

Bilateral acute exertional compartment syndrome of the forearms in a sport climber: a case report

CASE REPORT

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Introduction: Acute exertional compartment syndrome (AECS) of the forearm is rare, and bilateral cases are reported only sporadically. In sport climbers, the far more familiar entity is chronic exertional compartment syndrome (CECS) of the forearm, for which fasciotomy is elective. That mismatch in expected time pressure, paired with sparse upper-extremity AECS literature, drives delayed diagnosis and avoidable functional loss.

Case Presentation: A previously healthy man in his early thirties presented with bilateral forearm tightness, swelling, median and ulnar paresthesia, and inability to make a fist after a single high-volume climbing day combining outdoor multi-pitch effort with extended hangboard and campus-board training. Examination showed bilateral volar swelling, cyanotic discoloration, and reduced two-point discrimination with intact distal pulses. Serum creatine kinase peaked at 31,420 units/L, and forearm magnetic resonance imaging demonstrated diffuse volar compartment T2 hyperintensity. Right-sided compartment pressures (volar 58 and 62 mmHg, dorsal 38 mmHg, mobile wad 42 mmHg) prompted emergent two-incision fasciotomy. The contralateral forearm progressed clinically and was decompressed on hospital day 2. Both wounds underwent delayed primary closure. At six months the QuickDASH score was 14, and at three years the patient had returned to recreational climbing near his pre-injury level.

Discussion: Forearm AECS in athletes is uncommon and easily mistaken for chronic exertional compartment syndrome in the climbing population. The constellation of hours-acute symptom onset, marked rhabdomyolysis, and progressing neurologic deficit distinguished AECS from CECS in our patient and triggered emergent compartmental pressure measurement. Bilateral cases can present asynchronously; contralateral surveillance was the practical refinement that permitted timely staged decompression.

Conclusion: Bilateral forearm AECS in a sport climber is easily anchored to CECS. Recognition rests on the constellation of hours-acute onset, marked rhabdomyolysis, and progressing neurologic deficit, with contralateral surveillance

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for asynchronous bilateral progression. Decompression within the six-hour window can preserve near-baseline function.

Keywords: *Compartment Syndrome, Fasciotomy, Rhabdomyolysis*

INTRODUCTION

The acute compartment syndromes are conventionally divided into acute traumatic compartment syndrome (ATCS), driven by a discrete injury, and acute exertional compartment syndrome (AECS), driven by extreme physical effort; both produce rising intracompartmental pressure that compromises microcirculation and, untreated, progresses to ischemic muscle and nerve injury¹. ACS predominantly affects young men, with an annual incidence of 7.3 per 100,000 reported in this group². ATCS is comparatively easy to suspect given its near-invariable link to fracture or crush; AECS, by contrast, is a diagnosis of exclusion that requires a high index of suspicion at presentation³.

Several features push AECS toward delayed recognition. AECS is rare relative to ATCS, in which fracture-driven cases (most commonly tibial shaft) account for roughly three quarters of reported events^{4,5,6}. The published distribution of AECS itself skews toward the lower extremity, leaving upper-extremity cases sparse⁷; the most often-cited series, from Livingston and colleagues, is a pediatric population, and adult upper-extremity data are still scarcer⁷. In a sport climber, forearm pain leads most clinicians to consider chronic exertional compartment syndrome (CECS) of the forearm first, an entity for which fasciotomy is elective rather than emergent^{8,9}. The mismatch between CECS expectations and AECS time pressure drives diagnostic delay and worse functional outcomes⁷. Existing operative guidance addresses thresholds for fasciotomy but underspecifies which compartments warrant measurement in the first place. We report this case of bilateral forearm AECS in a sport climber to address that gap and articulate the clinical constellation that, in our experience, distinguishes acute from chronic exertional disease at the bedside.

CASE PRESENTATION

A previously healthy male in his early thirties presented to the emergency department with bilateral forearm tightness, painful swelling, paresthesia involving the median and ulnar nerve distributions of both hands, and an inability to make a full fist. The diagnostic timeline was as follows: climbing-related symptom onset in the late evening; emergency department arrival approximately 14 hours after symptom onset; serum chemistries returned within the first hour of arrival; magnetic resonance imaging completed at approximately 18 hours from symptom onset; intracom-

partmental pressure measurement at approximately 22 hours; and right-sided two-incision fasciotomy completed at approximately 24 hours from symptom onset.

He attributed the onset to a single climbing day the previous afternoon, an outdoor multi-pitch session followed several hours later by an extended indoor training block of hangboard repeaters and campus-board work, totaling approximately four hours of sustained gripping after the cliff descent. Symptoms developed within hours of completing the training block and worsened through the night.

On examination both forearms were diffusely swollen and tender, the volar compartments tauter than the dorsal, with cyanotic discoloration of the distal forearms and dorsa of the hands. Passive extension of the fingers reproduced severe pain in both forearms; pain was rated 8/10 on the right and 6/10 on the left at presentation. Grip strength was reduced bilaterally, although objective dynamometry was not performed at any visit. Two-point discrimination was diminished across the median and ulnar distributions of both hands. Radial and ulnar pulses remained palpable bilaterally with capillary refill under three seconds.

Initial laboratory studies showed marked rhabdomyolysis. Serum creatine kinase measured 31,420 units/L on the first draw and 29,150 units/L two hours later. Serum AST was elevated at 286 units/L, ALT at 84 units/L, and basic chemistries were within normal limits (estimated GFR 99 mL/min, potassium 4.1 mmol/L, calcium 9.2 mg/dL, albumin 4.2 g/dL). Complete blood count, urinalysis, and electrocardiogram were unremarkable.

Magnetic resonance imaging without contrast was obtained to characterize the extent of muscle injury. The specific scanner field strength and full sequence list were not recorded in the operative file at the time and could not be retrieved retrospectively (see Limitations); axial and coronal T2-weighted images formed the basis of clinical interpretation. The right forearm demonstrated diffusely increased T2 signal across the volar superficial and deep flexor compartments, with prominent involvement of flexor digitorum profundus and flexor pollicis longus and patchy signal change in the mobile wad (Fig. 1). The dorsal compartment showed mild T2 signal change without bulk enlargement. The left forearm showed an analogous pattern of diffuse volar T2 hyperintensity involving flexor digitorum superficialis and profundus, with milder mobile-wad and dorsal-compartment involvement (Fig. 2).

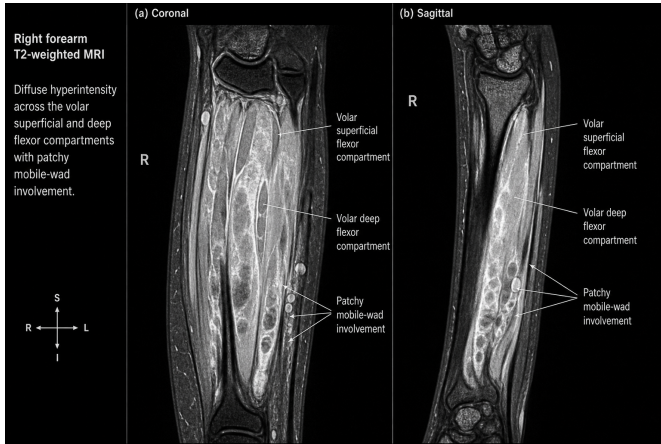


Figure 1. Right forearm T2-weighted magnetic resonance imaging, paired coronal (a) and sagittal (b) views, demonstrating diffuse hyperintensity across the volar superficial and deep flexor compartments with patchy mobile-wad involvement.

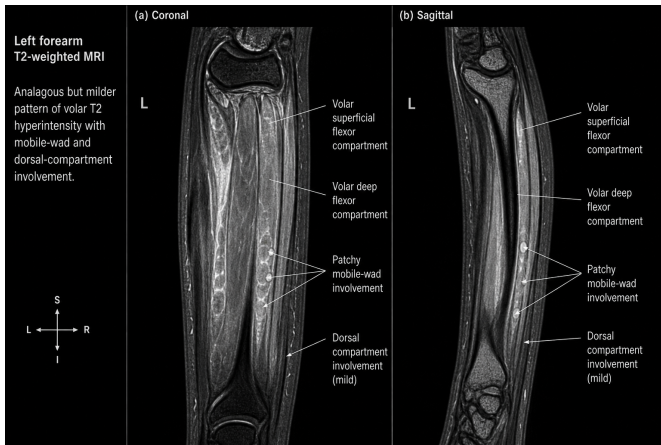


Figure 2. Left forearm T2-weighted magnetic resonance imaging, paired coronal (a) and sagittal (b) views, showing an analogous but milder pattern of volar T2 hyperintensity with mobile-wad and dorsal-compartment involvement.

The composite of clinical history, neurologic findings, marked rhabdomyolysis, and the MRI muscle-edema pattern raised concern for AECS and prompted compartmental pressure measurement. Pressures were obtained at rest using a commercially available compartment-pressure monitor (Stryker Intra-Compartmental Pressure Monitor System; Stryker). Specific technique parameters — needle type, exact intracompartmental position, number of measurements per compartment, and use of a side-port technique — were not documented in the operative record at the time of the case and are noted as a limitation of this report. On the right, the volar superficial compartment measured 58 mmHg, the volar deep compartment 62 mmHg, the dorsal compartment 38 mmHg, and the mobile wad 42 mmHg. On the left, the corresponding compartments measured 26, 28, 18, and 22 mmHg. The right-sided values sat unequivocally above the operative threshold and,

in the setting of progressing symptoms, warranted emergent decompression.

The right forearm was decompressed first, approximately 24 hours from symptom onset. A volar curvilinear (Henry-style) incision was carried from the antecubital fossa across the volar wrist crease and continued distally as a carpal tunnel release; the antebrachial fascia was opened along the entire length of the incision, the superficial flexor compartment released, and the deep flexor compartment opened by retracting the flexor carpi radialis and flexor digitorum superficialis to expose its underlying fascia. The mobile wad was released through the same incision. A separate longitudinal dorsal incision permitted release of the dorsal compartment. Tourniquet use and the specific fascial coverings preserved versus excised were not recorded in the operative dictation in retrievable detail (see Limitations). Muscles in both compartments bulged from their fascial envelopes on release; the proximal third of flexor digitorum profundus appeared dusky but contracted in response to electrical stimulation. The wound was packed and dressed without primary closure.

Overnight, the left forearm progressed clinically with worsening pain and increasing swelling. Contralateral decompression was performed approximately 36 hours from symptom onset (hospital day 2) using the same two-incision technique, with intra-operative findings of moderate volar bulge and intact muscle contractility throughout each released compartment without focal duskeness or contractile failure. Both forearms underwent delayed primary closure approximately one week later, with no requirement for split-thickness skin graft. Postoperative care included serial neurologic examinations, aggressive hydration, monitoring of renal function for myoglobinuric injury, and early hand-therapy involvement for range-of-motion preservation.

At one-month follow-up the patient demonstrated near-full forearm range of motion bilaterally, recovering grip strength on clinical examination (objective dynamometry was not performed), and a QuickDASH (Disabilities of the Arm, Shoulder and Hand short form) score of 31 on a 0–100 scale where higher scores indicate greater disability and lower scores reflect better function ¹⁰. By the six-month visit the QuickDASH score had improved to 14, with no swelling, tenderness, or warmth in either forearm. The patient reported intermittent paresthesia along the volar aspect of the right ring finger and a positive Tinel sign over the right cubital tunnel; nerve conduction studies of both upper extremities were ordered to evaluate residual ulnar neuropathy, but the patient declined to attend the appointment. We attributed the residual symptoms to post-AECS compression neuropathy on the basis of the asymmetry of the residual complaints relative to a symmetric operative

exposure; without nerve conduction studies, an iatrogenic component cannot be definitively excluded.

Long-term review at three years confirmed return to recreational sport climbing at a grade approximately one letter-grade below the patient's pre-injury maximum, without further compartment-related complaints. The patient continued to report intermittent right-hand paresthesia under sustained gripping load and again declined nerve-conduction testing. Outcome scoring was not formally repeated at the three-year visit; the patient described function as essentially restored for daily activity and only marginally limited at maximal climbing effort. The patient's pre-injury engagement included regular outdoor leading at a recreationally competitive grade, and at three-year review climbing remained recreational rather than formally competitive; this manuscript accordingly describes him as a sport climber rather than a competitive sport climber.

DISCUSSION

In a sport climber presenting with forearm pain, the strong clinical anchor is chronic exertional compartment syndrome (CECS) of the forearm, an entity for which fasciotomy is elective rather than emergent ^{8,9}. Our case argues that an acute exertional process can override that anchor when the time course, biochemical signature, and neurologic findings depart from the chronic pattern. We present this case not to revisit fasciotomy thresholds, which are well established, but to articulate the clinical constellation that should redirect a climber's forearm presentation from elective workup to emergent decompression.

The trigger in our patient was a single training day combining outdoor multi-pitch climbing with extended hangboard repeaters and campus-board work, a load profile in which sustained gripping demand exceeds forearm flexor recovery capacity. Schöffl et al. measured intracompartmental pressures during sport-specific climbing ergometry and described the physiologic ranges that delineate normal climbing load from sustained pathologic compression, with resting forearm flexor pressures in healthy climbers reported at approximately 5–13 mmHg and exercise-induced pressures during sustained handgrip rising into the 60–70 mmHg range before normalizing within minutes of cessation ⁸. Acute exertional compartment syndrome has been precipitated in athletic populations by analogous single-session overload patterns, often after a sudden change in training intensity or volume ¹¹.

Three features in our patient distinguished AECS from CECS at the bedside. First, the time course: symptoms developed within hours of completing the training block and progressed through the night, in contrast to the activity-correlated, exercise-resolving pattern of CECS. Second,

the biochemical signature: a peak creatine kinase of 31,420 units/L. Marked rhabdomyolysis is well-recognized in AECS and may be the strongest single laboratory clue, as CECS is not associated with rhabdomyolysis of this magnitude ¹¹. Third, the neurologic findings: bilateral median and ulnar paresthesia with diminished two-point discrimination at presentation, representing fixed deficits rather than the transient symptoms typical of CECS. Liu et al. described a parallel example in which an AECS patient initially diagnosed with muscle strain underwent eight days of conservative management before fasciotomy, ultimately requiring necrotic muscle debridement ¹². The classic five-finding mnemonic of pain, paresthesia, pallor, paralysis, and loss of distal pulses remains a useful bedside reminder, although our patient like many AECS patients retained palpable distal pulses ¹³. Magnetic resonance imaging served as a confirmatory adjunct: bilateral T2 hyperintensity across the volar flexor compartments with bulk muscle swelling is the expected pattern of exercise-induced compartment edema ¹⁴.

Diagnostic criteria differ between the chronic and acute presentations and are easily conflated. The modified Padowitz criteria (resting pressure ≥ 15 mmHg, ≥ 30 mmHg one minute after exercise, or ≥ 20 mmHg five minutes after exercise) were derived from exercise stress testing and apply to chronic exertional compartment syndrome ¹⁵, and are referenced here only as the framework for considering CECS as the differential diagnosis. For an acute exertional presentation the operative diagnostic anchor is absolute compartment pressure relative to operative threshold (≥ 45 mmHg by the Matsen criterion ¹⁷) together with the perfusion-pressure deficit (ΔP , the difference between diastolic blood pressure and intracompartmental pressure) per McQueen and Court-Brown, with $\Delta P < 30$ mmHg indicating decompression ¹⁹. Our patient's right-sided values met both the absolute and ΔP criteria for emergent decompression; the left side did not at initial measurement but progressed clinically within twelve hours.

Published evidence in acute upper-extremity compartment syndrome suggests that fasciotomy within six hours of presentation is associated with substantially fewer complications and shorter hospitalization than later decompression, although that evidence base rests on a single retrospective cohort of modest size and should be interpreted accordingly ¹⁶. Our patient's right-sided volar pressures (58 and 62 mmHg) and dorsal pressure (38 mmHg) sat above the 45 mmHg operative threshold described by Matsen et al. ¹⁷; decompression occurred at approximately 24 hours from symptom onset, beyond the published six-hour benchmark, with the contralateral side decompressed at approximately 36 hours after a twelve-hour interval of clinical and pressure surveillance. The left-sided pressures,

although individually below the absolute operative threshold at the time of initial measurement, were elevated above the resting forearm flexor pressures of approximately 5–13 mmHg reported in healthy climbers ⁸ and progressed clinically within twelve hours to require the same operative response. Bilateral cases in this category may present asynchronously, as in our patient; the contralateral side warrants close clinical and pressure surveillance even when initial values do not meet operative criteria. Maintaining this surveillance threshold, rather than adopting a uniformly low fasciotomy threshold, was the practical refinement that permitted timely staged decompression.

At three years our patient had returned to recreational climbing one letter-grade below his pre-injury maximum. Long-term outcome data for forearm AECS specifically are sparse. A 2024 systematic review of forearm CECS fasciotomy reported 94.2% return to sport at any level and 86.8% at pre-injury level or higher across 500 patients ¹⁸; that review is the closest comparable functional dataset for this anatomic location, though direct comparability to AECS outcomes is limited because CECS reflects elective decompression of a chronic process whereas our patient sustained a single severe acute event. Our patient's outcome is consistent with that range despite the more severe acute presentation, an outcome we attribute in part to decompression within the first 24 hours of symptom onset on the index side and within 36 hours on the contralateral side. The residual right-hand paresthesia and positive Tinel sign over the right cubital tunnel are most consistent with post-AECS compression neuropathy on the basis of the asymmetry of the residual complaints relative to a symmetric operative exposure; without electrodiagnostic confirmation, an iatrogenic contribution cannot be definitively excluded.

This report has several specific limitations. First, the single-patient design precludes inference about generalizability or about the relative weight of the three diagnostic features beyond what one case can demonstrate. Second, the patient declined nerve conduction studies on two occasions, which leaves the residual neuropathy electrodiagnostically uncharacterized and prevents definitive separation of post-AECS compression neuropathy from any iatrogenic contribution. Third, formal patient-reported outcome scoring (QuickDASH or equivalent) was not repeated at the three-year visit, leaving the long-term outcome described qualitatively. Fourth, objective grip-strength measurement by dynamometry was not performed at any visit. Fifth, several technical details (MRI scanner field strength and full sequence list, compartment-pressure measurement technique, intra-operative tourniquet parameters, and fascial coverings preserved versus excised) were not recorded in

retrievable detail at the time of the case and could not be reconstructed retrospectively.

CONCLUSION

In a sport climber presenting with forearm pain, the strong tendency to anchor on chronic exertional compartment syndrome can delay recognition of acute exertional compartment syndrome. Three features distinguish AECS from CECS in real time: an hours-acute time course, marked rhabdomyolysis, and progressing neurologic deficit. Contralateral surveillance, rather than reflexive bilateral decompression, was the practical refinement that permitted timely staged decompression in our patient. Published evidence supports decompression within six hours of presentation to minimize complications ¹⁶; in our case decompression occurred at approximately 24 hours on the index side and approximately 36 hours on the contralateral side, and near-baseline functional recovery and return to sport were nonetheless achieved at three-year follow-up.

ETHICS STATEMENT

In accordance with our institution's policy, this single-patient retrospective case report did not require formal Institutional Review Board approval and was deemed exempt from full board review. The work was conducted in accordance with the principles of the Declaration of Helsinki.

CARE CHECKLIST

This report adheres to the CARE (CAse REport) guidelines, and a completed CARE checklist is provided as supplementary material.

DECLARATIONS

Funding: No external funding was received for this work.

Conflicts of Interest: None

Patient Consent: The case described in this report is fully de-identified. No recognizable patient images, dates, or distinguishing demographic details are presented. Per institutional policy and HIPAA Safe Harbor (or applicable regional equivalent), formal informed consent for publication was not required.

IRB / Ethics Approval: Institutional Review Board approval was not required for this report per institutional policy.

Data Availability: Data available on reasonable request

Author Contributions (CRediT): M.S. — Writing — original draft; Data curation; Project administration; S.B. — Conceptualization; Validation; Resources; Data curation; Investigation

Use of Generative AI: The authors did not use generative AI tools in the conception, drafting, or revision of this manuscript.

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