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Case Report

Rhabdomyolysis following minimally invasive transforaminal lumbar interbody fusion: Case report

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Abstract

Background: Rhabdomyolysis results from the release of large quantities of muscle cell contents into plasma resulting in a classic triad of symptoms – muscle pain, weakness, and brown urine. Only a handful of rhabdomyolysis cases occurring after spinal surgery have been reported.

Case Description: A 36-year-old male underwent an uneventful right-sided, minimally invasive transforaminal lumbar interbody fusion (miTLIF) for intractable lower back pain and right lower extremity radiculopathy attributed to L4-S1 degenerative spondylosis (DS). Postoperatively, the patient complained of intractable lower extremity pain resistant to medical management. He was subsequently diagnosed with rhabdomyolysis, and aggressive intravenous fluid resuscitation resulted in complete recovery.

Conclusions: Rhabdomyolysis should be diagnosed and treated promptly with aggressive intravenous fluid resuscitation to avoid acute kidney injury following miTLIF surgery.

Key Words: Acute kidney injury, AKI, minimally invasive spine surgery, MISS, rhabdomyolysis, transforaminal lumbar interbody fusion, transforaminal lumbar interbody fusion

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INTRODUCTION

Rhabdomyolysis (RM) results from the release of large quantities of muscle cell contents into plasma. Most frequent causes of RM include crush injury, strenuous exercise, toxins, infections, muscle dystrophies, disturbances in potassium or phosphate homeostasis, epilepsy, Lou Gehrig's disease, acute psychotic disorders, Reye syndrome, bowel ischemia, graft-versus-host disease, and eosinophilic fasciitis.[4] Few cases of RM in patients undergoing spinal surgery have been described;[2,3,5-7] one report noted RM after minimally invasive surgery.[2] Here, we present a patient who developed RM after undergoing an uneventful minimally invasive (MI) transforaminal lumbar interbody fusion (miTLIF).

CASE REPORT

A 36-year-old male with two prior lumbar laminectomies (2012) presented in 2015 with increased

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intractable lower back pain and right lower extremity radiculopathy of 3 months duration. Magnetic resonance imaging (MRI) of the lumbosacral spine revealed lumbar spondylosis with degenerative disc disease at L4-S1 with a right paracentral disc herniation and neuroforaminal stenosis at L4-5 and biforaminal stenosis at L5-S1. Social history was significant for manual labor and smoking one pack per day for approximately 14 years. On physical examination, the patient had a body mass index (BMI) of 23 kg/m^2, as well as nonfocal neurological examination except for decreased sensation on the lateral aspect of the right thigh and calf.

In 2015, he underwent an uneventful right-sided miTLIF using intraoperative computed tomography (CT) guidance. The patient was positioned on a Jackson table. Intraoperatively, he remained hemodynamically stable (medications: midazolam 2 mg, lidocaine 80 mg, propofol 280 mg, fentanyl 400 mcg, rocuronium 180 mg, cefazolin 1 g, acetaminophen 1 g, phenylephrine 2 mg, hydromorphone 1 mg, glycopyrrolate 0.5 mg, neostigmine 3.5 mg, ondansetron 4 mg, and sevophlorane). The operative time to extubation was 6 hours and 57 minutes, and the surgical time from incision to skin closure was 4 hours and 53 minutes. The patient received a total of 2.5 L of normal saline, his urine output was 410 mL, and the estimated blood loss was 150 mL.

Postoperatively, he complained of severe lower back and leg pain, and was started on intravenous (IV) hydromorphone, valium, and oral cyclobenzaprine. On postoperative day (POD) one, severe pain continued, for which he was given celebrex, oxycodone, and fentanyl, via a patient controlled analgesia pump. On the evening of POD two, the patient was overnarcotized requiring Narcan, but his blood pressure remained within range. At this point, his serum creatine kinase (CK) was 11,492 unit/L. He was promptly started on IV fluids, and switched over to an oral pain regimen. His CK continued to decrease, and was 1,699 unit/L on the day of discharge, i.e. POD seven. Patient remained neurologically stable throughout the hospital stay with full strength in both lower extremities on the day of discharge.

On his first postoperative office visit, his CK had reduced to 400 unit/L, and he reported near complete resolution of his lower back pain. At the 3-month postoperative follow-up visit, the patient was off pain medication, pain free, and working.

DISCUSSION

RM is the result of skeletal muscle fiber breakdown with release of fiber contents into the bloodstream and urine. Usually, it presents as muscle fatigue, pain, cramps, and weakness. Reddish-brown urine indicating myoglobinuria is highly suggestive of RM.[4] Diagnostic laboratory testing is outlined in Table 1. An arbitrary value of 500 to 1,000 IU/L or 5 to 10 times of the upper limit from normal is frequently used to define RM.[8] Serial CK measurements can be used to track treatment success or failure.

Table 1: Laboratory Testing for Initial Evaluation of Rhabdomyolysis. This table is reproduced with the permission of the authors and Chest[8]

Test	Abnormal Value for RM	Comments
Creatine Kinase	>500 IU/L	Diagnostic for rhabdomyolysis; increased risk of kidney injury if >5,000 IU/L
Potassium	>6.0 mmol/L	Marker of severity of muscle injury and renal dysfunction
	<2.0 mmol/L	Potential cause of rhabdomyolysis
Phosphorous	>6.0 mg/dL	Marker of severity of muscle injury and renal dysfunction
	<2.0 mg/dL	Potential cause of rhabdomyolysis
Calcium	Decreased (< 8.0 mg/dL)	Deposition in damaged muscle
Creatinine	Increased	Marker of decreased renal function
BUN: creatinine	<10:1, often<6:1	Increased conversion of muscle creatine to creatinine
Anion gap	Increased	Increased organic acids due to muscle injury or renal dysfunction
Blood alcohol level	Elevated	Potential cause of rhabdomyolysis
Urine blood dipstick	Positive	Detects myoglobinuria in absence of RBCs in urine
Urine drug screen	Positive	Potential drug-related cause of rhabdomyolysis

Pathophysiology of RM involves muscle fiber lysis caused by damage to the sarcolemma or by metabolic disturbances related to a biochemical or genetic abnormality. The crucial factor in the mechanism of injury in RM is elevated intracellular free calcium (Ca) levels. Disruption of Ca homeostasis leads to activation of

Table 2: Causes of Rhabdomyolysis. This table is reproduced with the permission of the authors and Chest[8]

Hypoxic	Physical	Chemical	Biologic
External	External	External	External
Carbon monoxide exposure	Crush injury	Alcohol	Bacterial, viral, & parasitic myositis
Cyanide exposure	Trauma	Prescription medications	Organic toxins
	Burns	Over-the-counter medications	Snake venom
	Electrocution	Illicit drugs	Spider bites
	Hypothermia		Insect stings (ants, bees, wasps)
	Hyperthermia (heat stroke)		
Internal	Internal	Internal	Internal
Compartment syndrome	Prolonged and/or extreme	Hypokalemia	Dermatomyositis, polymyositis
Vascular compression	exertion	Hypophosphatemia	Endocrinopathies
Immobilization	Seizures	Hypocalcemia	Adrenal insufficiency
Bariatric surgery	Status asthmaticus	Hypo-/hypernatremia	Hypothyroidism
Prolonged surgery	Severe agitation		Hyperaldosteronism
Sickle cell trait	Neuroleptic malignant syndrome		Diabetic ketoacidosis
Vascular thrombosis	Malignant hyperthermia		Hyperosmolar state effect
Vasculitis			

BUN: Blood urea nitrogen, RBC: Red blood cell, RM: Rhabdomyolysis[8]

proteases and phospholipases, which break down proteins that make up the contractile apparatus, cell membrane, and cytoskeleton.[4]

Common causes of RM were outlined by Zimmerman *et al.*,[8] and can be divided into four categories – hypoxic, physical, chemical, and biologic – with direct muscle injury being the most common cause of RM[2] [Table 2]. To date, only a handful of cases developed RM due to spinal surgery,[2,3,5-7] and only one report concerned with a MI direct lateral interbody fusion (miDLIF);[2] however, none involved miTLIF.

There have been multiple case rerpots of RM after prolonged spinal procedures, but only one involved MI surgery. The etiology of RM was variously attributed to: Ziser *et al*[9] prolonged surgery 7 to 10 hours; Foster[3] obese patient with 6 hour revision of lumbar fusion using the Jackson table; Nayak *et al*[6] a 22-year-old with BMI of 35.6 kg/m^2, RM after 9 hours to resect an L3 giant cell tumor; Dakwasr *et al*[2] five patients undergoing miDLIF, BMI from 25 to 40 kg/m^2, operative time of 5.25 to 10 hours, and various medical comorbidities. In our case, the only risk factor for RM was the relatively prolonged operative time.

The treatment of RM aims at prevention of acute kidney injury, and consists of early recognition and aggressive volume resuscitation to restore adequate renal perfusion.[1]

CONCLUSION

RM is a rare complication typically of prolonged spinal surgery. It should be rapidly diagnosed and treated with aggressive fluid resuscitation to avoid renal failure.

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Conflicts of interest
There are no conflicts of interest.

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