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Rhabdomyolysis, Acute Kidney Injury, and Exudative Retinal Detachment Associated with Nonsteroidal Anti-Inflammatory Drug and Herbal Product Use: A Case Report

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ABSTRACT

Background: Rhabdomyolysis is a potentially life-threatening condition that may lead to acute kidney injury (AKI). Although nonsteroidal anti-inflammatory drugs (NSAIDs) are widely used, diclofenac-associated rhabdomyolysis is exceedingly rare. Herbal products, often perceived as harmless, may also contribute to nephrotoxicity through contamination or pharmacokinetic interactions.

Case Report: A 20-year-old woman presented with muscle pain, weakness, and dark-colored urine after using diclofenac potassium and a homemade herbal mixture. Laboratory findings revealed severe rhabdomyolysis and AKI. Kidney biopsy confirmed myoglobin-induced acute tubular injury. During treatment, the patient developed hypertension and blurred vision; ophthalmologic evaluation revealed exudative retinal detachment. Hemodialysis, ultrafiltration, and blood pressure control resulted in complete renal and visual recovery.

Conclusion: This case highlights the unpredictable toxicity that may arise from the combined use of NSAIDs and herbal products. Clinicians should routinely inquire about herbal supplement use, particularly in patients presenting with unexplained rhabdomyolysis or AKI.

Keywords: Acute kidney injury, herbal product, nonsteroidal anti-inflammatory drug, retinal detachment, rhabdomyolysis.

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INTRODUCTION

Rhabdomyolysis is a clinical syndrome resulting from the breakdown of skeletal muscle cells and may lead to severe systemic complications. Acute kidney injury (AKI) is one of the most serious complications of rhabdomyolysis, with a reported incidence ranging from 10% to 55%. Patients typically present with myalgia, dark-colored urine, muscle weakness, and markedly elevated creatine kinase (CK) levels. In adults, rhabdomyolysis is most commonly associated with trauma, infections, and toxic exposures. The pathophysiology of kidney injury involves renal vasoconstriction, hypovolemia, myoglobin-mediated toxicity, and tubular obstruction. Although nonsteroidal anti-inflammatory drugs (NSAIDs) have a broad adverse-effect profile,

rhabdomyolysis is an exceptionally rare complication, with only a limited number of cases reported. While the precise role of NSAIDs in the development of rhabdomyolysis remains unclear, mitochondrial dysfunction, increased oxidative stress, and impaired stabilization of the myocyte membrane have been proposed as plausible mechanisms. The primary therapeutic goals include elimination of the precipitating trigger, prompt and adequate volume replacement, correction of electrolyte and acid–base disturbances, and initiation of renal replacement therapy when indicated.[1] Herbal products are widely used worldwide and are often consumed without physician awareness because they are perceived as "natural." However, nephrotoxicity may occur due to uncertain composition, contamination, improper processing, and pharmacodynamic or pharmacokinetic interactions.[2] In addition, herbal products may alter drug metabolism and renal elimination via cytochrome P450 enzymes, P-glycoprotein, and organic anion transporters (OATs), potentially increasing the risk of unexpected toxicities. Therefore, herbal product use should be routinely queried in cases of unexplained rhabdomyolysis or AKI. Rhabdomyolysis has been reported following the use of various herbal supplements.[3] Hypertension and hypervolemia may develop during the course of AKI and can affect the choroidal circulation, disrupting the retinal pigment epithelium barrier and leading to exudative retinal detachment. This ocular manifestation often resolves rapidly with appropriate blood pressure and volume control.[4] Exudative retinal detachment may also occur secondary to multiple etiologies beyond malignant hypertension, including inflammatory, infectious, autoimmune, neoplastic, and drug-related causes.[5] Nevertheless, in cases related to hypertension and volume overload, prompt clinical improvement following blood pressure and volume optimization is characteristic.

Herein, we report a distinctive case that contributes to the literature by describing the concurrent occurrence of rhabdomyolysis, AKI, and hypertension-associated exudative retinal detachment following the concomitant use of NSAIDs and unregulated herbal products.

CASE REPORT

A 20-year-old woman presented with fatigue, diffuse myalgia, restricted mobility, and dark-colored urine. Following an upper respiratory tract infection, she had taken a total of 1,000 mg of diclofenac potassium over seven days and had concurrently consumed a homemade herbal mixture ("Atom Tea") containing ginger, hibiscus, turmeric, allspice, clove, cinnamon, and galangal. Physical examination revealed tachycardia, oliguria, +2 pretibial edema, and marked muscle weakness. Laboratory testing demonstrated

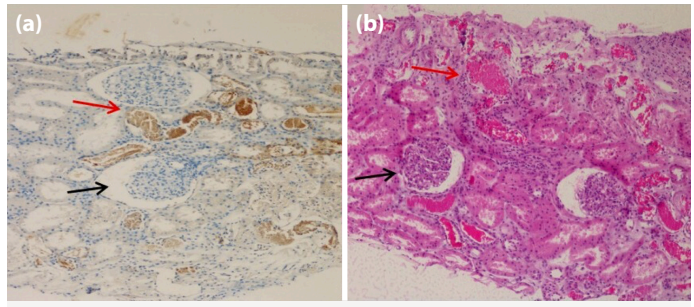

Figure 1. (a) Kidney core needle biopsy showing myoglobin-positive cast material within the tubular lumens (red arrows). Black arrows indicate glomeruli with preserved histological architecture (×200, immunoperoxidase). **(b)** Kidney core needle biopsy demonstrating tubular cast material (red arrows) with glomeruli within normal histological limits (black arrows) (×200, hematoxylin and eosin staining).

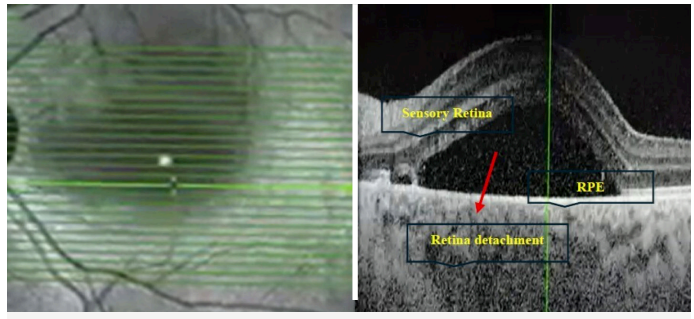

Figure 2. Optical coherence tomography (OCT) scan showing a cross-sectional view of the retina. The central dome-shaped elevation indicates retinal detachment. Retinal layers are clearly visualized; hyperreflective bands represent structural layers, while hyporeflective areas correspond to fluid accumulation. The green line marks the scanning axis used to obtain this cross-sectional image.

elevated renal function parameters and muscle enzyme levels (Table 1). Urine microscopy showed granular casts. Renal ultrasonography revealed bilateral grade 2 increased echogenicity. Kidney biopsy demonstrated pigmented tubular casts, interstitial edema, and mild mononuclear infiltration; myoglobin positivity on immunohistochemistry confirmed acute tubular injury (Fig. 1).

Isotonic fluid therapy was initiated at 200 mL/kg/day. Although early fluid resuscitation is recommended in rhabdomyolysis, the patient was closely monitored clinically and hemodynamically, as aggressive hydration in the setting of oliguria may increase the risk of hypervolemia and hypertension. Hemodialysis was initiated following the development of hyperkalemia,

Table 1. Laboratory testing

Laboratory parameter	Day 1	Day 7	Day 14	Day 21	Post-discharge follow-up
BUN, mg/dL	61.1	44.1	30.8	9.0	7.0
Creatinine, mg/dL	3.98	5.74	4.9	1.5	1.14
eGFR, mL/min	15.3	9.83	11.9	47	69.3
Sodium, mEq/L	133	136	140	145	141
Potassium, mEq/L	6.33	3.54	3.65	3.95	3.71
Calcium, mEq/L	5.62	10.2	7.98	7.79	7.9
Magnesium, mEq/L	1.33	0.86	0.69	0.41	0.7
Creatinine kinase, IU/L	38899	7600	856	56	25
Lactate dehydrogenase, u/L	5457	1641	955	692	559
Aspartate transaminase, u/L	6760	353	67	28	27
Alanine transaminase, u/L	1878	650	177	62	38
Proteinuria, g	6.3	1.1	0.3	0.02	0.01
Complete urinalysis	Granular casts				
Urine, blood, and stool cultures	Negative				
C3/C4 levels	Normal				
Anti-PLA2R	Normal				
Albumin, g/dL	3.6	3.01	4.4	3.72	3.9
White blood cell count	21.2	7.8	9.8	8.7	7.6
Hemoglobin	13.2	9.8	10.2	12.6	12.5
Platelet count	187	137	142	157	167
Free kappa/lambda ratio	2.1				
Hemoglobin A1c	5.4				
ANA, anti-dsDNA, ANCA Anti-PLA2R	Normal				
Hepatitis A, B, C, and HIV	Negative				
IgG, IgA, IgM, and IgG4 levels	Normal				
Blood gas (pH/HCO_3^-)	7.30/17	7.40/21	7.38/21	7.40/23	7.39/23
FENa	3%				
C-reactive protein	62	17	21	4	2
Procalcitonin	3.59	0.4	0.1	0.05	0.01

BUN: Blood urea nitrogen; eGFR: Estimated glomerular filtration rate; C3/C4: Complement components 3 and 4; Anti-PLA2R: Anti–phospholipase A2 receptor antibody; FENa: Fractional excretion of sodium; ANA: Antinuclear antibody; anti-dsDNA: Anti–double-stranded DNA antibody; ANCA: Antineutrophil cytoplasmic antibody.

metabolic acidosis, and volume overload. The primary and urgent indication for hemodialysis was life-threatening hyperkalemia, and ultrafiltration was additionally employed to achieve volume control and blood pressure regulation.

During follow-up, the patient developed hypertension and blurred vision. Ophthalmologic evaluation revealed macular edema and exudative retinal detachment (Fig. 2). These retinal findings were interpreted as objective manifestations of end-organ damage secondary to volume overload and uncontrolled hypertension. Hypervolemia and hypertension were managed with antihypertensive therapy and ultrafiltration. On day 16, AKI entered a polyuric phase, and renal function improved rapidly (Fig. 3). By day 19, the retinal findings had completely resolved, and visual acuity returned to baseline. Laboratory parameters also normalized.

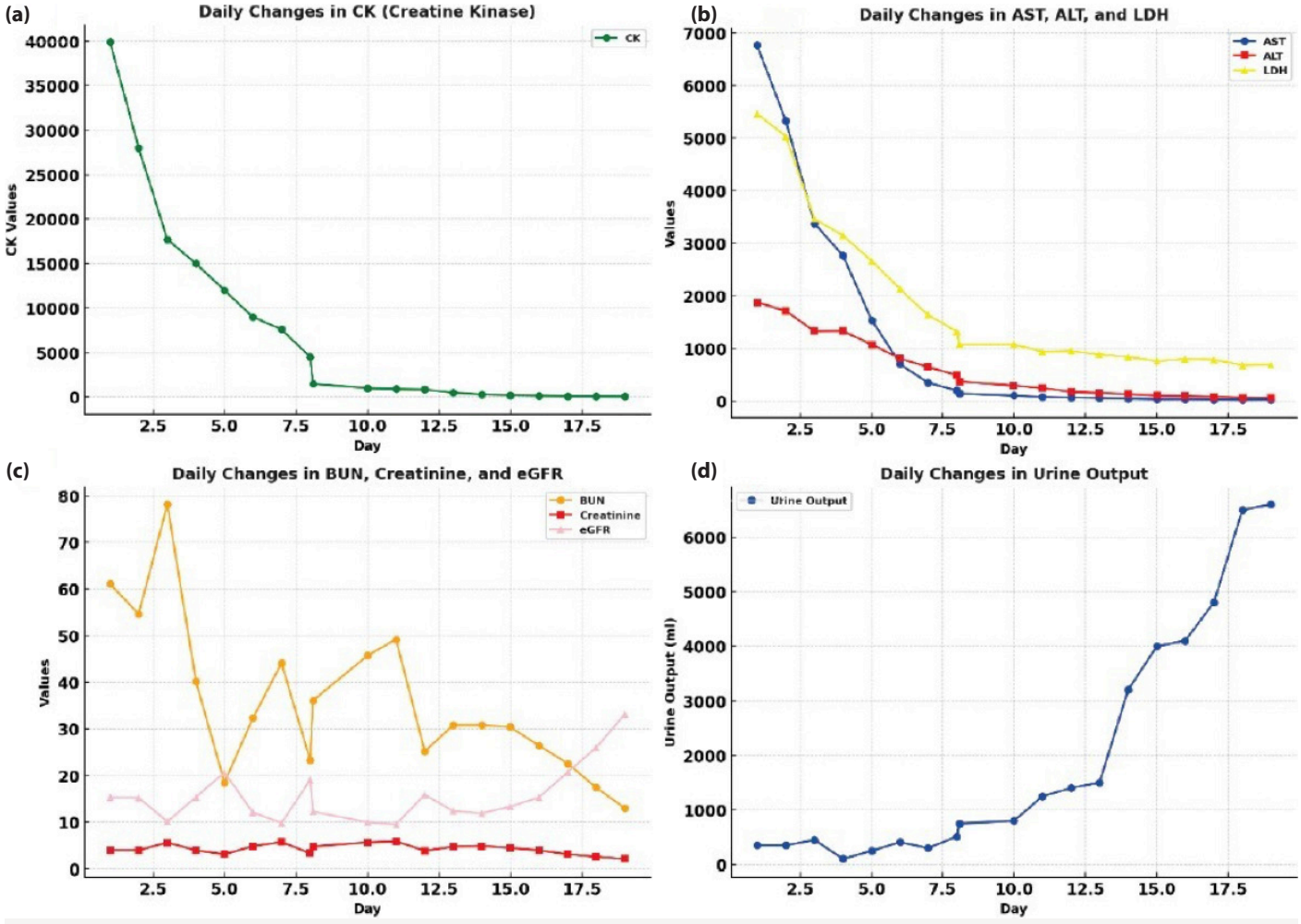

Figure 3. (a) The green arrow shows the day-to-day trend of creatine kinase (CK). **(b)** The blue arrow indicates the trend in aspartate aminotransferase (AST), the red arrow alanine aminotransferase (ALT), and the yellow arrow lactate dehydrogenase (LDH) over time. **(c)** The orange arrow represents daily changes in blood urea nitrogen (BUN), the red arrow creatinine, and the light yellow arrow estimated glomerular filtration rate (eGFR). **(d)** The blue arrow shows changes in urine output over the course of hospitalization.

DISCUSSION

This case represents one of the rare examples in the literature in which diclofenac use and unregulated herbal product consumption were temporally associated with rhabdomyolysis and AKI, accompanied by hypertension-related exudative retinal detachment. AKI develops in approximately 10%–55% of patients with rhabdomyolysis, and 5%–15% may require renal replacement therapy. Although diclofenac-associated rhabdomyolysis is rare, only a limited number of cases have been reported, including a pediatric case. Moreover, analyses of the World Health Organization (WHO) pharmacovigilance database have identified reports of diclofenac-associated rhabdomyolysis, with symptom improvement noted after drug discontinuation in some cases.[6–8] In our patient, although causality cannot be definitively established, the relatively high cumulative dose administered over a short period is noteworthy.

Although several components of the herbal mixture have been suggested to possess nephroprotective properties in experimental settings, herbal products may pose significant clinical risks due to contamination, pesticide residues, heavy metals, or unknown toxic substances. In addition, herbal preparations are known to modulate CYP450 enzymes, P-glycoprotein, and organic anion transporters, potentially affecting diclofenac metabolism and increasing systemic exposure. Such interactions may have amplified muscle and renal toxicity in this case.[9]

The exudative retinal detachment observed in conjunction with rhabdomyolysis and AKI was consistent with a hypertensive crisis and volume overload. Hypertensive retinopathy and serous retinal detachment associated with malignant hypertension have been previously described.[10] In our patient, the bilateral involvement, temporal association with hypertension, and rapid resolution following ultrafiltration strongly support a pathophysiological mechanism driven by volume overload and uncontrolled blood pressure.

Upper respiratory tract infections may precipitate rhabdomyolysis through viral myositis or systemic inflammatory responses. In this case, viral serologies and extended myositis panels were not evaluated, which should be acknowledged as a limitation.

CONCLUSION

This case highlights that herbal products are not inherently safe and that their concomitant use with nonsteroidal anti-inflammatory drugs may precipitate severe and unpredictable toxicities, including rhabdomyolysis and acute kidney injury. A structured assessment of herbal product exposure should therefore be routinely incorporated into the evaluation of unexplained rhabdomyolysis and acute kidney injury. Despite the absence of toxicological, pharmacogenetic, and pharmacokinetic analyses, this report underscores a critical and often overlooked source of preventable drug-related harm.

Ethics Committee Approval: This is a single case report, and therefore ethics committee approval was not required in accordance with institutional policies.

Informed Consent: Written informed consent was obtained from the patient.

Conflict of Interest: The authors have no conflicts of interest to declare.

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