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Case Report **Medicine**

Sacubitril/Valsartan Induced Rhabdomyolysis in a High-Risk Patient with Multi-Morbidity: A Case Report and Comprehensive Review of the Literature

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Abstract

Background: Sacubitril/Valsartan, an angiotensin receptor-neprilysin inhibitor (ARNI), represents a paradigm shift in the management of heart failure with reduced ejection fraction (HFrEF). Its robust efficacy in reducing mortality and hospitalization is well-established. However, as its use becomes ubiquitous, the recognition of rare and serious adverse events is paramount for optimizing patient safety. ***Case Presentation:*** A 57-year-old male with a complex medical history including type 2 diabetes, dyslipidemia, hypertension, chronic kidney disease (CKD stage 3a), and NYHA Class III HFrEF was initiated on Sacubitril/Valsartan 24/26 mg twice daily. Two weeks post-initiation, he presented with severe bilateral proximal myalgia, profound weakness, and classic tea-colored urine. Physical examination revealed significant muscle tenderness. Laboratory investigations were diagnostic for severe rhabdomyolysis, with a peak creatine kinase (CK) level of 18,540 U/L and concomitant acute kidney injury (AKI), evidenced by a rise in serum creatinine from a baseline of 1.4 mg/dL to 2.1 mg/dL. A meticulous workup excluded other common etiologies of rhabdomyolysis, including recent trauma, strenuous exertion, hypothyroidism, and illicit substance use. The patient had been on a stable, long-term dose of atorvastatin without prior incident. The Naranjo Adverse Drug Reaction Probability Scale score was 7, indicating a "probable" adverse drug reaction. Management involved immediate and permanent discontinuation of Sacubitril/Valsartan, aggressive intravenous fluid resuscitation, and close monitoring. A positive dechallenge was observed, with rapid symptomatic improvement and normalization of CK and renal function over the following week. ***Conclusion:*** This case provides compelling evidence for Sacubitril/Valsartan as a rare but potent precipitant of rhabdomyolysis, particularly in patients with predisposing factors such as CKD and concomitant statin use. It underscores the critical importance of clinician vigilance, pre-emptive patient education, and the prompt institution of management upon symptom recognition to prevent life-threatening complications like acute renal failure.

Keywords: Sacubitril/Valsartan, ARNI, Heart Failure, Diabetes, Dyslipidemia, Hypertension, Chronic Kidney Disease.

INTRODUCTION

The management of chronic heart failure with reduced ejection fraction (HFrEF) has been fundamentally transformed over the past decades, evolving from a focus on symptom palliation to a comprehensive, guideline-directed medical therapy (GDMT) approach aimed at prolonging life and reducing hospitalizations [1]. The advent of Sacubitril/Valsartan (Entresto), the first-in-class angiotensin receptor-neprilysin inhibitor (ARNI), marked a watershed moment in this therapeutic landscape [2]. This compound synergistically combines the established benefits of an angiotensin II receptor blocker (Valsartan) with the novel action of a neprilysin inhibitor (Sacubitril). Neprilysin is a neutral endopeptidase responsible for the degradation of endogenous vasoactive peptides, including natriuretic peptides,

bradykinin, and adrenomedullin. By inhibiting neprilysin, Sacubitril potentiates these protective pathways, promoting natriuresis, diuresis, vasodilation, and counteracting the maladaptive neurohormonal activation that characterizes the heart failure syndrome [3].

The paradigm-shifting efficacy of Sacubitril/Valsartan was unequivocally established by the Prospective comparison of ARNI with ACEI to Determine Impact on Global Mortality and morbidity in Heart Failure (PARADIGM-HF) trial [4]. This landmark study demonstrated a staggering 20% reduction in the composite endpoint of cardiovascular death or heart failure hospitalization for Sacubitril/Valsartan compared to the gold-standard ACE inhibitor, enalapril. This compelling evidence led to its rapid incorporation into international clinical guidelines, where it now holds a Class I recommendation for eligible patients with symptomatic HFrEF [5,6].

With the widespread and growing adoption of this life-prolonging therapy, the comprehensive understanding of its safety profile has become increasingly crucial. The most frequently reported adverse effects, as identified in clinical trials and post-marketing surveillance, include symptomatic hypotension, hyperkalemia, and worsening renal function [4,7]. These are largely predictable, given the drug's potent vasodilatory and neurohormonal modulating effects, and are often manageable with dose adjustment and monitoring.

However, the phenomenon of drug-induced rhabdomyolysis occupies a distinct and concerning niche in pharmacovigilance. Rhabdomyolysis is a potentially fatal clinical syndrome characterized by the rapid dissolution of skeletal muscle and the subsequent release of intracellular contents, including myoglobin, creatine kinase (CK), and electrolytes, into the systemic circulation [8]. The classic triad of symptoms includes myalgia, weakness, and dark urine, though the presentation can be variable. The most feared complication is acute kidney injury (AKI), which occurs in up to 50% of cases, primarily due to myoglobin-induced tubular toxicity and intrarenal vasoconstriction [9].

Drug-induced myotoxicity exists on a spectrum, ranging from benign myalgia to myositis (muscle inflammation with elevated CK) and, at its most severe, fulminant rhabdomyolysis. The 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors, or statins, are the most notorious and well-characterized class of drugs associated with this sequela, with an incidence of clinically significant rhabdomyolysis estimated at approximately 1.5 per 100,000 patient-years [10]. The risk is heightened by factors such as advanced age, renal or hepatic impairment, hypothyroidism, and drug interactions, particularly those involving cytochrome P450 (CYP) inhibitors.

In the context of Sacubitril/Valsartan, the prescribing information and initial clinical trial data reported myalgia as an uncommon occurrence, with no significant difference compared to the enalapril group [4,11]. However, as real-world experience with the drug expands, a trickle of case reports has begun to emerge, describing a temporal association between Sacubitril/Valsartan initiation and the development of severe rhabdomyolysis [12,13]. This rare adverse effect is not yet widely recognized by the broader medical community, creating a potential for delayed diagnosis and management.

Therefore, the primary objective of this detailed case report is to present a robust and meticulously documented instance of Sacubitril/Valsartan-induced rhabdomyolysis in a patient with multiple classic risk factors. We aim to comprehensively describe the clinical presentation, diagnostic journey, and successful management; while engaging in a deep discussion of the potential pathophysiological mechanisms and the critical lessons this case imparts for clinical practice. By doing so, we seek to elevate clinician awareness of this rare but serious adverse event, thereby enhancing the safe and effective application of this otherwise transformative heart failure therapy.

CASE PRESENTATION

Patient Information and Historical Context:

The patient was a 57-year-old Caucasian man who presented to the emergency department via personal transport with a chief complaint of "severe leg pain, weakness, and Coca-Cola colored urine" that had developed over the preceding 72 hours. He described the pain as a deep, aching, and constant sensation localized predominantly to his proximal thighs and buttocks, bilaterally symmetric, and rated as 8/10 in severity. The pain was exacerbated by any movement or palpation and was associated with profound lower extremity weakness, making it difficult for him to rise from a seated position without assistance. The onset of dark brown, tea-colored urine was the most alarming symptom that prompted his visit.

While review of his past medical history (table 1) revealed a complex tapestry of chronic conditions reflective of advanced cardiometabolic syndrome. Table 2 listing his home medications

Table 1: Past medical history

Past Medical History	
Heart Failure	Ischemic cardiomyopathy, diagnosed 4 years prior, with a most recent left ventricular ejection fraction (LVEF) of 30% by echocardiogram. His functional status was consistent with New York Heart Association (NYHA) Class III.
Chronic Kidney Disease (CKD)	Stage 3a, with a documented baseline serum creatinine of 1.4 mg/dL and an estimated glomerular filtration rate (eGFR) of 55 mL/min/1.73m², attributed to a combination of hypertensive and diabetic nephropathy.
Type 2 Diabetes Mellitus	Diagnosed 10 years prior, managed with Metformin 500 mg twice daily. His glycemic control was suboptimal, with a recent HbA1c of 8.2%.
Dyslipidemia	Treated with Atorvastatin 20 mg daily for over three years.
Hypertension	A long-standing history, well-controlled on his current regimen.

Table 2: List of home medication

Aspirin 81 mg daily	Furosemide 40 mg daily
Atorvastatin 20 mg daily	**Bisoprolol 5 mg daily**
Metformin 500 mg twice daily	

Crucially, two weeks before this presentation, his heart failure regimen had been optimized in the cardiology clinic. His previous angiotensin-converting enzyme inhibitor (Lisinopril 10 mg daily) was discontinued, and he was initiated on Sacubitril/Valsartan (Entresto) at a dose of 24/26 mg twice daily. He reported tolerating the first few doses without immediate incident, specifically denying any lightheadedness or dizziness.

The patient clearly denied any recent history of trauma, crush injury, seizure, syncope, or loss of consciousness. There was no history of unaccustomed, strenuous physical exertion. He was a non-smoker and reported no alcohol or illicit drug use. There was no personal or family history of inherited metabolic myopathies or neuromuscular disorders. His review of systems was otherwise negative for fever, rash, arthralgia, infectious symptoms, or sensory deficits.

Clinical Findings and Diagnostic Assessment:

Upon presentation, the patient was alert, oriented, but visibly uncomfortable. His vital signs were stable: blood pressure 128/78 mmHg, heart rate 68 beats per minute (regular), respiratory rate 16 breaths per minute, temperature 36.8°C (98.2°F), and oxygen saturation of 98% on room air. Cardiopulmonary auscultation revealed a regular rhythm with a soft S3 gallop but no murmurs, rubs, or evidence of pulmonary edema. His abdominal examination was benign.

The musculoskeletal and neurological examinations were critical. There was marked tenderness to palpation over the quadriceps, hamstrings, and gluteal muscles bilaterally. Manual muscle testing revealed significant proximal muscle weakness, graded 4/5 for hip flexion and abduction, with relative preservation of distal strength (5/5). There was no muscle swelling, erythema, or skin changes. The remainder of the neurological exam, including cranial nerves, sensation, and deep tendon reflexes, was intact.

Given the classic symptomatology, the initial diagnostic focus was on confirming rhabdomyolysis and assessing for complications. The results of the initial and subsequent laboratory investigations are summarized in Table 3.

Table 3: Serial Laboratory Parameters during Hospitalization

Parameter (Unit)	**Reference Range**	**Day 1 (Admission)**	**Day 2**	**Day 3**	**Day 7 (Discharge)**
CK (U/L)	39-308	9,850	15,200	18,540	450
Creatinine (mg/dL)	0.7-1.3	2.1	1.9	1.8	1.5
eGFR (mL/min/1.73m²)	>90	32	36	38	48
Myoglobin (ng/mL)	<110	>3000	N/A	N/A	N/A
Potassium (mmol/L)	3.5-5.1	5.0	4.9	4.8	4.6
Phosphate (mg/dL)	2.5-4.5	5.1	4.6	4.2	3.8
Calcium (mg/dL)	8.6-10.2	8.2	8.4	8.6	9.0
ALT (U/L)	7-52	68	60	55	40
AST(U/L)	13-39	95	78	70	35
Urinalysis	Blood 3+, No RBCs				Clear

The laboratory profile was diagnostic for severe rhabdomyolysis. The CK level was markedly elevated, peaking at over 60 times the upper limit of normal. The presence of significant myoglobinuria was confirmed by the dipstick positive for blood in the absence of red blood cells on microscopic examination, and the serum

myoglobin level was profoundly elevated. The acute kidney injury (AKI) was evident from the rise in serum creatinine, consistent with Stage 1 AKI according to KDIGO (kidney disease: Improving Global Outcomes) criteria [14]. Mild hyperphosphatemia and hypocalcemia were also present, which are common electrolyte disturbances in rhabdomyolysis due to the release of intracellular contents and sequestration of calcium in damaged muscle, respectively. The transaminase elevation (AST>ALT) is a frequent finding in muscle injury and does not necessarily indicate primary hepatic pathology.

A comprehensive workup was undertaken to systematically exclude other potential causes of rhabdomyolysis in table 4.

Table 4: Rule out of potential causes

Potential causes	Rule out
Statin Myopathy	The patient had been on a stable, low-to-moderate dose of atorvastatin for over three years with excellent tolerance and a previously normal CK level. While statin rechallenge was not performed, the temporal relationship overwhelmingly pointed to the newly introduced agent.
Endocrine	Thyroid-stimulating hormone (TSH) was within normal limits, ruling out hypothyroidism.
Toxicological	A comprehensive urine toxicology screen was negative for cocaine, amphetamines, and other illicit substances
Infectious/Inflammatory	C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were normal. Blood and urine cultures showed no growth.
Metabolic	There was no clinical history suggestive of an underlying metabolic myopathy.
Ischemic/Traumatic	No history was elicited, and physical exam showed no signs of compartment syndrome.

The Naranjo Adverse Drug Reaction Probability Scale was applied, yielding a score of 7 [15]. Key points contributing to this "probable" score included: the definitive temporal sequence following drug exposure, the confirmed rhabdomyolysis as a known (though rare) reaction, improvement upon dechallenge, and the exclusion of alternative causes.

Therapeutic Intervention and Clinical Outcome:

The management strategy was twofold: treat the acute rhabdomyolysis and its complication (AKI), and remove the suspected offending agent.

1. Immediate Cessation of Sacubitril/Valsartan: The drug was permanently discontinued on admission.
2. Temporary Hold of Atorvastatin: As a precautionary measure, atorvastatin was temporarily withheld to eliminate any potential confounding effect, with a plan to reintroduce it after resolution.
3. Aggressive Intravenous Fluid Resuscitation: The cornerstone of management was vigorous hydration with 0.9% normal saline. An initial bolus of 1 liter was administered over the first hour, followed by a continuous infusion titrated to maintain a urine output of 200-300 mL per hour. This strategy aims to maintain renal perfusion and promote the clearance of nephrotoxic myoglobin.
4. Close Monitoring: The patient was admitted to a step-down unit for continuous cardiac monitoring and strict input/output monitoring. Serum electrolytes, creatinine, and CK were measured every 12-24 hours.

The clinical response was prompt and favorable. The patient reported a significant reduction in muscle pain within 48 hours of admission. His muscle strength improved progressively. Biochemically, his CK level demonstrated a consistent downward trend, falling to 450 U/L by day 7. Concurrently, his renal function recovered, with serum creatinine decreasing to 1.5 mg/dL, approaching his baseline. His electrolyte disturbances normalized with hydration alone.

He was discharged on day 7 in a stable condition. His discharge medications included his original regimen, with atorvastatin restarted and his heart failure therapy switched back to Lisinopril 10 mg daily. At a follow-up visit two weeks later, he remained asymptomatic, with a CK level of 180 U/L and a serum creatinine of 1.4 mg/dL, confirming a complete biochemical recovery.

DISCUSSION

This case provides a compelling and meticulously documented account of a rare, serious adverse effect of a widely used cardioprotective drug. The diagnosis of Sacubitril/Valsartan-induced rhabdomyolysis is supported by a strong temporal relationship, a positive dechallenge, the exclusion of competing etiologies, and a "probable" score on a validated adverse drug reaction algorithm. This discussion will delve into the mechanistic hypotheses, contextualize this case within the emerging literature, and explore its profound implications for clinical practice.

Mechanistic Pathophysiological Hypotheses:

The precise mechanism by which Sacubitril/Valsartan induces skeletal muscle injury remains enigmatic, as it is not a classically recognized myotoxin. However, several plausible, non-mutually exclusive hypotheses can be proposed.

Idiosyncratic Hypersensitivity Reaction:

The most compelling explanation is an idiosyncratic reaction. This implies an unpredictable, non-dose-dependent, and likely immune-mediated response unique to the individual [16]. The rapid onset (within two weeks) in our patient is highly suggestive of a hypersensitivity phenomenon, akin to rare cases of statin-induced autoimmune necrotizing myopathy associated with anti-HMG-CoA reductase antibodies [17]. It is conceivable that Sacubitril, Valsartan, or one of their metabolites could act as a hapten, triggering an immune response directed against muscle fibers. Future research could investigate whether similar autoantibodies are present in patients who develop this reaction.

Dysregulation of Vasoactive Peptides and Perfusion:

The fundamental pharmacodynamic action of Sacubitril/Valsartan is the potentiation of vasoactive peptides like bradykinin. While this effect is beneficial for systemic and coronary vasodilation, it could theoretically lead to localized dysregulation of skeletal muscle perfusion. A sudden shift in the microvascular tone of muscle beds could potentially result in a relative ischemia-reperfusion injury, initiating the cascade of muscle cell necrosis [18]. Patients with pre-existing microvascular disease, such as our diabetic patient, might be particularly susceptible to such shifts in perfusion pressure.

Electrolyte Shifts and Cellular Instability:

Both components of the drug can influence electrolyte balance. Valsartan, via its effect on the renin-angiotensin-aldosterone system (RAAS), can promote potassium retention. Sacubitril, by enhancing natriuretic peptides, promotes sodium excretion. While these effects are generally systemic and manageable, it is hypothetically possible that they create an unfavorable electrochemical gradient across muscle cell membranes in susceptible individuals, predisposing them to breakdown, especially under physiological stress [19].

Pharmacokinetic Interactions and Synergistic Myotoxicity:

Our patient was on concurrent atorvastatin. While he had a long history of tolerance, the introduction of a new agent can alter the risk landscape. Sacubitril/Valsartan is not a potent inhibitor of major CYP enzymes like 3A4, which metabolizes atorvastatin [11]. However, drug interactions can occur through other pathways, such as transport proteins (e.g., OATP1B1). A potential, albeit speculative, interaction could lead to increased intracellular statin concentration in myocytes, pushing a previously stable patient over the threshold into overt myotoxicity [20]. This represents a synergistic "two-hit" model rather than a direct causation by either drug alone.

Context within the Existing Medical Literature:

Our case report adds a significant data point to a small but growing body of literature. A systematic review of the FDA Adverse Event Reporting System (FAERS) database and published case reports reveals a consistent pattern [12,13,21]. The onset of symptoms typically occurs within the first few weeks of initiation, as seen in our patient. Many reported cases, like ours, involve patients with one or more classic risk factors for drug-induced myopathy, including advanced age, diabetes, CKD, and concomitant statin use [22].

The presentation is often dramatic, with CK levels frequently exceeding 10,000 U/L, indicating extensive muscle damage. The consistent observation of a positive dechallenge—rapid clinical and biochemical improvement upon drug withdrawal—across nearly all reported cases strengthens the causal inference. Our case aligns perfectly with this profile, reinforcing the validity of this rare adverse drug reaction.

It is crucial to distinguish this from the myalgia reported in the PARADIGM-HF trial [4]. The trial reported myalgia as an uncommon and non-serious adverse event, with no significant difference between the Sacubitril/Valsartan and enalapril groups. The cases now emerging in the post-marketing phase represent a much more severe, distinct clinical entity—true rhabdomyolysis with a risk of AKI—that was likely too rare to be captured in a clinical trial, even one as large as PARADIGM-HF. This highlights the critical importance of post-marketing surveillance and case reports in refining the safety profile of new therapeutics.

Clinical Implications and Recommendations for Practice:

The primary lesson from this case is the necessity of heightened clinical vigilance. For cardiologists, internists, and primary care physicians prescribing Sacubitril/Valsartan, it is no longer sufficient to monitor only for hypotension, hyperkalemia, and renal dysfunction. The following recommendations are proposed:

1. Pre-Emptive Patient Education: Before initiating therapy, patients should be counseled about potential adverse effects. This counseling must now include a specific warning to report any unexplained muscle pain, tenderness, weakness, or darkening of the urine immediately. This empowers patients to become active participants in their own safety.
2. Proactive Monitoring in High-Risk Patients: For patients with multiple risk factors (e.g., CKD, diabetes, concomitant statin use), consideration could be given to obtaining a baseline CK level prior to initiation. This provides a reference point should symptoms develop later.
3. A Low Threshold for Investigation: The development of significant myalgia,

particularly if proximal and accompanied by weakness, should not be dismissed. It warrants immediate evaluation with a serum CK test and renal function panel.

4. Prompt and Decisive Management: If rhabdomyolysis is diagnosed, the management is unequivocal: immediate and permanent discontinuation of Sacubitril/Valsartan. The decision to also temporarily hold a concomitant statin is prudent. Aggressive fluid resuscitation remains the cornerstone of medical management to prevent AKI.
5. Reporting to Pharmacovigilance Authorities: It is imperative that such cases are formally reported to national drug safety monitoring agencies (e.g., the FDA's MedWatch program). This collective reporting is essential for quantifying the true incidence and identifying potential risk factors.

CONCLUSION

Sacubitril/Valsartan is an undeniably life-saving therapy that has redefined the standard of care for HFrEF. However, our case unequivocally demonstrates that it can, in rare instances, precipitate severe rhabdomyolysis. The presentation in our patient was classic, the biochemical confirmation was unequivocal, and the causal link was strongly supported by the temporal sequence and positive dechallenge. This report serves as a critical alert to the medical community, underscoring that the benefits of this drug, while profound, do not come without a potential, albeit rare, risk. A proactive approach involving patient education, clinical vigilance, and prompt intervention is paramount to ensuring that the application of this transformative therapy is both effective and safe. Further research is needed to elucidate the underlying pathophysiological mechanisms and to identify genetic or clinical markers that may predispose certain individuals to this serious adverse reaction.

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