

Survived Despite High Creatinine

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ABSTRACT The importance of creatinine level in determining pathology in the body is still unclear. When creatinine is evaluated with other kidney function tests, it is thought to be a sign of another problem. In this study, we examined the findings of a 66-year-old patient with no history of chronic disease, whose creatinine level was measured well above the reference range (46.78 mg/dL). Despite these results, our patient survived with treatment. In conclusion, appropriate treatment can improve the prognosis, even at alarming high creatinine. Prognosis may be inversely related to creatinine level. Also, with routine tests such as creatinine, patients can be followed up, and the current disease diagnosis can be investigated.

Keywords: Acute renal failure; creatinine; dialysis

Acute renal failure begins with nephron damage due to ischemic, toxic, traumatic or any other reason and progresses with the development of tubular damage.¹ Filtration is impaired, and blood urea nitrogen (BUN), urea and creatinine values in the blood increase, and the amount of urine decrease. Treatment should be aimed at preventing further nephron damage and its chronicity. Nephrotoxicity is reduced by dialysis, which is one of the best options.

Creatinine is formed by protein metabolism in the kidneys, small intestine, and pancreas mucosa. Most of it is stored in the muscles. In healthy people, creatinine is filtered by the kidneys and is not reabsorbed. Its value in blood gives more specific information at every stage of kidney failure compared to its importance in urine. Like BUN, it is more reliable because it is less affected by diet and physiological conditions.² Its measurement is inexpensive and practical.

There is limited data on how creatinine level affects the severity of kidney damage and the prognosis. This article will discuss a case with an excellent clinical course with dialysis and rapid recovery of the examination results despite the high creatinine value.

CASE REPORT

A 66-year-old male patient applied to the emergency department with complaints of vomiting and weakness 3-4 times a day for 1-2 weeks. He had no comorbidity or history of chronic drug use. He stated that he had taken non-steroidal anti-inflammatory drugs for the pain that developed in the last few days. The patient had a history of smoking 1 pack a day/for 35 years and had no history of alcohol use. Body mass index was 22 kg/m² (60 kg/165 cm). Blood pressure was 130/70, heart rate was 102, and SPO₂ was 98%. His general condition was good. Glaskow Coma Scale was 15. There was no neurological

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TABLE 1: The patient's test results.

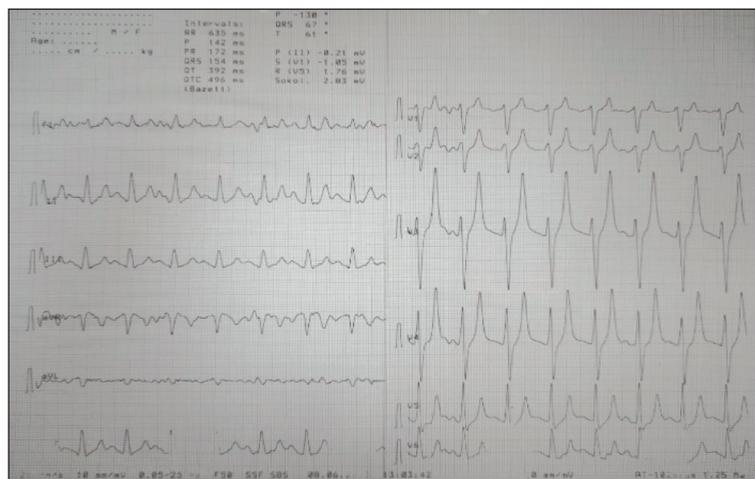
	Application day	3 rd the day of HD	Discharge
Hgb (g/dL)	6.8	7	10.5
Hct (%)	22.2	22.1	32.6
PLT (10 ⁹ /L)	227	110	277
MCV (fL)	91.7	92.7	88.1
WBC (10 ⁹ /L)	9.37	9.69	11.1
Urea (mg/dL)	603	169	154
Creatinine (mg/dL)	46.78	13.04	5.49
Potassium (mmol/L)	8.5	4	4.8
Sodium (mmol/L)	139	147	135
GFR (mL/min/1.73 m ²)	1	4	10
CRP (mg/dL)	1.72	2.67	1.18
pH	7.145	7.38	7.44
PCO ₂ (mmHg)	27.6	39.8	34.7
HCO ₃ (mg/dL)	9.3	23	23.3
Lactate (mg/dL)	14.1	13.7	12.7

Hgb: Haemoglobin; Hct: Haematocrit; PLT: Platelet; MCV: Mean corpuscular volume; WBC: White blood cell; GFR: Glomerular filtration rate; CRP: C-reactive protein; pH: Blood acid-base balance; PCO₂: Partial carbon dioxide; HCO₃: Bicarbonate; HD: Hemodialysis.

deficit, and lung exams were bilaterally regular. Abdominal examination findings were normal, with no bloody vomit or rectal bleeding. In the blood tests taken, pH: 7.14, PCO₂: 27 mmHg, HCO₃: 9.3 mg/dL, haemoglobin: 6.5 g/dL, hematocrit: 21%, urea: 478 mg/dL, creatinine: 46.78 mg/dL, sodium 139 mmol/L, potassium: 8.5 mmol/L, C-reactive protein (CRP): 1.72 mg/dL, glomerular filtration rate (GFR): 1 mL/minute/1.73 m², other tests were within the normal range (Table 1). Electrocardiogram was in nor-

mal sinus rhythm, with a sharp T-wave (Figure 1). A bladder catheter was inserted, and 50 cc urine output was achieved. Abdominal tomography showed bilateral Grade 2 hydronephrosis (Figure 2).

According to the patient's findings, acute renal failure and emergency dialysis indication were considered, and internal medicine was consulted. Urgent haemodialysis was applied to the patient, who was followed up in the intensive care unit. Haemodialysis was applied to the patient for 3 consecutive days, after which it continued every other day. During his stay in the hospital, erythrocyte suspension was inserted, and a hemogram was followed. Antibiotherapy treatment and hydration were started. Considering partial postrenal obstruction, the urology implanted a left-sided double J stent. Urine output was 2,000 cc/day. Renal failure was thought to be related to the use of analgesics. A kidney biopsy was not performed on the patient. Blood and urine tests were done. Anti-nuclear antibody and hepatitis tests were negative. A urine culture was taken due to elevated CRP and leukocytes in the urine test. *Acinetobacter* spp. and *Enterococcus faecium* were found to grow. Tamsulosin hydrochloride and piperacillin-tazobactam were added to the treatment. Fundamental examination showed signs of Grade-2 hypertensive retinopathy. The echocardiography report showed Type 1 diastolic dysfunction, mild mitral insufficiency, and left ventricular concentric hypertrophy. After 21 days of follow-up, urea decreased to

**FIGURE 1:** Electrocardiogram demonstrates peaked T waves consistent with hyperkalemia.

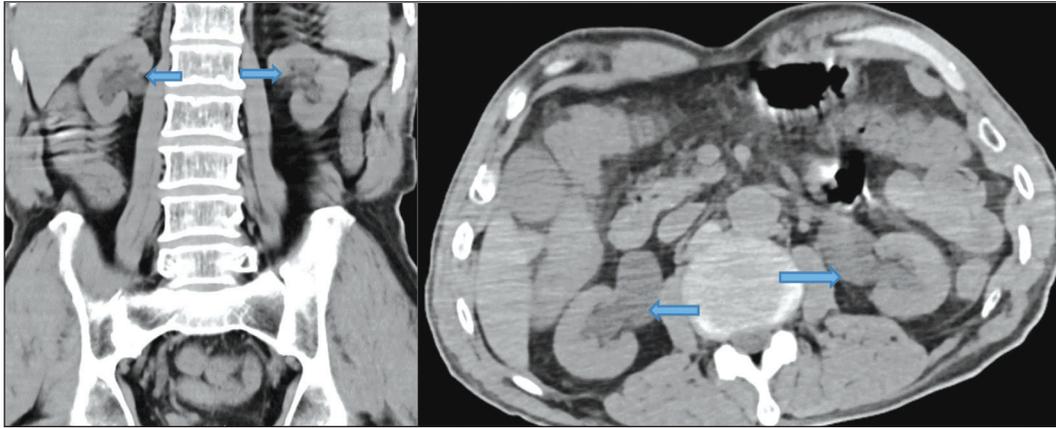


FIGURE 2: Coronal and axial section of the abdominal computed tomography demonstrates bilateral Grade 2 hydronephrosis (blue arrows).

154 mg/dL and creatinine to 5.49 mg/dL (Table 1). GFR was 10 mL/min/1.73 m² at discharge. Control abdominal computed tomography showed dilatation in the right ureter while improvement in the left. He was discharged with antihypertensive treatment. Haemodialysis was planned 3 times a week. The double J stent was replaced 2 months later. A urologist and nephrologist followed him up.

Informed consent has been taken from the patient.

DISCUSSION

In many diseases, measurement of kidney function tests guides the clinician. Significantly changes in creatinine level help determine treatment. Thus, findings such as kidney damage, whether a toxic state has developed, and the level of filtration can be detected in advance. Schillaci et al. found that the risk of developing cardiovascular pathology increased. The prognosis worsened when the creatinine value increased over time in patients with hypertension (HT) who did not receive treatment.³ It is recommended to check the creatinine value in the follow-up of Type-1 and Type-2 diabetes mellitus (DM) patients.⁴ Especially in the group with vascular pathology, such as patients with HT and DM, the follow-up of simple tests such as creatinine may be helpful to predict organ damage or to follow the success of the treatment if they are receiving treatment. Our patient's eye and cardiac examinations determined that there might be uncontrolled HT, and blood tests improved

rapidly after the treatments were given. Treatment was initiated with the diagnosis of post-renal acute renal failure. Because minimal cardiac failure was detected on echocardiography, prescribed HT treatment was thus increasing the patient's quality of life.

A few cases of very high creatinine levels have been reported in the literature. The most elevated measured serum creatinine (73.8 mg/dL) belongs to a 23-year-old male with a history of kidney transplant.⁵ The patient with the second highest creatinine, measured 61.3 mg/dL, was 20 years old and had a history of HT.⁶ When the other patients are examined, it is seen that most of them have chronic diseases, and the average age is in their 20s. It was stated that all patients benefited from haemodialysis, and all survived. The case in our study, on the other hand, is different from the others due to the absence of a known chronic disease history and its age. Accordingly, a high creatinine level does not affect the prognosis regardless of age and comorbidity.

In the study of Cerdá et al. with anuric patients, they found that the prognosis of patients with high creatinine levels was better than patients with lower creatinine. These patients were thought to have pre-existing chronic renal failure.⁷ Our patient was also anuric and responded quickly to the treatment. The situation might be exacerbated by adding minimal infection to the existing pathology. The high infection test (such as C-RP) confirms this thesis. Therefore, there is a high probability of pre-existing chronic kidney disease.

The kidneys can be considered a warning organ for investigating the real problem. When such a situation develops, kidney damage should be minimised with additional filtering methods such as dialysis, and other organs should be prevented from being affected.

Today, kidney failure is a big problem in every age group. Early diagnosis and prevention of cardiovascular pathologies may be possible with a simple test such as creatinine.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: Bedriye Feyza Kurt; **Design:** Bedriye Feyza Kurt, Oya Güven; **Control/Supervision:** Lale Tuna, Erman Kurt; **Data Collection and/or Processing:** Metehan Sarıcı, Bedriye Feyza Kurt; **Analysis and/or Interpretation:** Oya Güven, Bedriye Feyza Kurt; **Literature Review:** Metehan Sarıcı, Erman Kurt; **Writing the Article:** Bedriye Feyza Kurt, Oya Güven; **Critical Review:** Lale Tuna, Erman Kurt; **References and Fundings:** Erman Kurt, Metehan Sarıcı; **Materials:** Oya Güven.

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