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Compartment syndrome of the hand: A case report and review of literature

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ABSTRACT

Elevation of pressure within tightly bound myofascial compartments has detrimental consequences if not treated promptly, leading to a loss of circulation, ischemia, myonecrosis, nerve damage, and limb loss. They are commonly seen in the distal upper and lower extremities; however, compartment syndrome of the hand is rarely encountered and prompt recognition can prevent permanent damage and tissue loss. This case study presents a complicated case of compartment syndrome of the hand and discusses the interrelationship between compartment syndrome and rhabdomyolysis. An emphasis is placed on pathophysiology of this relationship to allow a better understanding of the imaging features as well as early clinical recognition of compartment syndrome. Magnetic resonance imaging findings are specifically discussed as it remains the best imaging tool to evaluate the extent of the damage and surgical planning.

Introduction

Compartment syndrome is a dreaded condition caused by increasing pressures within tightly bound myofascial compartments [1–3]. Compartment syndrome is most commonly seen in the forearm and legs, especially the lower leg [4]. However, rare cases of compartment syndrome involving the hand have been reported [3–5]. Rapid recognition and emergent fasciotomy to relieve the compartment pressure is imperative to prevent irreversible damage. The hallmark of compartment syndrome, if untreated, is elevation of the compartment pressure leading to vascular compromise and decreased perfusion. This cycle of pressure elevation continues as capillary permeability increases secondary to tissue damage, further exacerbating compartment edema [3]. Without treatment, tissue death ensues. Rhabdomyolysis is an associated condition defined by muscle breakdown and leakage of muscle contents into the circulation [2,6,7]. There is an intimate interplay between compartment syndrome leading to muscle breakdown and rhabdomyolysis-induced edema causing elevation of compartment pressure, which makes the 2 inherently co-dependent in tight myofascial compartments. Imaging plays an important role in evaluation of compartment syndrome and monitoring the progression of the disease process from edema to myonecrosis. Magnetic resonance imaging (MRI) is the best

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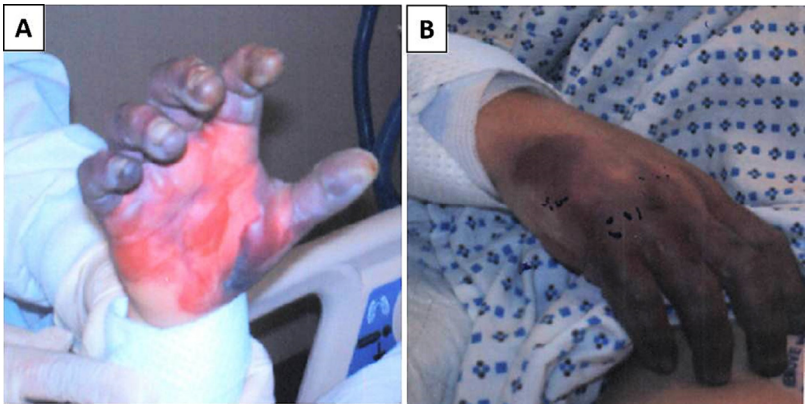

Fig. 1 – Palmer (A) and dorsal (B) aspects of the patient's right hand at the time of presentation demonstrate a molted appearance of all 5 digits.

modality that allows evaluation of the extent of tissue damage and management planning [6,7].

Case report

A 44-year-old white female with a history of multiple sclerosis and polysubstance abuse presented to the hospital after being found unresponsive and lying on her right hand for an unknown duration. She was reportedly seen in that position 14 hours prior by her spouse. On physical examination, her right hand appeared cool and mottled (Fig. 1). At the time of presentation, her vitals were as follows: temperature: 101°F, blood pressure: 96/77, heart rate: 100 bpm, and respiratory rate: 16 bpm with 100% O_2 saturation on room air. Arterial Doppler demonstrated patency of the major vessels of the right upper extremity including the radial and the ulnar arteries. Compartment pressures were not recorded.

Her laboratories at time of admission demonstrated leukocytosis with a white blood cell count of 25.8 th/L, creatinine of 8.83 mg/dL, potassium of 5.4 mmol/L, CK of 37453 IU/L (CK-MB of 98.3 ng/mL), and myoglobin of 3925 ng/mL. A CK-MM was not obtained. Her toxicology results were positive for benzodiazepines and opiates.

She was diagnosed with rhabdomyolysis complicated by renal failure. Her hand was deemed to be necrotic and gangrenous for which she eventually underwent wrist disarticulation (Fig. 2).

A hand radiograph demonstrated dorsal soft tissue swelling without underlying fracture or bone erosion. Before amputation, an MRI of the right hand with and without intravenous gadolinium contrast was obtained (Figs. 3 and 4). The MRI demonstrated diffuse skin thickening and enhancing subcutaneous edema throughout the right hand; there was nonenhancement throughout the subcutaneous fat dorsal to the third through the fifth metacarpals and at the thenar eminence, consistent with necrosis; there was also no enhancement of all the thenar, hypothenar, digitorum, and lumbrical muscles, consistent with muscular necrosis.

Pathologic analysis of the amputated right hand demonstrated gangrenous necrosis with fungal and rare hyphae infiltration of the necrotic skin, skin ulceration with purulent inflammation and focal fat necrosis, dermal fibrosis, and reactive epidermal changes at the resection margin.

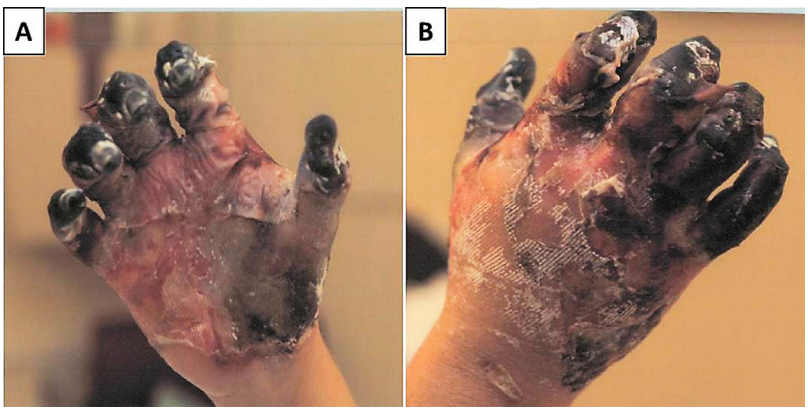

Fig. 2 – Palmer (A) and dorsal (B) aspects of the patient's right hand at the time of MRI demonstrate necrotic tissues within all 5 digits as well hand desquamation. MRI, magnetic resonance imaging.

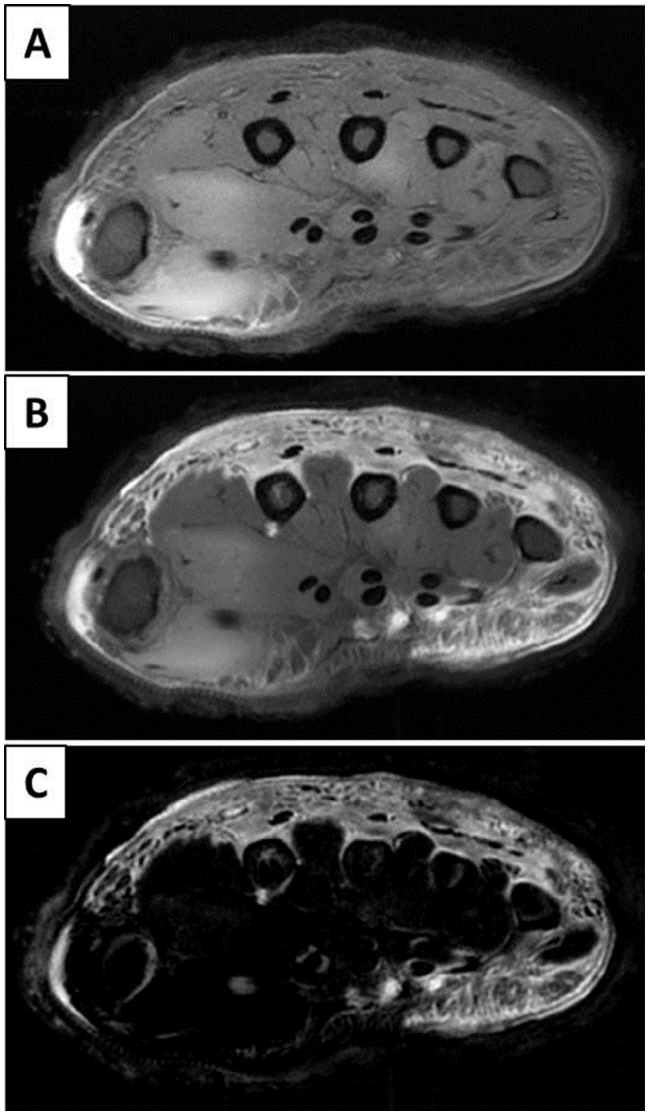

Fig. 3 – Diffuse skin thickening and enhancing subcutaneous edema with no enhancement within the thenar and hypothenar musculature consistent with myonecrosis. Axial T1-FS precontrast (A), postcontrast (B), and subtraction (C) images.

Discussion

Compartment syndrome and rhabdomyolysis

Compartment syndrome is caused by the development of high pressure within tightly confined myofascial compartments bound by thick connective tissue or bone [1–3]. Vascular collapse and hypoxia secondary to elevated pressure are the hallmarks of compartment syndrome [3,8]. Elevation in pressure can be caused either by an increase in intracompartmental volume or external compression. Examples of intracompartmental causes of pressure elevation include edema, hematoma, and fracture, whereas external compression factors include burns, prolonged immobilization, and tight wrapping [3,7,8]. An elevation in pressure leads to venous occlusion and arterial collapse, capillary ischemia, and nerve damage [1–4,8]. As the energy-dependent transcellular pumps fail because of ischemia, muscle cell swelling ensues, further contributing to increased pressures within the compartment, continuing the cycle of hypoxia and muscle damage [2,3]. The clinical presentation is variable based on the degree of injury and usually includes disproportional pain, weakness or paralysis, hypoesthesia or paresthesia, and tightness and pallor from circulatory compromise [3,7]. Early recognition and immediate fasciotomy is required for decompression of the affected compartment and to prevent progression of muscle damage [1–3,6,7]. Intracompartmental pressure monitoring becomes very important evaluating compartment pressures [1,3]. Compartment pressure of over 30 mm Hg has been shown to cause significant clinical muscle ischemia and can aid the clinician with the decision to perform immediate fasciotomy [2,3,6,7]. Prolonged compartment pressure leads to reversible damage within 4 hours and irreversible myonecrosis and nerve damage by 8 hours, requiring surgical removal of the dead tissue [3,7,9].

Rhabdomyolysis is a known complication of compartment syndromes. By definition, rhabdomyolysis is rapid muscle breakdown with destruction of the myocytes with subsequent leakage of intracellular contents into the circulation [2,6,7]. In addition to compression-based injury, multiple other causes of rhabdomyolysis have been identified such as trauma, severe exercise, ischemia, burns, autoimmune disease, seizures, prolonged immobility, and toxins, including drugs, medications, and alcohol abuse [2,7]. The clinical presentation of rhabdomyolysis and treatment options are variable given

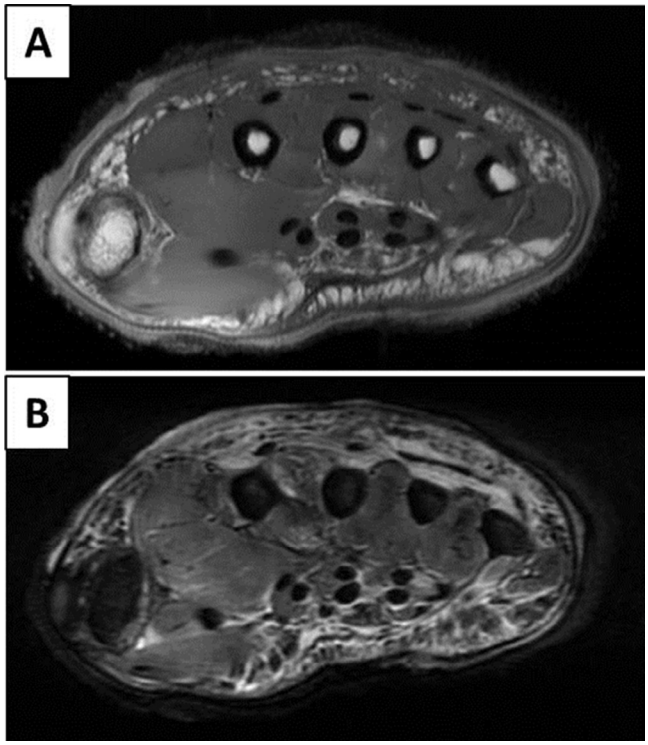

Fig. 4 – (A) Axial T1 image demonstrates normal bone marrow signal. (B) Axial T2 STIR image demonstrates intramuscular edema. STIR, short tau inversion recovery.

the variety of underlying causes and can range from minor to severe based on the extent of the muscle damage [1,6,7].

Rhabdomyolysis can lead to many complications, the most common of which is acute kidney injury. Other well-established complications include electrolyte imbalance secondary to muscle breakdown, volume depletion, acidosis, and disseminated intravascular coagulation. Furthermore, compartment syndrome in itself can be a complication of rhabdomyolysis caused by the increasing edema associated with rhabdomyolysis in tightly bound compartments, leading to increase in the intracompartmental volume [2,7].

The diagnosis of rhabdomyolysis is made with laboratory values that measure markers of muscle breakdown. These laboratories include elevated serum creatine kinase (CK), more specifically, CK-MM subtype, which is the most sensitive marker of muscle breakdown [7]. CK rises within 2-12 hours after the initial muscle injury with a peak at 24-72 hours; this demonstrates gradual decline over 7-10 days for a resolving injury [7]. CK may continue to persist in cases of continued damage as seen in compartment syndrome [7]. A value of 5 times the normal limit is generally accepted as an indicator of rhabdomyolsis [7]. Other markers include serum and urine myoglobin which, when abnormal, are reliable indicators of rhabdomyolysis [2,7]. Serum myoglobin elevation is, in fact, considered pathopneumonic for rhabdomyolysis [7].

Compartment syndrome of the hand

Compartment syndrome of the hand is uncommon and caused by the same general causes mentioned previously like trauma, drug overdose, and crush syndrome [3–5]. Anatomically, the hand can be divided into 10 separate compartments and includes the hypothenar, thenar, adductor, carpal canal, finger, and 4 interosseous compartments [3,4,9]. The arterial supply to the hand is provided by deep and superficial arches supplied by the ulnar and radial arteries [4]. The most common presenting symptom of hand compartment syndrome is swelling, minus posturing and pain out of proportion to the injury, exacerbated by passive stretching [3,4]. The diagnosis is often made based on clinical suspicion, although intracompartment pressures may aid in the diagnosis and monitoring the hand [4,5]. Emergent fasciotomy remains the primary treatment for hand compartment syndrome. Because extensive data on hand compartment syndrome are lacking, no definitive consensus exists for its diagnosis, and clinical judgment on the part of the clinician is imperative [4,5]. Hand compartment syndrome usually requires multiple incisions to relieve pressures in all involved compartments [4].

MRI features

Compartment syndrome is a clinical diagnosis, and no imaging is needed for initial diagnosis or emergent fasciotomy. However, MRI can enhance diagnostic accuracy as it is the ideal imaging modality to examine the extent of muscle involvement and the degree of potential reversibility [6,7]. It is able to provide information on progression of rhabdomyolysis to myonecrosis as well as assess early ischemic changes vs irreversible myonecrosis [6]. Additionally, MRI is the modality of choice for accurate localization of the affected muscle groups for treatment planning, including surgical debridement [6,7].

Early muscle damage is characterized by muscle edema which is seen as hyperintense signal abnormality on T2W or short tau inversion recovery sequences [1,6,7]. These findings are always seen with increased intra- or extracellular free water content that defines muscle edema [1]. Muscle edema is nonspecific and seen with multiple pathologic processes, such as myositis or myopathy [1,6]. However, MRI does provide nearly 100% sensitivity in detecting the early edematous changes associated with rhabdomyolysis and compartment syndrome [7]. T1 signal may be isointense or hyperintense in the early stages. Contrast-enhanced images generally demonstrate diffuse muscular enhancement. The severity of the injury correlates with the degree of signal intensity alteration and recovers in parallel with clinical improvement [1].

As the muscle progresses toward myonecrosis, postcontrast MRI imaging demonstrates a complete lack of internal muscular enhancement, as would be expected with dead tissue. Peripheral and stippled enhancement of any remaining surviving muscle fibers has also been described [6,10]. Gradual decrease in T1W and T2W signal intensity evolving to both T1W and T2W hypointensity indicate fibrotic changes and hemosiderin deposition from muscular damage and hemorrhage [1,6]. For chronic, untreated myonecrosis, an overall reduction in muscle mass would also be expected.

REFERENCES

[1] May DA, Disler DG, Jones EA, Balkissoon AA, Manaster BJ. Abnormal signal intensity in skeletal muscle at MR imaging: patterns, pearls, and pitfalls. Radiographics 2000;20:S295–315. Spec No.

[2] Vanholder R, Sever MS, Erek E, Lameire N. Rhabdomyolysis. J Am Soc Nephrol 2000;11(8):1553–6.

[3] Oak NR, Abrams RA. Compartment syndrome of the hand. Orthop Clin North Am 2016;47(3):609–16.

[4] Reichman EF. Compartment syndrome of the hand: a little thought about diagnosis. Case Rep Emerg Med 2016;2016:2907067.

[5] Codding JL, Vosbikian MM, Ilyas AM. Acute compartment syndrome of the hand. J Hand Surg Am 2015;40(6):1213–6.

[6] Cheng YC, Lan HHC, Shih CH, Chen CCC, Lee SK. Magnetic resonance imaging of rhabdomyolysis: muscle necrosis versus ischemia. J Radiol Sci 2013;38(4):143–8.

[7] Keltz E, Khan FY, Mann G. Rhabdomyolysis. The role of diagnostic and prognostic factors. Muscles Ligaments Tendons J 2013;3(4):303–12.

[8] Ahmad FU, Madhavan K, Trombly R, Levi AD. Anterior thigh compartment syndrome and local myonecrosis after posterior spine surgery on a Jackson table. World Neurosurg 2012;78(5):553, e5–8.

[9] Chandraprakasam T, Kumar RA. Acute compartment syndrome of forearm and hand. Indian J Plast Surg 2011;44(2):212–8.

[10] Cunningham J, Sharma R, Kirzner A, Hwang S, Lefkowitz R, Greenspan D, et al. Acute myonecrosis on MRI: etiologies in an oncological cohort and assessment of interobserver variability. Skeletal Radiol 2016;45(8):1069–78.