

Severe Influenza A-Associated Rhabdomyolysis Without Acute Kidney Injury: A Case Report

Sean Pack¹, Mahesh Shrestha¹, FNU Aparna²

1. Internal Medicine, St. Francis - Emory Healthcare, Columbus, USA 2. Nephrology, St. Francis - Emory Healthcare, Columbus, USA

Corresponding author: Sean Pack, packsean4@gmail.com

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Abstract

A 26-year-old man without known chronic illness presented with acute myalgias, fatigue, and tea-colored urine and tested positive for influenza A. Urinalysis showed 3+ blood with one red blood cell per high-power field on microscopy, supporting pigmenturia. Creatine kinase peaked at 292,109 U/L, while serum creatinine remained 0.7-0.9 mg/dL throughout hospitalization. He was treated with intravenous isotonic saline, oseltamivir, and serial biochemical monitoring. Symptoms improved, and creatine kinase decreased to 49,878 U/L by discharge without renal replacement therapy. Subsequent myositis antibody testing was negative; however, a family history of similar episodes raised concern for an underlying inherited susceptibility. This case demonstrates that profound influenza-associated muscle injury can occur without documented acute kidney injury and that renal risk should be assessed using serial kidney function, electrolytes, volume status, and urine output rather than creatine kinase magnitude alone.

Categories: Internal Medicine, Nephrology

Keywords: acute kidney injury, creatine kinase, influenza a, pigmenturia, rhabdomyolysis

Introduction

Rhabdomyolysis is a clinical syndrome caused by skeletal muscle breakdown and the release of intracellular constituents, including creatine kinase (CK), myoglobin, and electrolytes, into the circulation. Its presentation ranges from asymptomatic enzyme elevation to muscle pain, weakness, and dark urine, with potential complications, including electrolyte disturbances and acute kidney injury (AKI). Renal injury may result from myoglobin-associated tubular toxicity, intratubular cast formation, and impaired renal perfusion. Although CK is useful for identifying and following muscle injury, its concentration alone does not fully characterize renal risk [1].

Influenza A is a recognized infectious precipitant of rhabdomyolysis. Muscle involvement may be overlooked because myalgias are common during influenza, and severe muscle injury can occur without prominent respiratory manifestations [2]. Disproportionate muscle pain, pigmenturia, or persistent symptoms should therefore prompt consideration of rhabdomyolysis even when the respiratory examination and initial creatinine are reassuring. Published influenza-associated cases have demonstrated heterogeneous renal outcomes, including marked CK elevation without AKI and extreme elevations accompanied by renal failure requiring renal replacement therapy [3,4]. These contrasting courses make concurrent assessment of kidney function, electrolytes, hydration, and urine output clinically important.

We report a 26-year-old man with laboratory-confirmed influenza A whose CK peaked at 292,109 U/L while serum creatinine remained 0.7-0.9 mg/dL throughout hospitalization. The distinctive contribution of this case is the combination of profound biochemical muscle injury, preserved bedside strength, no prominent respiratory compromise, and no documented AKI during an eight-day inpatient course. A family history of similar episodes further raises the possibility that influenza unmasked an inherited susceptibility rather than acting as the sole cause. This case adds to the reported spectrum of influenza-associated rhabdomyolysis and illustrates why the severity of muscle injury, short-term renal outcome, and possible underlying predisposition should be assessed separately.

Case Presentation

A 26-year-old man with no known significant medical history presented after approximately one day of generalized body aches, fatigue, malaise, chills, subjective fever, and tea-colored urine. He denied chest pain or dyspnea, took no chronic medications, and reported no prior diagnosis of rhabdomyolysis or baseline muscle weakness. A family history of similar rhabdomyolysis episodes was documented.

Admission vital signs are presented in Table 1. He was alert and without respiratory distress. Cardiopulmonary and abdominal examinations were unremarkable, with no peripheral edema. Strength was 5/5 in all extremities.

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Parameter	Recorded value	Adult resting reference
Temperature	36.6 °C	36.5-37.3 °C
Systolic blood pressure	146 mmHg	90-120 mmHg
Diastolic blood pressure	94 mmHg	60-80 mmHg
Respiratory rate	16 breaths/minute	12-18 breaths/minute
Oxygen saturation	100% on room air	95-100%
Heart rate	85	60-100 beats/minute

TABLE 1: Vital signs at presentation

Normal adult resting ranges are shown [5,6].

Abbreviations and units: °C, degrees Celsius; mmHg, millimeters of mercury

Admission complete blood count showed a white blood cell count of 6.8 × 10³/μL, hemoglobin of 15.4 g/dL, and platelet count of 190 × 10³/μL. The complete blood count with differential is presented in Table 2.

Test	Result	Adult normal range
White blood cell count	$6.8 \times 10^3/\mu\text{L}$	$4.5\text{--}11.0 \times 10^3/\mu\text{L}$
Red blood cell count	$5.13 \times 10^6/\mu\text{L}$	$4.3\text{--}5.9 \times 10^6/\mu\text{L}$
Hemoglobin	15.4 g/dL	13.5–17.5 g/dL
Hematocrit	42.1%	41–53%
Mean corpuscular volume	82 fL	80–100 fL
Mean corpuscular hemoglobin	30.1 pg	25–35 pg
Mean corpuscular hemoglobin concentration	36.6 g/dL	31–36 g/dL
Red cell distribution width	12.2%	11.5–14.5%
Platelet count	$190 \times 10^3/\mu\text{L}$	$150\text{--}400 \times 10^3/\mu\text{L}$
Mean platelet volume	8.3 fL	7.5–12.0 fL
Neutrophils	48.2%	40–70%
Lymphocytes	39.4%	20–40%
Monocytes	7.3%	2–8%
Eosinophils	4.6%	1–4%
Basophils	0.5%	0.5–1%
Monocyte distribution width	23.6	Assay-specific; see legend
Absolute neutrophil count	$3.3 \times 10^3/\mu\text{L}$	$1.5\text{--}7.5 \times 10^3/\mu\text{L}$
Absolute lymphocyte count	$2.7 \times 10^3/\mu\text{L}$	$1.0\text{--}4.0 \times 10^3/\mu\text{L}$
Absolute monocyte count	$0.5 \times 10^3/\mu\text{L}$	$0.2\text{--}0.8 \times 10^3/\mu\text{L}$
Absolute eosinophil count	$0.3 \times 10^3/\mu\text{L}$	$0.0\text{--}0.5 \times 10^3/\mu\text{L}$
Absolute basophil count	$0.0 \times 10^3/\mu\text{L}$	$0.0\text{--}0.2 \times 10^3/\mu\text{L}$

TABLE 2: Complete blood count with differential at presentation

Adult normal ranges are shown, with male ranges for red blood cell count, hemoglobin, and hematocrit [7]. The assay-specific reference interval for monocyte distribution width was unavailable.

Abbreviations and units: μL , microliter; g/dL, grams per deciliter; fL, femtoliter; pg, picogram

Initial creatinine was 0.9 mg/dL, blood urea nitrogen (BUN) was 14 mg/dL, potassium was 3.5 mmol/L, aspartate aminotransferase (AST) was 362 U/L, and alanine aminotransferase (ALT) was 126 U/L. Bilirubin and alkaline phosphatase were normal. Creatine kinase (CK) was initially reported as >1,850 U/L. Additional admission results included bicarbonate of 31 mmol/L, calcium of 9.0 mg/dL, albumin of 4.3 g/dL, and total bilirubin of 0.7 mg/dL. Prothrombin time was 13.3 seconds, and the international normalized ratio was 0.93. Influenza A testing was positive, while influenza B and severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) testing were negative (Table 3).

Test	Result	Normal range or expected result
Sodium	137 mmol/L	136-146 mmol/L
Potassium	3.5 mmol/L	3.5-5.0 mmol/L
Chloride	100 mmol/L	95-105 mmol/L
Bicarbonate	31 mmol/L	22-28 mmol/L
Blood urea nitrogen	14 mg/dL	7-18 mg/dL
Creatinine	0.9 mg/dL	0.6-1.2 mg/dL
Glucose	98 mg/dL	70-100 fasting; <140 random (mg/dL)
Calcium	9.0 mg/dL	8.4-10.2 mg/dL
Total protein	7.5 g/dL	6.0-7.8 g/dL
Albumin	4.3 g/dL	3.5-5.5 g/dL
Total bilirubin	0.7 mg/dL	0.1-1.0 mg/dL
Alkaline phosphatase	45 U/L	25-100 U/L
Aspartate aminotransferase	362 U/L	12-38 U/L
Alanine aminotransferase	126 U/L	10-40 U/L
Estimated glomerular filtration rate	118 mL/min/1.73 m ²	≥90 mL/min/1.73 m ²
Anion gap	6 mmol/L	Common comparator: 8-12 mmol/L
Lipase	68 U/L	13-60 U/L
Creatine kinase	>1,850 U/L	39-308 U/L (male)
Prothrombin time	13.3 seconds	11-15 seconds
International normalized ratio	0.93	0.8-1.2
Partial thromboplastin time	27.4 seconds	25-40 seconds
Influenza A	Positive	Negative
Influenza B	Negative	Negative
SARS-CoV-2	Negative	Negative

TABLE 3: Initial chemistry, coagulation, and respiratory viral testing

Adult normal ranges and expected qualitative results are shown [7,8]. Glucose fasting status was not reported. An estimated glomerular filtration rate ≥90 is a clinical category, not a stand-alone exclusion of kidney disease. The initial creatine kinase value was a lower-bound report.

Abbreviations and units: SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; mmol/L, millimoles per liter; mg/dL, milligrams per deciliter; g/dL, grams per deciliter; U/L, units per liter; mL/min/1.73 m², milliliters per minute per 1.73 square meters

Urinalysis showed 3+ blood with one red blood cell per high-power field on microscopy, supporting pigmenturia in this setting. Urine was light orange and clear, with specific gravity 1.039, pH 6.0, trace ketones, and protein 300 mg/dL (Table 4). Direct myoglobin measurement was unavailable. These findings accompanied the reported tea-colored urine and supported further assessment of muscle injury despite initially preserved renal function.

Test	Result	Normal range or expected result
Color	Light orange	Light yellow to yellow
Appearance	Clear	Clear
pH	6.0	5.0-8.0
Specific gravity	1.039	1.001-1.030
Glucose	Normal	Negative
Bilirubin	Negative	Negative
Ketones	Trace	Negative
Blood	3+	Negative
Protein	300 mg/dL	Negative to trace
Urobilinogen	2 mg/dL	Assay-dependent; e.g., 0.2 mg/dL
Nitrite	Negative	Negative
Leukocyte esterase	Negative	Negative
White blood cells	18 cells/HPF	0-5 cells/HPF
Red blood cells	1 cell/HPF	0-2 cells/HPF
Bacteria	1+	None seen
Squamous epithelial cells	Occasional	None to few
Mucus	1+	None to small amount

TABLE 4: Urinalysis at presentation

Normal ranges and expected findings are shown [9]. Urine cell counts are expressed as cells per high-power field. Semiquantitative grades are retained as reported.

Abbreviations and units: pH, measure of hydrogen ion activity; HPF, high-power field; mg/dL, milligrams per deciliter. Specific gravity is dimensionless.

He was admitted and treated with intravenous isotonic normal saline and oseltamivir. Potassium was supplemented, and CK, electrolytes, and renal function were monitored serially. Generalized muscle soreness and tenderness persisted initially, but strength remained preserved. He maintained urine output and hemodynamic stability without clinical volume overload.

CK rose to 173,895 U/L on hospital day 2 and peaked at 292,109 U/L on day 5. At the peak, creatinine was 0.7 mg/dL, BUN was 12 mg/dL, and potassium was 4.4 mmol/L. AST peaked at 1,122 U/L on day 5; ALT reached 497 U/L on day 6. Bilirubin remained normal. CK subsequently declined to 204,693, 121,781, and 49,878 U/L on days 6, 7, and 8, respectively (Table 5). Creatinine remained 0.7-0.9 mg/dL throughout hospitalization, with no documented AKI or need for renal replacement therapy.

Hospital day	CK (U/L)	Creatinine (mg/dL)	BUN (mg/dL)	AST (U/L)	ALT (U/L)
Reference	39-308	0.6-1.2	7-18	12-38	10-40
Day 1	>1,850	0.9	14	362	126
Day 2	173,895	0.8	11	Not reported	Not reported
Day 3	209,496	0.7	9	Not reported	Not reported
Day 4	256,728	0.7	8	877	356
Day 5	292,109	0.7	12	1,122	479
Day 6	204,693	0.7	10	910	497
Day 7	121,781	0.7	15	644	471
Day 8 / discharge	49,878	0.7	11	360	405

TABLE 5: Serial creatine kinase, renal function, and aminotransferases

The reference row shows adult normal ranges, with the male range for CK [7,8]. Not reported denotes unavailable measurements, not normal values. Day 1 CK was reported only as >1,850 U/L.

Abbreviations and units: CK, creatine kinase; BUN, blood urea nitrogen; AST, aspartate aminotransferase; ALT, alanine aminotransferase; U/L, units per liter; mg/dL, milligrams per deciliter

The initial CK result was a lower-bound report rather than an exact concentration, so the precise magnitude of the increase from presentation cannot be calculated. Subsequent numeric measurements established both the peak and the sustained decline before discharge. CK was 209,496 U/L on day 3 and 256,728 U/L on day 4, when AST was 877 U/L, and ALT was 356 U/L (Table 5).

Evaluation for alternative contributors showed normal thyroid-stimulating hormone and a negative urine toxicology screen. A subsequently obtained extended myositis antibody panel was negative (Table 6). Genetic or metabolic testing was not documented. Corticosteroids were not initiated because symptoms and CK improved with supportive treatment.

Test	Result	Normal range or expected result
Thyroid-stimulating hormone	2.07 mIU/L	0.4-4.0 mIU/L
Amphetamines	Negative	Negative
Barbiturates	Negative	Negative
Benzodiazepines	Negative	Negative
Cannabinoids	Negative	Negative
Cocaine	Negative	Negative
Opiates	Negative	Negative
Methadone	Negative	Negative
Tricyclic antidepressants	Negative	Negative
Phencyclidine	Negative	Negative
Anti-Jo-1	<20 units (negative)	<20 units (negative)
Anti-TIF-1γ	<20 units (negative)	<20 units (negative)
Anti-MDA5	<20 units (negative)	<20 units (negative)
Anti-NXP2	<20 units (negative)	<20 units (negative)
Anti-SAE1	<20 units (negative)	<20 units (negative)
Anti-PM/Scl-100	<20 units (negative)	<20 units (negative)
Anti-SS-A 52 kD	<20 units (negative)	<20 units (negative)
Anti-U1 RNP	<20 units (negative)	<20 units (negative)
Anti-PL-7	Negative	Negative
Anti-PL-12	Negative	Negative
Anti-EJ	Negative	Negative
Anti-OJ	Negative	Negative
Anti-SRP	Negative	Negative
Anti-Mi-2	Negative	Negative
Anti-Ku	Negative	Negative
Anti-U2 RNP	Negative	Negative
Anti-U3 RNP	Negative	Negative

TABLE 6: Thyroid testing, urine toxicology, and myositis antibody panel

The normal adult thyroid range [7] and negative antibody thresholds [10] are shown. Toxicology reference results are qualitative; a negative screen does not exclude all exposures.

Abbreviations and antigen names: TIF-1γ, transcription intermediary factor 1-gamma; MDA5, melanoma differentiation-associated protein 5; NXP2, nuclear matrix protein 2; SAE1, small ubiquitin-like modifier activating enzyme subunit 1; PM/Scl-100, 100-kilodalton polymyositis/scleroderma-associated antigen; SS-A 52 kD, 52-kilodalton Sjögren syndrome antigen A (Ro52); RNP, ribonucleoprotein; SRP, signal recognition particle; Jo-1, histidyl-transfer RNA synthetase; PL-7, threonyl-transfer RNA synthetase; PL-12, alanyl-transfer RNA synthetase; EJ, glycyl-transfer RNA synthetase; OJ, isoleucyl-transfer RNA synthetase-associated antigen; Mi-2, nucleosome remodeling antigen; Ku, DNA-binding Ku antigen; U1 and U2 RNP, U1 and U2 small nuclear ribonucleoproteins; U3 RNP, fibrillarin-associated ribonucleoprotein; RNA, ribonucleic acid; DNA, deoxyribonucleic acid; kD, kilodalton; mIU/L, milli-international units per liter

By discharge on day 8, myalgias had substantially improved and urine discoloration had resolved. Creatinine was 0.7 mg/dL, AST was 360 U/L, and ALT was 405 U/L. He received instructions to maintain hydration, temporarily avoid strenuous activity, and obtain outpatient follow-up.

Discussion

This case demonstrates that extreme CK elevation can coexist with preserved renal function during influenza A infection. Published cases show variable biochemical severity and renal outcomes. Ibrahim et al. reported a peak CK of 28,216 U/L [2]. Velho et al. described CK of 59,407 U/L without AKI [3]. Bamber et al. reported CK exceeding 1,000,000 U/L in a patient with influenza A and sickle cell trait who required continuous renal replacement therapy [4]. Our patient's course adds an example of CK approaching 300,000 U/L without a creatinine increase; it does not establish a CK threshold below which renal complications are unlikely.

The biological basis of influenza-associated muscle injury remains incompletely established. Desdouts et al. demonstrated productive infection of cultured human skeletal muscle cells by pandemic and seasonal influenza A(H1N1) viruses, providing experimental support for direct muscle involvement [11]. This finding establishes biological plausibility but cannot identify the mechanism in an individual patient. In the present case, influenza was identified through clinical diagnostic testing; no muscle biopsy, muscle viral testing, or influenza subtyping was available. Accordingly, this report describes an association supported by timing and clinical findings, without claiming direct viral invasion or histologically confirmed viral myositis.

The heme-positive urine with few erythrocytes supported pigmenturia. In the context of severe muscle injury, myoglobin was the likely pigment, although dipstick testing cannot distinguish myoglobin from hemoglobin. CK measures muscle injury rather than nephrotoxic exposure directly. Myoglobin-related renal injury involves tubular toxicity, cast formation, and altered renal perfusion; volume status and other clinical factors influence the outcome [1]. Consequently, CK should be interpreted alongside renal function, electrolytes, acid-base status, and urine output.

Preserved bedside strength is also relevant to recognition. This patient had diffuse pain and tenderness with 5/5 strength, illustrating that severe biochemical muscle injury need not produce an obvious motor deficit. Likewise, the absence of dyspnea, hypoxemia, or radiographic pulmonary disease did not reflect the severity of the extrapulmonary presentation. In this encounter, the combination of muscle symptoms and abnormal urine appearance was more informative than the respiratory examination. These observations favor symptom-directed evaluation of influenza complications rather than using the apparent severity of respiratory disease to decide whether muscle injury is possible.

Prompt isotonic crystalloid administration and serial monitoring are central components of rhabdomyolysis management. Fluid delivery should be individualized to urine output and volume status to avoid fluid overload; routine bicarbonate or diuretics for AKI prevention are not supported by the American Association for the Surgery of Trauma consensus document [12]. In this patient, early hydration may have contributed to renal preservation, but a single case cannot establish treatment causality. Exact infusion rates, cumulative fluid balance, and quantitative urine output were unavailable, limiting assessment of the resuscitation strategy.

Continued surveillance remains necessary while muscle injury evolves because a reassuring admission chemistry panel does not describe the entire subsequent course. Potassium in this patient was 3.5 mmol/L initially and 4.4 mmol/L at peak CK. These measured values should not be extrapolated to unreported intervals or interpreted as evidence that profound muscle injury cannot produce electrolyte complications. Consensus guidance emphasizes serial electrolyte assessment and reserves renal replacement therapy for established clinical indications rather than prophylactic treatment of an elevated CK concentration [12]. No such intervention was required during the documented hospitalization.

The aminotransferase pattern also requires careful interpretation. AST predominated, and its decline broadly paralleled CK, while bilirubin remained normal. Skeletal muscle can contribute to both AST and ALT elevations during rhabdomyolysis [13]. This pattern supports a muscle contribution but does not independently exclude concomitant hepatic injury. Persistent or discordant abnormalities would warrant further assessment.

The timing of the enzyme peaks provides additional context: AST peaked with CK on day 5, whereas ALT reached its highest reported value on day 6 and remained elevated at discharge. Different trajectories of CK and aminotransferases can occur during recovery from muscle injury [13]. Thus, an ALT value that decreases more slowly than CK does not automatically establish a separate hepatic disorder. Interpretation should incorporate the overall trend, bilirubin, other liver-associated measurements, exposure history, and clinical findings rather than relying on the label of an abnormal liver panel alone.

Influenza A was the probable acute trigger given the temporal association with symptomatic, laboratory-confirmed infection. Normal thyroid testing, absent chronic medication exposure, and negative toxicology reduced support for several alternative causes. However, a negative toxicology screen does not exclude all exposures, and the available history did not comprehensively establish the absence of exertion, trauma, supplements, or other triggers. Negative myositis antibodies also do not exclude seronegative inflammatory myopathy. Preserved strength and improvement without immunosuppression favored an acute, self-limited

process.

The autoimmune evaluation should similarly be interpreted within its scope. The tested panel included antisynthetase, myositis-specific, and myositis-associated antibodies, all reported as negative. This result provided no serologic support for the tested antibody-defined syndromes, but it did not identify the cause of the episode. An antibody panel cannot evaluate inherited defects of muscle energy metabolism. Diagnostic evaluation of rhabdomyolysis therefore depends on the clinical phenotype and exposure history in addition to laboratory testing; specialized investigations are selected according to the suspected acquired or inherited disorder [14].

The family history is an important limitation to assigning a single etiology. An inherited metabolic or genetic susceptibility could have been unmasked by infection. Because the affected relatives, circumstances of their episodes, and results of genetic evaluation were not available, influenza should be described as an associated precipitant rather than a proven sole cause. Clarification of the pedigree and consideration of neuromuscular or genetic evaluation would be appropriate, particularly if episodes recur or CK remains elevated after recovery.

A more detailed family and personal history could help direct that assessment. Relevant questions include whether relatives experienced episodes with fever, fasting, exercise, or medication exposure and whether the patient had previously noticed exertional intolerance, cramps, or dark urine without seeking care. Inherited disorders involving glycogen metabolism, fatty acid oxidation, mitochondrial function, or structural muscle proteins can present with rhabdomyolysis [14]. These possibilities are not diagnoses established in this patient. Their relevance is that a first recognized episode during infection does not necessarily represent the first manifestation of an otherwise detectable predisposition.

Discharge occurred during clear clinical and biochemical improvement, but CK remained markedly elevated at 49,878 U/L. This observation should not be interpreted as evidence that discharge at this value is generally safe. Individual decisions require assessment of renal and electrolyte stability, oral hydration, symptoms, ongoing injury, and reliable follow-up. The absence of postdischarge results prevents confirmation of complete biochemical recovery. For hospital providers, severe myalgias or dark urine during influenza should prompt evaluation for rhabdomyolysis even when initial creatinine and respiratory findings are reassuring.

This report is limited to the available inpatient course and subsequent antibody testing. Serial creatinine measurements strongly support preservation of filtration during hospitalization, but quantitative urine output and markers of subclinical tubular injury were unavailable. The findings therefore establish the absence of documented clinical AKI rather than proving that no renal stress occurred. Similarly, clinical improvement before discharge cannot substitute for confirmation of CK normalization. These distinctions preserve the educational value of the favorable short-term outcome while avoiding conclusions about long-term renal health or the absence of an underlying muscle disorder.

Conclusions

Profound influenza A-associated rhabdomyolysis can occur with preserved renal function. This case supports early recognition, individualized isotonic hydration, and serial renal and electrolyte assessment rather than prognostication from CK alone. A suggestive family history warrants attention to an underlying predisposition, and persistent biochemical abnormalities require follow-up after discharge.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Sean Pack

Acquisition, analysis, or interpretation of data: Sean Pack, FNU Aparna, Mahesh Shrestha

Drafting of the manuscript: Sean Pack

Critical review of the manuscript for important intellectual content: Sean Pack, FNU Aparna, Mahesh Shrestha

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