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Case Report

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Thrombotic microangiopathy unmasked: alpha-lipoic acid poisoning in an adolescent

Abstract

Alpha-lipoic acid is widely used as an antioxidant in dietary supplements, including for weight loss. The article presents a rare case of severe acute alpha-lipoic acid poisoning in a 12-year-old girl with anorexia nervosa. The clinical picture included recurrent episodes of vomiting, diarrhea, seizures, progressive metabolic lactic acidosis, severe thrombocytopenia, acute kidney injury with anuria, and multiple organ failure. Against the background of intensive therapy, including renal replacement therapy, the patient survived. Percutaneous kidney biopsy revealed acute tubular necrosis, consistent with drug-induced kidney injury. This case is the first in the literature describing a relatively favorable outcome in a child with multiple organ failure and anuria due to alpha-lipoic acid poisoning. The importance of targeted history-taking in patients with eating disorders is emphasized.

Keywords: alpha-lipoic acid, poisoning, acute kidney injury, lactic acidosis, thrombocytopenia, anorexia nervosa, children, adolescents, multiple organ failure

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Abbreviations: CKiD U25 Crea, creatinine-based estimated kidney function is calculated using an equation from the Chronic Kidney Disease in Children study, specifically designed for people under age 25; ALA, alpha-lipoic acid; BMI, body mass index; AST, Aspartate Aminotransferase; ALT, Alanine Aminotransferase CK, creatine kinase; LDH, lactate dehydrogenase; aPTT, Activated Partial Thromboplastin Time; DIC, Disseminated Intravascular Coagulation.

Introduction

Alpha-lipoic acid (ALA, thioctic acid) is an endogenous antioxidant involved in mitochondrial energy metabolism.[1] In clinical practice, alpha-lipoic acid is used for diabetic polyneuropathy, and in recent years it has been actively used in dietary supplements for body weight correction, improvement of metabolism, and reduction of oxidative stress in obesity, metabolic syndrome, and arterial hypertension.[1,2] The drug is considered relatively safe; however, its over-the-counter availability creates preconditions for cases of intentional or accidental overdose.[3]

Acute alpha-lipoic acid poisonings are rare, which is associated with its low toxicity at therapeutic doses. Nevertheless, ingestion of high doses can lead to severe, life-threatening conditions. The literature describes only 9 cases of acute alpha-lipoic acid poisoning.[4] Severe forms are accompanied by metabolic acidosis, seizures, hematological disorders, and target organ damage.[4–7]

The present clinical observation is of interest due to the extremely severe course of acute ALA poisoning in a child with an eating disorder, the development of multiple organ failure, and morphologically confirmed toxic kidney injury with a relatively favorable outcome, which has not been previously described in the literature.

Case description

Girl G., born from the 3rd physiological pregnancy and 3rd delivery at 40 weeks. Birth weight 3944 g (z-score: 1.46; 92.8‰). Early development without peculiarities. She has been followed by an endocrinologist for excessive body weight (BMI z-score: 1.24), a gastroenterologist for chronic gastroduodenitis, and a traumatologist-orthopedist for congenital hip dislocation and scoliosis. Family history: mother — hypothyroidism; maternal grandmother — type 2 diabetes mellitus, arterial hypertension; grandfather — urolithiasis; older sister — hypothyroidism since age 3.

At the age of 12, she experienced an episode of repeated vomiting, tremor, diarrhea, and profuse sweating for the first time. Due to the severity of her condition, she was admitted to the intensive care unit. Examination revealed metabolic acidosis (pH 7.29, BE -4.4, HCO_3 22 mmol/L), thrombocytopenia (140×10^9/L), estimated GFR (CKiD U25 Crea) — 53.2 mL/min/1.73 m², and urinary syndrome with microhematuria. Ultrasound of the kidneys and abdominal organs showed reactive changes in the pancreas; ECHO-CG — mitral valve insufficiency grade 1. Neurological status: emotionally labile. Due to tremor, further examination was performed: brain CT and EEG showed no pathology. By day 3 of the disease, against the background of infusion and antibacterial therapy, positive dynamics were noted — normalization of stool and acid-base balance — although thrombocytopenia persisted (71×10^9/L). On day 8 of therapy, platelet count normalized (165×10^9/L), creatinine decreased to 79 µmol/L, eGFR (CKiD U25 Crea) — 62.6 mL/min/1.73 m². Based on the above, the girl's condition was assessed as manifestations of acute intestinal infection and acute tubulointerstitial nephritis.

One day after discharge from the hospital, a recurrent episode of vomiting and loose stools occurred, accompanied by tonic-clonic

seizures. Examination revealed thrombocytopenia (59 × 10^9/L), metabolic acidosis (pH 6.72, BE -30.7, lactate 24.39 mmol/L, HCO_3 4.4), eGFR (CKiD) 33.9 mL/min/1.73 m², and urinary syndrome with macrohematuria and proteinuria (3.8 g/L). Infusion and anticonvulsant therapy were administered.

On the second day of the disease, her condition deteriorated: bleeding from injection sites, small hemorrhagic rash on the hands and feet, diarrhea, anuria, BP 130/111 mm Hg, leading to transfer to mechanical ventilation. Laboratory findings: thrombocytopenia (37 × 10^9/L), hemoglobin 143 g/L; signs of cytolysis (AST 672 U/L, ALT 441 U/L, LDH 4946 U/L, CK 2321 U/L, α-amylase 1410 U/L); impaired renal excretory function: eGFR (CKiD) 13.9 mL/min/1.73 m²; blood electrolytes normal; lactate 4.04 mmol/L; signs of hypocoagulation: aPTT 70.2 sec, fibrinogen 0.7 g/L.

On the 3rd day, the condition remained extremely severe: coma grade 3, GCS 3 points, on medication sedation; persistent anuria; signs of DIC syndrome — bleeding from catheter insertion sites despite hemostatic therapy. Examination showed normoregenerative normocytic anemia (hemoglobin 72 g/L), thrombocytopenia (58 × 10^9/L), hyperazotemia with progression: eGFR (CKiDu25) 8.4 mL/min/1.73 m²; elevated CK (20782 U/L), elevated LDH (>4450 U/L); negative Coombs test; D-dimer increased to 63282 ng/L; C3 and C4 complement components normal. Viral and intestinal infections were ruled out. Esophagogastroduodenoscopy (EGD) revealed gastric erosions. Renal replacement therapy was initiated with veno-venous hemofiltration (2 sessions), followed by hemodialysis sessions and subsequent initiation of peritoneal dialysis as the condition stabilized. She was transferred from the intensive care unit on day 10 for continued treatment. Figure 1 shows the dynamics of the disease.

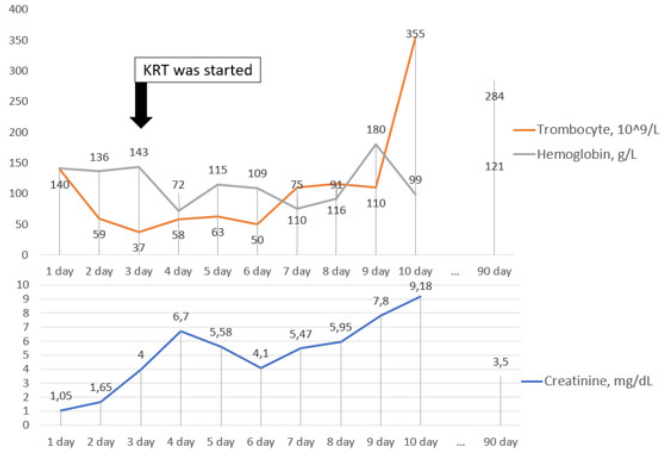

Figure 1 The course of the disease. KRT — kidney replacement therapy.

One and a half months later, stabilization of the condition was noted: no hemodynamic disturbances, eGFR (CKiDu25) 18 mL/min/1.73 m². Ultrasound showed signs of decreased kidney size and reduced renal blood flow. Given the acute onset of the disease, hemodynamic disturbances, anuria, and impaired renal excretory function, a percutaneous kidney biopsy was performed to differentiate from prerenal azotemia. The specimen contained 12 glomeruli, none sclerotic. Glomeruli were not enlarged, without mesangial or endocapillary hypercellularity. Capillary loop walls were not thickened and single-contoured. Focal edema and developing interstitial fibrosis with associated tubular atrophy involving less than 10% of the renal parenchyma. Focal interstitial infiltration with lymphocytes, admixed with single neutrophils and plasma cells, and tubulitis phenomena (up to 2–3 lymphocytes per tubular cross-section), mainly in subatrophic tubules. In some tubules, damage to the tubular epithelium with complete or partial loss of the brush border and decreased height of the tubular epithelium was noted.

Arteries and arterioles — without peculiarities. Immunofluorescence: negative. Conclusion: Focal interstitial nephritis. Acute tubular necrosis (Figure 2).

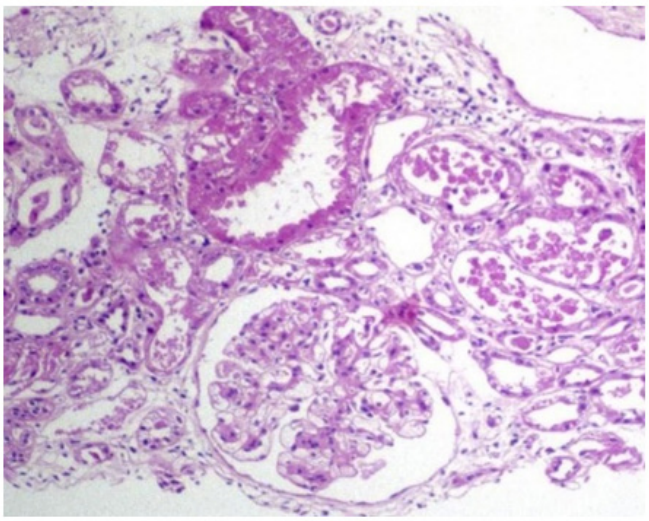

Figure 2 Focal interstitial nephritis. Acute tubular necrosis.

Discharged on supportive symptomatic therapy with antihypertensive drugs and iron preparations. Repeated history-taking revealed that the patient was obsessed with diets, counted calories, and actively engaged in physical exercise. The mother found in the girl's mobile phone browser history that she had searched for the regimen of ALA intake for weight loss. During the conversation, the girl confirmed taking the supplements but denied an overdose. She was consulted by a psychiatrist, and a diagnosis of anorexia nervosa was established.

Discussion

Under physiological conditions, alpha-lipoic acid and its reduced form (dihydrolipoic acid, DHLA) are potent antioxidants. At very high concentrations, ALA can exhibit prooxidant properties, generate free radicals, disrupt cellular redox balance, and enhance oxidative stress, leading to damage to mitochondria and cell membranes, particularly in the kidneys.[1,2] In toxic doses, mitochondrial dysfunction leads to impaired aerobic glycolysis and lactate accumulation. Severe metabolic lactic acidosis develops, which exacerbates tissue hypoxia and contributes to multiple organ failure. Penetration through the blood-brain barrier leads to direct effects on the central nervous system: seizures, confusion, coma.[5,8] There is no antidote for alpha-lipoic acid poisoning.[1]

One of the causes of acute tubular necrosis is the use of certain nephrotoxic drugs.[3] Data on acute ALA poisonings are extremely rare: 9 cases have been published, of which 6 were in children and adolescents.[3] The literature also describes 37 cases of insulin autoimmune syndrome with hypoglycemia induced by alpha-lipoic acid.[4]

Acute ALA poisoning usually develops within several hours after ingestion, with nausea, vomiting, confusion, and sweating.[5–7] As in our case, Halabi Z. et al. reported the development of refractory seizures and severe metabolic acidosis.[4] Severe cases of ALA poisoning

have been accompanied by thrombocytopenia, rhabdomyolysis, kidney injury with anuria, decreased myocardial contractility and arrhythmias, supraventricular tachycardia, and less commonly, gastrointestinal bleeding.[3,7–10] In our patient, in addition to seizures, there were pronounced signs of lactic acidosis with subsequent development of multiple organ failure.

Outcomes vary from complete recovery within several days to death within 24 hours. The toxic threshold has not been established, as it depends on timely medical care, patient body weight, individual sensitivity, and the duration of drug intake.[3,6,11–13] Kidney injury and anuria have been described in 2 cases (22%), which were associated with multiple organ failure leading to a fatal outcome.[3,7] Clinical manifestations and outcomes of acute alpha-lipoic acid poisoning are presented in Table 1.

Table 1 Comparative characteristics and outcomes of reported clinical cases of acute alpha-lipoic acid poisoning

Clinical case	Amount of drug	Clinical manifestations	Laboratory findings	Outcome	Reference
Woman, 42 years	6 g (92.3 mg/kg)	Refractory seizures, severe agitation, supraventricular tachycardia, decreased myocardial contractility, kidney injury, anuria	Metabolic acidosis, thrombocytopenia, rhabdomyolysis	Fatal	3
Woman, 38 years	6 g	Delirium	Metabolic acidosis, thrombocytopenia, rhabdomyolysis	Discharged in a stabilized condition	9
Woman, 22 years	18 g	Altered mental status, confusion, tachycardia, tachypnea, horizontal nystagmus, moderate hypotension	Decompensated metabolic acidosis, T-wave inversion on ECG	Discharged on day 3	10
Adolescent, 14 years	6 g (150 mg/kg)	Seizures, decreased myocardial contractility, anuria, multiple organ failure	Thrombocytopenia, coagulopathy	Fatal	7
Child, 16 months	1800 mg	Confusion, refractory seizures, gastrointestinal bleeding	Metabolic acidosis	Discharged on day 12	8
Child, 14 months	Dose not specified	Lethargy, ongoing involuntary movements, refractory myoclonic seizures	Mild metabolic acidosis	Discharged on day 8	11
Child, 20 months	2400 mg (226 mg/kg)	Nausea, vomiting, lethargy, involuntary movements 4 hours after ingestion	Laboratory changes not detailed	Discharged on day 5	5
Elderly woman	6 g	Confusion, nausea, vomiting, sweating, seizure-like activity	Lactic acidosis	Discharged on day 3	6
Adolescent, 16 years	1800 mg (30 mg/kg)	Confusion, vomiting, involuntary movements	Hyperglycemia, leukocytosis, metabolic acidosis, coagulopathy	Discharged on day 5	12

Conclusion

The presented clinical case demonstrates the possibility of a life-threatening condition in a child taking alpha-lipoic acid for weight loss against the background of anorexia nervosa. There are no descriptions in the literature of surviving patients with multiple organ failure, anuria, and acute kidney injury with anuria due to alpha-lipoic acid poisoning. The severity of the condition required differential diagnosis with hemolytic uremic syndrome and thrombotic thrombocytopenic purpura. The tubular necrosis identified on kidney biopsy confirmed drug-induced kidney injury. Timely initiation of renal replacement therapy made it possible to achieve a favorable outcome.

This case expands the understanding of the spectrum of manifestations and outcomes of alpha-lipoic acid poisoning and draws the attention of physicians to the need for thorough history-taking in children and adolescents with eating disorders and unexplained severe multiple organ dysfunction.

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None.

Conflicts of interest

The authors declare that there is no conflicts of interest.

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