (ED). The kidneys are essential organs that carry out several bodily activities, such as maintaining the body's acid-base and mineral balance, breaking down and eliminating chemicals, and controlling blood pressure and volume [1]. The type of ED and particular activities that the patient engages in are risk factors for renal disease in ED. In pediatric medicine, anorexia nervosa-restricting subtype (AN-R), in which weight reduction is accomplished by fasting and dieting, is rather prevalent [1–3].

Large population-based studies on acute kidney damage (AKI) and the development of chronic kidney disease (CKD) in AN patients are scarce. Nevertheless, after 21 years, some studies predict that more than 5 % of AN patients will experience end-stage renal disease (ESRD). The term end-stage renal disease (ESRD) describes the irreversible loss of kidney function that is predicted to cause mortality in a matter of days or weeks

due to consequences such as hyperkalemia (serum potassium levels greater than 5 mEq/L) or pulmonary oedema in the absence of dialysis or a kidney transplant. Although there are case series and case reports of AN patients who developed ESRD and underwent dialysis, the rate of dialysis in AN patients is unknown [4].

In retrospective research, renal function was found to be compromised in 37 % of adolescents hospitalized with a very recent diagnosis of AN (subtype not specified). Analyzing secondary data from 120 teenagers and young adults hospitalized for medical instability from AN, it was discovered that 33 % of patients had impaired kidney function; fast weight loss and severe bradycardia were risk variables linked to renal impairment in this study [2].

A cross-sectional study conducted in the past on hospitalized adolescents suffering from severe malnutrition from AN-R revealed that 72 % of the patients had impaired kidney function. Of these, 59 % had eGFRs between 89 and 60 mL/min (stage 2 mild CKD), 12 % had eGFRs between 59 and 45 mL/min (stage 3A moderate CKD), and 2 % had eGFRs between 44 and 30 mL/min (stage 3B moderate CKD) [1, 5].

In ED, kidney dysfunction is complicated and poorly understood. The following are risk factors for kidney disease: body mass index (BMI), bradycardia, hypokalemia, nephrocalcinosis (calcium deposits in the kidneys), length of the disease, chronic dehydration brought on by reduced oral intake and/or purging behaviours, and nutritional condition [1]. However, since proper nutrition is required for kidney function to be at its best, malnutrition itself is a risk factor for renal disease. Low protein intake lowers renal plasma flow and GFR, which is evident in people on restrictive diets. Malnutrition high in protein and calories affects the kidneys' capacity to concentrate urine and eliminate acid and salt. Although some research suggests that having a low body mass index increases the risk of kidney illness, AN also has decreased kidney function, highlighting the possible consequences of starving on renal function even at higher weight [1, 6].

AN has been linked to kidney failure brought on by rhabdomyolysis, a condition in which the body breaks down muscles as a result of trauma, overexertion, or stroke and releases waste materials into the circulation [6, 7].

Thus, the renal disease can develop in the ED but is frequently misdiagnosed, including ESRD, which requires dialysis. Physicians must take into account assessing kidney function measures in all ED patients, particularly those with protracted disease courses. Aim. To analyze a pecularities of the kidney injury in a child with eating disorder.

2. Material and methods

A case study and medical records were analyzed in the study. The study was conducted in 2023 in Clinical Pediatric Hospital No. 6, where the Clinical Base of the Department of Pediatrics No. 4 of Bogomolets National Medical University is located. Ethical agreement for the study conduction was obtained from Bogomolets National Medical University Ethical Committee (Protocol No. 166, 19.12.2022). The work was carried out following the Code of Ethics of the World Medical Association (Declaration of Helsinki). Written consent was obtained from the patient prior to publication. The data is expressed as actual parameters. Data will be made available at a reasonable request. The authors confirm they did not use artificial intelligence technologies when creating the current work.

3. Case presentation

A female patient, 14 years old, was admitted to the endocrinology unit of the pediatric hospital with complaints of weight loss of 7 kg/3 months and thirst (drinks 2 L of water/day). She was referred to our hospital by her physician for suspicion of diabetic ketoacidosis. No significant pathologies emerged in the review of the patient's family history.

The following data were revealed: weight 43 kg, height 1.57 m, BMI 17.4 kg/m^2; blood pressure 105/75 mmHg; heart rate 62/min; respiratory rate 16/min; cutaneous temperature 36.7 °C. Almost all of the teeth underwent odontoiatric cures or were substituted. Hair and nails appeared normal, and skin appeared dry. Menstruation since the age of 12 was irregular, with a delay of up to 9 days.

Diagnosis at admission: eating disorder, cardiomyopathy.

Laboratory findings obtained during the first 24 h stay in hospital: HbA1c 4.9 %, blood glucose 4.71 mmol/L, serum creatinine 102.6 mmol/L (increased 1.7 times as compared to normal value), BUN 8.3 mmol/L, ALAT 7.8 IU, ASAT 10.5 IU, total blood protein 73.0 g/L. Urinalysis: protein 0.033 g/24 h. Analysis of urine concentration kidney capacity shows hyposthenuria (Table 1).

Table 1

Urine concentration kidney capacity

No.	Time	Urine gravity	Urine volume, mL
1	6–9	1004	600
2	9–12	1007	750
3	12–15	1009	80
4	15–18	1006	400
5	18–21	1008	300
6	21–00	1005	750
7	00–3	–	–
8	3–6	1003	550

The patient was transferred to the ICU. Laboratory findings obtained during the stay 1st day in ICU are the following: creatine kinase MB-isoenzyme 0.2 ng/ml, ferritin 113.90 ng/ml, iron 19.5 μmol/l, transferrin 2.07 g/l, cystatin C 2.36 mg/l, GFR 28 ml/min./1.73 m2, cyanocobalamin 450 pg/ml, folic acid 5.8 ng/ml, creatine kinase MB-isoenzyme 0.2 ng/ml, ferritin 113.90 ng/ml, iron 19.5 μmol/l, transferrin 2.07 g/l, cystatin C 2.36 mg/l, GFR 28 ml/min./1.73 m2, cyanocobalamin 450 pg/ml, folic acid 5.8 ng/ml.

Anti-nuclear IgG (U1-RNP, SS-A/Ro, SS-B/La, centromere B, Scl-70, Jo-1, fibrillarin, RNA Pol III, Rib-P, PMScl, PCNA, Mi-2/Sm, dsDNA): 0,3 (negative);

dsDNA<0.5 (negative); ADNA 2.6 (negative). Acid-Base parameters: pH 7.42, pCO2 44 mm/Hg, pO2=21 g/L, tHb 120, Na 146 mmol/L, K 3.9 mmol/L, Ca 1.27 mmol/L, BE 2.9. Dynamics of the basic biochemical parameters obtained during the ICU stay are given in Table 2.

Table 2

Dynamics of the laboratory variables during the treatment in ICU

ICU day	Total blood protein, g/L	Blood albumin, g/L	Total bilirubin, mcmol/L	ALAT, IU	ASAT, IU	Serum creatinine, mcmol/L	BUN, mmol/L	Blood glucose	K, mmol/L	Na, mmol/L	Ca, mmol/L	Cl, mmol/L	GFR
Ist day	74.9	50.6	10.6	7	17.9	209	14.22	2.84	3.53	133.9	1.202	97.1	24
2nd day						182	14.44	5.18	3.25	141.2	1.223	103	28
5th day						95	6.88	5.12	4.14	139.6	1.075	98.1	54

5th day of the ICU stay:

Daily proteinuria: 0,293 g/24 h. Urine concentration kidney capacity is shown as hyposthenuria (Table 3).

Coagulogram: fibrinogen 355 mg/dL, PT 14.4 sec, PTI 97 %, APTT 27.0 c

ECG (1st day of the ICE stay): sinus tachycardia, pronounced diffuse metabolic changes in the myocardium (negative T wave in V4-V6, in II standard lead). Positive dynamics were observed on the 5th day of the ICU stay. Sub-specialist consultations were shown: cardiomyopathy (cardiologist) and a depressive state (psychologist) (Fig. 1).

Table 3

Urine concentration kidney capacity

No.	Time	Urine gravity	Urine volume, mL
1	6–9	–	–
2	9–12	1008	450
3	12–15	1005	200
4	15–18	1005	500
5	18–21	1003	500
6	21–00	1008	500
7	00–3	1007	200
8	3–6	1003	800

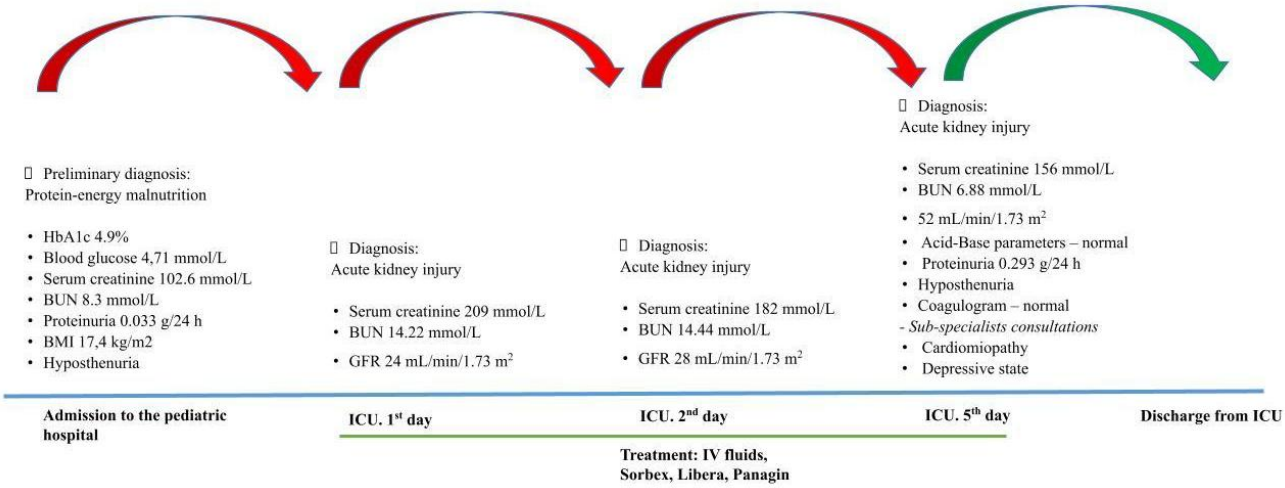

Fig. 1. Summarized picture of the examined patient's hospital course

Treatment done during the ICU period included infusion therapy with glucose-saline solutions for 3 days, Sorbex, Libera, Panangin. Under this treatment, Serum creatinine levels turned down, and patients were discharged from the ICU under the out-patient supervision of a nephrologist.

4. Discussion

With severe clinical signs such as hypothermia, hypotension, electrolyte imbalance, endocrine problems, and kidney failure, psychological EDs are becoming a more prevalent public health concern [1]. Volume overload and electrolyte imbalances are among the serious complications that can arise from AKI, which is defined as a "heterogeneous group of conditions characterized by a sudden decrease in glomerular filtration rate (GFR) followed by an increase in serum creatinine concentration or oliguria [low urine output]". AKI can also progress to chronic kidney disease (CKD). The hallmark of anorexia nervosa (AN) is a very low-calorie diet that

leaves the patient's body weight at least 85 % below average for their height and age [1–3].

After a thorough analysis of the latest research, three main routes leading to renal involvement were identified: nephrocalcinosis, chronic rhabdomyolysis, and chronic dehydration and hypokalemia. Renal tubule damage that cannot be reversed could result from prolonged potassium deficiency [1]. A gradual tubulointerstitial damage induced by hypokalemia was demonstrated in vitro in rat models. Chronic dehydration caused by AN can result in loss of water and salts due to the overuse of diuretics and laxatives, metabolic alkalosis (loss of acids through vomiting), and hyperaldosteronism due to hypovolemia [8].

A primary tubulopathy, such as Gitelman's disease, might be mistakenly diagnosed as an eating disorder, and vice versa. Renal lesions may result from three pathophysiological mechanisms: increased ammoniogenesis, vasoactive mediator activation, and arterial hypersensitivity to Na+ [8]. Hypokalemia causes ammoniogenesis, HCO3-reabsorption, and C3 amination. This activates the alternative complement pathway and causes protein deposition in the tubule. Vasoactive mediators are altered by hypokalemia: vasoconstrictor stimuli (ACE, ET-1, and subtype B α-adrenergic receptors) are increased, whereas vasodilatory stimuli (EDRF-1 and PGE2) are decreased [8, 9].

Additionally, higher activity of the cotransporter Na+K+2Cl– in the ascending limb of the loop of Henle is linked to hypokalemia; this association may result from hydrosaline retention or occasionally from elevated arterial pressure. Nephron atrophy, tubular cell damage, and calcified material blockage in the tubules can all cause renal failure. These alterations are accompanied by interstitial fibrosis and chronic inflammation [9].

Rhabdomyolysis in AN has been reported in a few case reports, either with or without acute renal failure [10]. We hypothesize that severe energy deficiency, prolonged dehydration, metabolic disturbance, food restriction and starvation. Severe vomiting and diarrhoea can also cause cell lysis and the release of toxins into the bloodstream, including myoglobin. The kidneys' filtration proteins are harmed by myoglobin. Although hospitalization with IV fluids can quickly manage this, it can be very dangerous.

Metabolic diseases have occasionally been identified as the cause of RRAS abnormalities in AN. The latter's outcomes are the variations in our patient's blood pressure and ECG.

The limitation of the study is the lack of pathomorphological verification of kidney damage.

Prospects for further research. This study is promising in terms of studying kidney-specific markers of kidney damage under the conditions of EDs.

5. Conclusions

Early stages of the AKI associated with AN can be undiagnosed and require monitoring of the kidney function parameters, i.e. serum creatinine, BUN, GFR, and electrolytes. However, most people improve with proper medical attention, which involves proper hydration and blood pressure normalization. These patients must get further kidney function monitoring.

Conflict of interest

The authors declare that they have no conflict of interest concerning this research, whether financial, personal, authorship or otherwise, that could affect the research and its results presented in this article.

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Data availability

Data will be made available at a reasonable request.

Use of artificial intelligence

The authors confirm that they did not use artificial intelligence technologies when creating the current work.

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