Chapter 57
Approach to Crush Injury and Compartment Syndrome in the ICU

57.1 Introduction

Crush injuries and compartment syndrome are critical conditions frequently encountered in intensive care units (ICUs), particularly following natural disasters, vehicular accidents, industrial mishaps, and other traumatic events. A crush injury results from prolonged compression of muscle tissue, leading to significant morbidity and mortality if not promptly recognized and managed. When the force or duration of compression is substantial, it can progress to crush syndrome, a systemic condition characterized by severe metabolic disturbances and acute kidney injury due to rhabdomyolysis. Compartment syndrome, an emergency condition marked by elevated intra-compartmental pressure within muscle compartments, can lead to irreversible muscle and nerve damage, limb loss, or even death without timely intervention. This chapter provides a comprehensive overview of the pathophysiology, clinical presentation, diagnosis, and management strategies for crush injuries and compartment syndrome in the ICU setting, emphasizing the importance of early recognition and intervention to optimize patient outcomes [1, 2] [Ref: Algorithm 57.1].

57.2 Pathophysiology

57.2.1 Crush Injury and Crush Syndrome

A crush injury occurs when significant force compresses muscle tissue, causing direct cellular damage and disruption of the sarcolemma. This disruption leads to the release of intracellular contents—including potassium, myoglobin, creatine kinase (CK), phosphate, and other metabolites—into the systemic circulation. The sudden influx of potassium can result in hyperkalemia, posing a risk for

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life-threatening cardiac arrhythmias. Myoglobin, a heme-containing protein released from damaged muscle cells, can precipitate in the renal tubules, especially under acidic and hypovolemic conditions, leading to acute tubular necrosis and subsequent acute kidney injury (AKI), a phenomenon known as myoglobin-induced nephropathy.

Rhabdomyolysis, the breakdown of muscle fibers with the release of their contents into the bloodstream, is a hallmark of crush syndrome. In addition to hyperkalemia and AKI, metabolic acidosis develops due to the accumulation of lactic acid from anaerobic metabolism in ischemic tissues and the release of phosphate and sulfate from damaged cells. Hypocalcemia and hyperphosphatemia may also occur as phosphate binds calcium, reducing serum calcium levels. The massive fluid sequestration into damaged muscle compartments can lead to hypovolemia, exacerbating renal impairment and hemodynamic instability.

57.2.2 Compartment Syndrome

Compartment syndrome arises when increased pressure within a closed osteofascial compartment compromises circulation and function of the tissues within that space. Elevated intra-compartmental pressures exceed capillary perfusion pressure, leading to tissue ischemia and hypoxia. As ischemia progresses, cellular metabolism shifts to anaerobic pathways, producing lactic acid and resulting in metabolic acidosis. The increased vascular permeability associated with ischemia causes edema formation, further elevating intra-compartmental pressure and perpetuating a vicious cycle of worsening ischemia and tissue damage. If unrecognized or untreated, compartment syndrome can lead to irreversible muscle and nerve damage within hours, emphasizing the urgency of prompt diagnosis and intervention.

57.3 Etiology and Risk Factors

Crush injuries commonly result from events such as natural disasters (e.g., earthquakes), vehicular accidents, structural collapses, industrial accidents, and situations involving prolonged immobilization under heavy objects. Compartment syndrome is frequently associated with fractures, particularly of the tibia and forearm bones, tight casts or circumferential dressings that compress underlying tissues, burns leading to circumferential eschar formation, high-energy trauma causing significant soft tissue damage, and reperfusion injuries following the relief of arterial occlusion. Prolonged immobilization and constrictive bandages can also precipitate compartment syndrome. Understanding these risk factors is crucial for early identification and prevention of complications in high-risk patients.

57.4 Clinical Presentation

57.4.1 Crush Injury and Crush Syndrome

Patients with crush injuries may present with localized signs such as swelling, pain, bruising, and sensory or motor deficits in the affected area. Systemic manifestations include myoglobinuria, evidenced by dark brown urine, which indicates rhabdomyolysis. Laboratory findings often reveal hyperkalemia, hypocalcemia, hyperphosphatemia, elevated creatine kinase levels, and metabolic acidosis. Acute kidney injury may develop due to myoglobin-induced nephropathy, necessitating prompt recognition and management. The severity of symptoms correlates with the extent of muscle damage and the duration of compression.

57.4.2 Compartment Syndrome

The clinical hallmark of compartment syndrome is pain out of proportion to the injury, often described as deep, persistent, and poorly localized. Pain is exacerbated by passive stretching of the muscles within the compartment. Other symptoms include paresthesia (numbness or tingling), indicating nerve ischemia, and, in later stages, paralysis due to nerve and muscle dysfunction. Physical examination may reveal tense and swollen compartments that feel firm upon palpation. Pallor and diminished pulses may occur, but importantly, the presence of distal pulses does not exclude the diagnosis, as arterial blood flow may remain intact in the early stages. Reliance solely on the "5 P's" (pain, pallor, paresthesia, paralysis, pulselessness) can be misleading; therefore, a high index of suspicion and careful clinical assessment are essential.

57.5 Diagnosis

57.5.1 Crush Syndrome

The diagnosis of crush syndrome involves a combination of clinical assessment and laboratory investigations. Key laboratory tests include serum electrolytes, revealing elevated potassium levels (hyperkalemia), decreased calcium levels (hypocalcemia), and increased phosphate levels (hyperphosphatemia). Renal function tests often show elevated creatinine and urea levels, indicating impaired renal function. Creatine kinase levels are markedly elevated, reflecting muscle breakdown, with levels exceeding 5000 U/L suggestive of significant rhabdomyolysis. Myoglobin

levels may also be elevated, although they are not routinely measured. Urinalysis showing myoglobinuria (positive dipstick for blood without red blood cells on microscopy) supports the diagnosis of rhabdomyolysis.

57.5.2 Compartment Syndrome

Compartment syndrome is primarily a clinical diagnosis, with emphasis on early recognition of symptoms. Intra-compartmental pressure measurement is a valuable adjunct when the clinical assessment is inconclusive, particularly in unresponsive or sedated patients. Pressures exceeding 30 mmHg are indicative of compartment syndrome. Alternatively, a differential pressure (diastolic blood pressure minus compartment pressure) less than 30 mmHg also supports the diagnosis. It is crucial to monitor intra-compartmental pressures in high-risk patients, even when clinical signs are subtle. Avoidance of regional anesthesia in suspected cases is important, as it can mask pain symptoms and delay diagnosis. Imaging studies are generally not helpful and can delay definitive treatment.

57.6 Management in the ICU

57.6.1 Immediate Stabilization

Initial management focuses on stabilizing airway, breathing, and circulation according to standard trauma protocols. Securing the airway and ensuring adequate oxygenation are essential, considering potential respiratory compromise from systemic effects. Rapid vascular access with large-bore intravenous lines is established to initiate aggressive fluid resuscitation. Isotonic saline is the fluid of choice, aiming to maintain a urine output of 100–200 mL/hour to prevent myoglobin-induced acute kidney injury. Potassium-containing fluids, such as lactated Ringer's solution, should be avoided due to the risk of exacerbating hyperkalemia.

57.6.2 Fluid Management

Aggressive intravenous hydration is critical to flush out nephrotoxic substances and preserve renal function. Isotonic saline is preferred to avoid introducing additional potassium into the circulation. Careful monitoring of fluid balance is necessary to prevent fluid overload or hypovolemia. Fluid resuscitation should be titrated based on hemodynamic parameters, urine output, and signs of volume status.

57.6.3 Electrolyte and Acid-Base Management

Hyperkalemia is a life-threatening complication that requires prompt treatment. Intravenous calcium gluconate is administered to stabilize cardiac membranes, followed by insulin with dextrose to facilitate the intracellular shift of potassium. Sodium bicarbonate may be used in cases of severe metabolic acidosis to further promote potassium uptake into cells and correct acidosis. Beta-2 agonists, such as nebulized salbutamol, can serve as adjunctive therapy to lower serum potassium levels. Hypocalcemia is corrected cautiously, typically only if symptomatic, to avoid the risk of arrhythmias due to rapid shifts in calcium levels. Continuous monitoring of electrolyte levels and acid-base status is essential to guide therapy.

57.6.4 Management of Compartment Syndrome

Prompt recognition and management of compartment syndrome are critical to prevent irreversible damage. Initial measures include removing any external constrictive devices, such as tight dressings or casts, and positioning the affected limb at the level of the heart to optimize venous return without compromising arterial flow. Elevation above heart level is avoided as it may decrease arterial perfusion.

Urgent surgical decompression via fasciotomy is indicated when clinical signs persist or intra-compartmental pressures exceed 30 mmHg. Fasciotomy involves longitudinal incisions to release all affected compartments, relieving pressure and restoring perfusion. The timing of fasciotomy is crucial, as delays can result in permanent tissue damage. All compartments at risk should be decompressed, and the incisions should be left open with appropriate wound management.

57.6.5 Postoperative Care

After fasciotomy, meticulous wound care is essential to prevent infection, a significant risk due to the open wounds created by the procedure. Regular inspection for signs of infection or necrosis is necessary, and repeated debridement may be required if necrotic tissue is present. Wound dressings should be maintained in a sterile manner, and appropriate antibiotic therapy initiated as prophylaxis against infection. Continuous reassessment of compartment pressures is important to detect any residual or recurrent compartment syndrome. Renal function should be monitored regularly, given the ongoing risk of myoglobin-induced nephropathy.

57.6.6 Renal Protection

Preventing acute kidney injury is a primary concern in crush syndrome management. Maintaining adequate urine output through aggressive hydration helps prevent myoglobin precipitation in renal tubules. Diuretics such as mannitol may be considered to promote diuresis but should be used cautiously and are not routinely recommended. Loop diuretics may be used in cases of fluid overload but are contraindicated in hypovolemic patients. Early initiation of renal replacement therapy, such as dialysis, is indicated if there is evidence of unresponsive renal failure or severe metabolic derangements.

57.6.7 Systemic Management

Pain control is a vital component of patient care but must be balanced against the need to monitor for signs of compartment syndrome. Opioid analgesics are administered judiciously, and regional anesthesia is avoided to prevent masking symptoms. Nutritional support with a high-protein diet is important to facilitate tissue repair and meet the increased metabolic demands of recovery. Infection control measures include prophylactic antibiotics post-fasciotomy and strict adherence to wound care protocols to prevent secondary infections.

57.6.8 Complications and Follow-Up

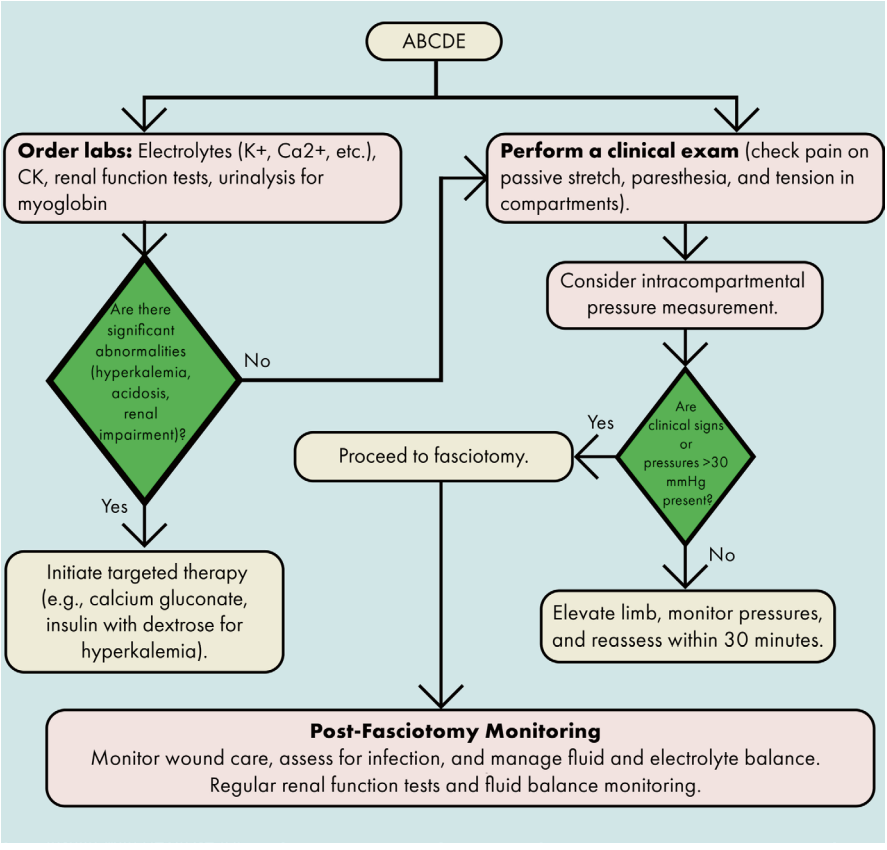
Patients with crush injuries and compartment syndrome are at risk for several complications. Acute kidney injury may progress to renal failure requiring dialysis. Systemic acidosis and persistent electrolyte imbalances, particularly hyperkalemia, can lead to hemodynamic instability and cardiac arrhythmias. Infection is a significant concern post-fasciotomy, necessitating vigilant wound care and appropriate antibiotic therapy. Delayed or inadequate treatment of compartment syndrome can result in irreversible muscle and nerve damage, leading to functional impairments.

Long-term follow-up includes regular assessment of limb function, wound healing, and renal function. Early involvement of rehabilitation services, such as physiotherapy and occupational therapy, is crucial to optimize functional recovery and assist patients in adapting to any residual deficits. Psychosocial support may be necessary to address the psychological impact of the injury and potential disability. Patient education on recognizing signs of complications and the importance of adherence to follow-up care is essential for optimal outcomes.

57.7 Conclusion

A structured and proactive approach to crush injury and compartment syndrome in the ICU is essential for optimizing patient outcomes. Early recognition of clinical signs, aggressive management of metabolic derangements, timely surgical intervention for compartment syndrome, and meticulous postoperative care are critical components in reducing morbidity and mortality. Interdisciplinary collaboration among critical care specialists, surgeons, nephrologists, and rehabilitation professionals is vital to provide comprehensive care and support to these patients. Through diligent monitoring and management, the risks of complications can be mitigated, limb function preserved, and survival rates improved.

Algorithm 57.1: Approach to crush injury and compartment syndrome in the ICU



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Part VI
Cardiology