

# A Case of Lower Extremity Compartment Syndrome with an Unusual Presentation

**AUTHORS:**

Magarinos J; Hurwich A; Beard JH

**CORRESPONDING AUTHOR:**

Jessica Magarinos, MD  
 Temple University Health Systems  
 3401 N. Broad Street  
 Philadelphia, PA 19140  
 Email: jessicamagarinos@gmail.com

**AUTHOR AFFILIATION:**

Department of General Surgery  
 Temple University Hospital  
 Philadelphia, PA 19140

<b>Background</b>	A male patient presented with acute quadriplegia caused by compartment syndrome and its resulting hyperkalemia.
<b>Summary</b>	<p>A 48-year-old man presented with quadriplegia following a seemingly minor knee injury three days prior. He reported spending most of the intervening time recuperating on his couch. On the day of presentation, he awoke with quadriplegia.</p> <p>Investigation revealed acute renal failure, rhabdomyolysis, and hyperkalemia, which explained his paralysis. A thorough physical examination identified the culprit: acute compartment syndrome in his right calf, likely due to prolonged immobility after the knee injury.</p> <p>He received emergent treatment for hyperkalemia and underwent a right calf four-compartment fasciotomy. Subsequently, his kidney function recovered fully, and he regained good ambulatory function in the affected leg.</p>
<b>Conclusion</b>	Acute compartment syndrome is a common surgical emergency treated with decompressive fasciotomy. This case report reinforces the critical role of a thorough physical examination in timely diagnosis, even when faced with atypical presentations.
<b>Key Words</b>	acute compartment syndrome; hyperkalemia; fasciotomy; rhabdomyolysis

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## Case Description

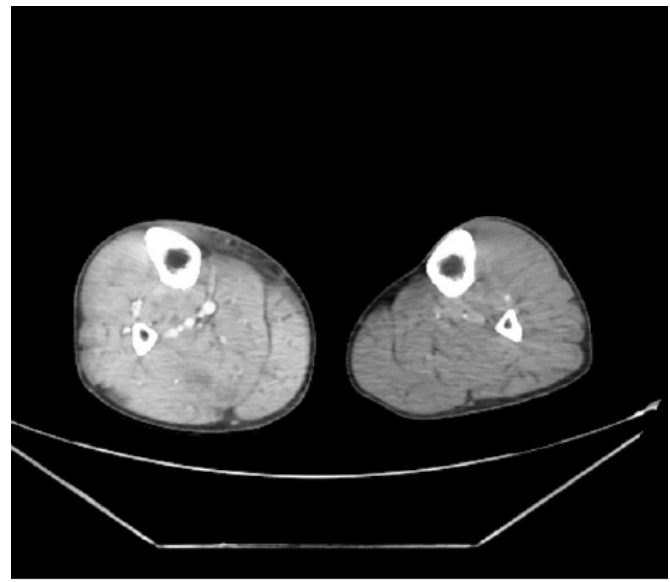
Acute compartment syndrome (ACS) is a surgical emergency characterized by increased pressure within a non-expandable space, compromising blood flow and leading to tissue ischemia.<sup>1</sup> Typically affecting the lower extremities following trauma,<sup>1</sup> ACS can also arise from restrictive dressings, circumferential burns with eschar formation, or prolonged pressure.<sup>2</sup> Diagnosis of ACS hinges on clinical evaluation through history and physical examination; imaging is not routinely required.<sup>1,2</sup> This case report presents an uncommon instance of ACS developing in a patient following a minor knee injury.

A 48-year-old man with a history of hypertension presented to the emergency department with quadriplegia. Three days prior, he fell on his right knee while playing football and experienced significant pain, managed conservatively with rest and unspecified pain medications. He became progressively immobile and, on the third day, woke up with paralysis of his arms and legs, prompting his visit.

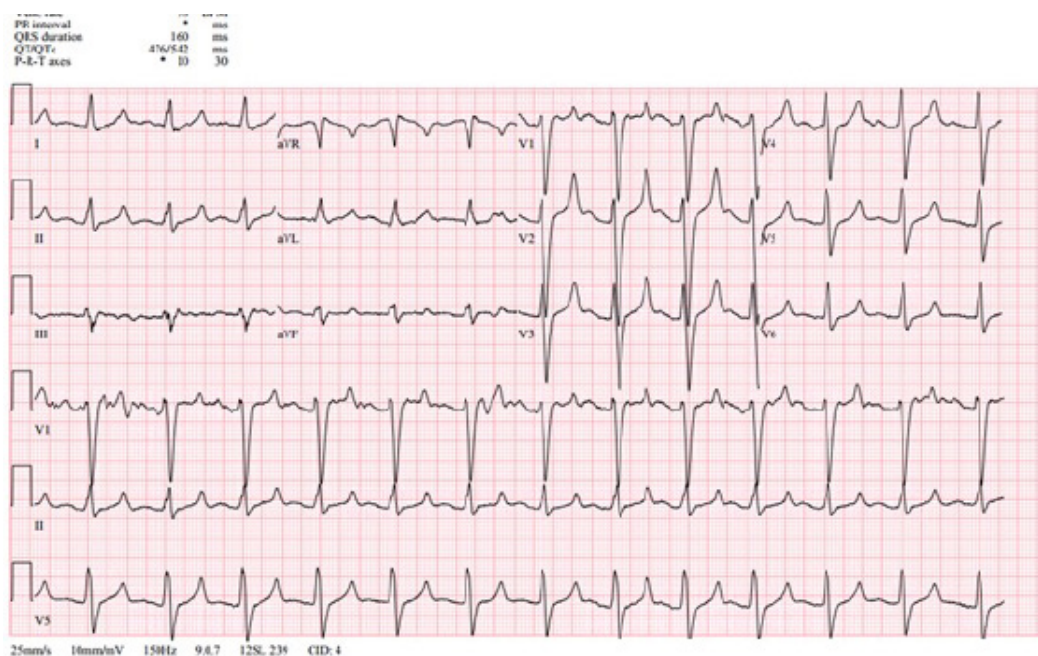
Examination revealed normal vital signs except for hypertension (170/100 mmHg). He had cervical spine tenderness on palpation without deformity. All four limbs showed complete paralysis (0/5 strength) with preserved sensation. His right calf was tense, tender, and painful with passive ankle dorsiflexion. Dorsalis pedis and posterior tibial pulses in the right foot were diminished.

Imaging studies were performed to investigate the cause of his paralysis and right leg symptoms. A cervical spine CT scan was obtained to assess for potential fractures due to tenderness and paralysis. Additionally, a CT angiogram of his bilateral lower extremities was performed to evaluate for occult fractures or arterial injury contributing to suspected right calf compartment syndrome (Figure 1). The initial electrocardiogram (ECG) is shown in Figure 2.

**Figure 1.** Bilateral Lower Extremity CT Angiography. Published with Permission



**Figure 2.** Initial Electrocardiogram



The ECG findings suggested severe hyperkalemia (widened QRS complex and peaked T-waves), which was confirmed by a laboratory potassium level of 8.1 mmol/L. Additionally, laboratory workup revealed rhabdomyolysis (creatinine kinase 207,584 U/L) and acute kidney injury (creatinine 19.4 mg/dL).

Prompt intervention focused on stabilizing the cardiac membrane with intravenous calcium and shifting potassium intracellularly using insulin and glucose. Imaging (CT scans of the cervical spine and lower extremities) ruled out fractures or vascular injuries but identified hypoattenuating areas within the right calf muscle, suggestive of rhabdomyolysis involvement. Due to airway compromise and critical electrolyte imbalance, the patient underwent emergent intubation and dialysis in the surgical ICU.

Following rapid normalization of his potassium levels, he underwent bedside four-compartment fasciotomies of the right calf, revealing bulging but viable muscle tissue with a palpable dorsalis pedis pulse post-procedure. The patient was extubated the following day and demonstrated gradual improvement in renal function. Physical examination confirmed knee joint stability. Following a period of negative pressure wound therapy, split-thickness skin grafting was performed on the fasciotomy sites.

He was discharged home on hospital day 22 with normal strength and function in all extremities except for a right foot drop. This resolved completely after outpatient physical therapy.

## Discussion

Acute compartment syndrome is a common complication arising from vascular injury, prolonged immobility/compression, or bony fractures. The classic presentation includes a painful, tense, and tender compartment, potentially progressing to arterial insufficiency. Early signs involve pain, often described as disproportionate to the physical exam and worsening with passive movement.<sup>1,2</sup> Pallor and poikilothermia may also be present initially.<sup>2</sup> Paresthesia, pulselessness, and paralysis represent later indicators. Diagnosis is primarily clinical, based on these symptoms. Compartment pressure measurements can be a helpful adjunct if the exam is inconclusive or the patient's mental state hinders a reliable assessment.<sup>1-3</sup> While no single pressure value is definitive, a reading exceeding 30 mmHg or a perfusion pressure below 30 mmHg may warrant surgical intervention.<sup>2</sup>

Management of ACS focuses on addressing the systemic sequela of ischemic tissues and performing urgent compartment decompression to prevent irreversible ischemia due to compromised blood flow.<sup>1-3</sup> Ischemic muscle cells can release myoglobin and other harmful metabolites (rhabdomyolysis), leading to various derangements. Myoglobinemia can cause renal failure necessitating dialysis, while lactic acidosis exacerbates cell death and potassium release, potentially resulting in hyperkalemia-induced cardiac arrhythmias and weakness.<sup>4,5</sup> Hyperkalemia typically manifests as paresthesia progressing to ascending paralysis, sparing the head and respiratory muscles. Calcium administration serves as the immediate treatment for hyperkalemia by stabilizing the myocardium through a shift in its resting potential.<sup>5</sup> However, this effect, though rapid, is short-lived (30-60 minutes), and definitive management like hemodialysis may still be necessary, as seen in our patient's case.

Following patient stabilization, fasciotomy is the preferred intervention for viable muscle tissue. However, for tissue exhibiting irreversible ischemia, as evidenced by rigor mortis, complete loss of vascular signal on imaging, and profound anesthesia, amputation may be necessary.<sup>6</sup> Delay or inadequate compartment release increases the risk of limb loss, further metabolic derangements, and even mortality.<sup>7,8</sup>

The two-incision, four-compartment fasciotomy is a common approach for the lower leg, aiming to decompress all four compartments.<sup>7,8</sup> This procedure involves an anterolateral incision along the fibula and tibial crest, allowing access to both the anterior and lateral compartments. Careful dissection is required to identify and preserve the superficial peroneal nerve within the lateral compartment. A second, posterior incision is made medial to the tibial border to access and release the posterior compartments.<sup>7</sup> In the subcutaneous tissue of this posterior incision, the saphenous vein is identified and preserved. Finally, after releasing the superficial compartment, the soleus muscle is divided from the medial tibia to release the deep compartment.

Fasciotomy carries the potential risk of reperfusion injury, leading to an initial worsening of metabolic imbalances and acidosis. Management strategies for this complication include diuresis or dialysis, along with appropriate buffering using intravenous bicarbonate solutions.<sup>9</sup> Once systemic illness resolves and physiological pressures return, fasciotomy closure can be attempted. Primary closure is

preferred if tension-free closure can be achieved; alternatively, skin grafting may be necessary.<sup>10</sup> Delayed primary closure is another option, often facilitated by negative pressure wound therapy techniques like the “shoelace” or “Roman sandal” methods. In some cases, wounds may be left to heal by secondary intention.<sup>11</sup>

This case presented a diagnostic challenge due to the atypical delayed onset of compartment syndrome three days following the knee injury. Despite this uncertain setting, readily available CT imaging was crucial in our workup. The CT scan ruled out alternative diagnoses like vascular injury that might have necessitated a different surgical approach. In the absence of skeletal or vascular findings on imaging, we suspect a prolonged period of compression on the second night after the injury likely triggered the development of compartment syndrome.

If there was a sedative component, his pain medication use could help explain the scenario, as a single instance of sedative medication use associated with ACS and paraplegia has been reported.<sup>12</sup> We propose that the patient’s immobility, potentially induced by pain medication or sedation, may have triggered compartment syndrome and subsequent rhabdomyolysis. This, in turn, could have progressed to the severe hyperkalemia and quadriplegia observed. However, it is important to acknowledge that this specific complication sequence has not been previously documented in the literature.

The timing of his fasciotomies was emergent, delayed only by the need for partial correction of his severe hyperkalemia. Theoretically, compartment release can exacerbate hyperkalemia and acidosis, potentially leading to life-threatening cardiac complications. To mitigate this risk, we prioritized hemodialysis for immediate hyperkalemia correction, followed by bedside fasciotomies in the ICU to minimize further delay in decompression.

Ideally, with a normal EKG and no signs of hyperkalemia, fasciotomy would have been performed immediately to preserve the viability of the lower leg and foot. Conversely, if irreversible tissue loss had been evident, amputation would have been considered as the alternative course of action.

## Conclusion

Acute compartment syndrome is a frequently encountered and potentially life-threatening clinical entity affecting the limbs. While history and physical examination are the cornerstones of diagnosis, this case highlights the importance of recognizing atypical manifestations. A meticulous physical exam is essential for timely identification. Emergent treatment of systemic sequelae followed by prompt fasciotomy involving all affected compartments, is crucial to minimize morbidity and mortality associated with compartment syndrome.

## Lessons Learned

Even seemingly minor injuries, particularly those causing immobility and compression, can lead to acute compartment syndrome and subsequent rhabdomyolysis. While both conditions carry significant life-threatening risks, rapid diagnosis and treatment, including corrective measures for metabolic imbalances and surgical decompression, can achieve optimal outcomes, even in severe presentations.

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