WHEN TUMOR ACTIVITY DRIVES MUSCLE INJURY

Chemotherapy-Responsive Rhabdomyolysis in Metastatic Pancreatic Neuroendocrine Neoplasm

UConn Health Department of Internal Medicine, Rushali Manhas, DO

KEY MESSAGE | CK increased from 2,706 to 20,815 U/L despite optimal fluid resuscitation. FOLFOX began on day 8; CK peaked on day 9, then decreased 82% by day 16. Temporal association does not establish causality.

BACKGROUND

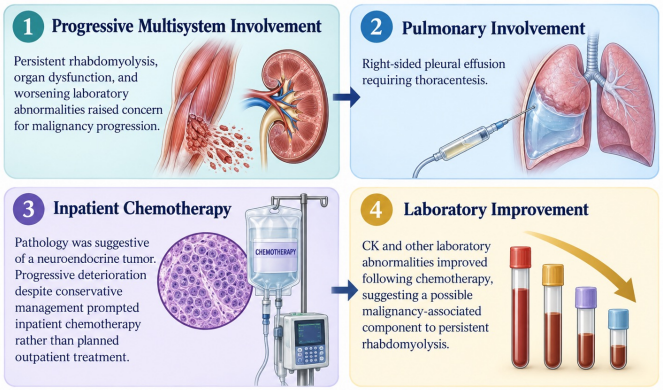
Rhabdomyolysis results from skeletal muscle injury and breakdown, releasing creatine kinase (CK), myoglobin, and intracellular electrolytes into the circulation. Severe cases can lead to myoglobin-associated acute kidney injury, electrolyte disturbances, and life-threatening arrhythmias. Early recognition and supportive management are essential to limit organ dysfunction. In oncology patients, potential contributors include medication toxicity, infection, metabolic disturbances, tumor-associated inflammation, and paraneoplastic processes. Attribution is often challenging because several insults may coexist. Persistent muscle injury despite supportive care warrants continual reassessment of potential triggers, including the possible contribution of active malignancy. Improvement following tumor-directed therapy may support this association, although a temporal relationship alone does not establish causality.

CASE PRESENTATION

38-year-old woman with metastatic grade 2 pancreatic neuroendocrine neoplasm, previously treated with distal pancreatectomy, hepatic resection/ablation, and radiation to mediastinal disease. History included hepatic and uterine metastases. Everolimus was discontinued because of progression/ineffectiveness.

Referred directly from oncology clinic for worsening generalized pain, weakness, nausea, vomiting, chills, diaphoresis, bloating, and marked functional decline. Reported approximately 60-lb weight loss over several months and spontaneous lower-extremity bruising.

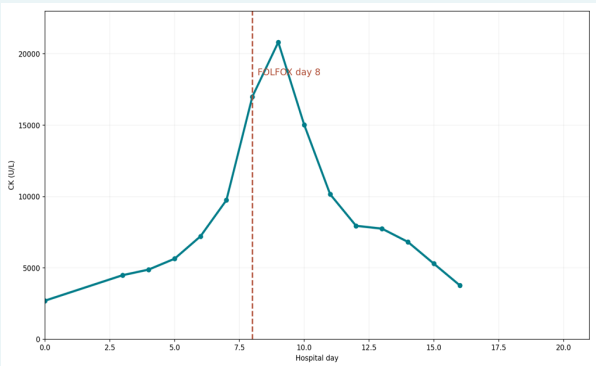
Recent CT demonstrated new mediastinal lymphadenopathy, raising concern for aggressive progression or transformation.



CLINICAL DECISION POINT

Despite IV fluid resuscitation and holding statins, CK continued to rise. Inpatient chemotherapy was selected instead of the originally planned outpatient start because of progressive clinical decline and disease burden. FOLFOX was initiated on hospital day 8.

SERIAL CK TRAJECTORY



The CK peak occurred one day after chemotherapy began, and notably decreased after initiation of FOLFOX on day 8

LABORATORY HIGHLIGHTS

	Admission	Peak / nadir	Later value
CK (U/L)	2,706	20,815 (D9)	3,784 (D16)
Creatinine (mg/dL)	0.57	2.07 (D11)	0.53 (D21)
Platelets (K/µL)	80	9 (D17)	96 (D21)
Hemoglobin (g/dL)	7.9	6.4 (D12)	7.5 (D21)

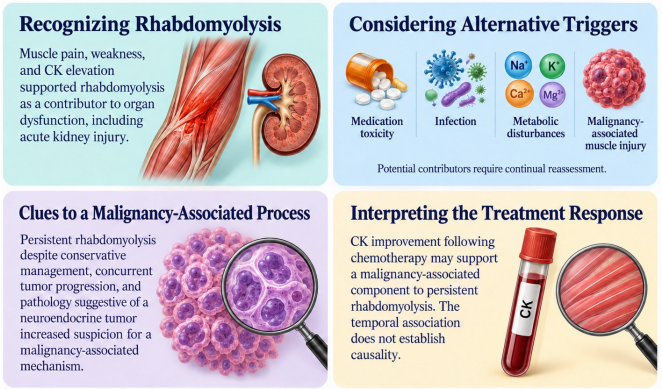
CK fell 82% between days 9 and 16. Creatinine peaked after the CK peak and returned near baseline by day 21.

REFERENCES

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Differential Diagnosis and Clinical Reasoning



CONCLUSIONS AND DISCUSSION

- Consider rhabdomyolysis in oncology patients presenting with muscle pain, weakness, and unexplained organ dysfunction, particularly acute kidney injury.
- Serial CK and renal indices help quantify severity and clinical evolution.
- Severe rhabdomyolysis may coexist with aggressive cancer progression and multisystem dysfunction.
- The decline in CK following chemotherapy may suggest that the underlying malignancy contributed to persistent rhabdomyolysis.
- Systemic therapy was initiated despite critical illness because rhabdomyolysis persisted despite conservative management.