ISSN: 2584-0355
DOI: doi.org/10.51219/MCCRJ/Julian-Lloyd-Bruce/408

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Vol: 3 & Iss: 4

Case Report

Seven-Compartment Fasciotomy for Venous-Driven Acute Compartment Syndrome Secondary to Iliofemoral Deep Venous Thrombosis: A Case Report

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Citation: Bruce JL, Xenis E, Christodoulou I. Seven-Compartment Fasciotomy for Venous-Driven Acute Compartment Syndrome Secondary to Iliofemoral Deep Venous Thrombosis: A Case Report. *Medi Clin Case Rep J* 2025;3(4):1450-1455. DOI: doi.org/10.51219/MCCRJ/Julian-Lloyd-Bruce/408

Received: 01 October, 2025; **Accepted:** 21 October, 2025; **Published:** 23 October, 2025

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ABSTRACT

Acute compartment syndrome (ACS) of the lower limb is a surgical emergency that can rapidly lead to irreversible neurovascular injury and limb loss. This report describes a 40-year-old man with rapidly progressive swelling, cyanosis, severe pain and evolving foot drop in the setting of extensive iliofemoral deep venous thrombosis and prothrombotic comorbidities (systemic lupus erythematosus, chronic myeloid leukemia, prior venous thromboembolism and exogenous testosterone). Diagnosis was made clinically with duplex confirmation of proximal venous outflow obstruction. The surgical team performed an emergent seven-compartment fasciotomy, decompressing all three thigh compartments and all four lower-leg compartments, verified intraoperatively by visual and tactile findings. Wounds were managed open with negative-pressure therapy and a planned second look; anticoagulation began once hemostasis was secure and culture-directed antibiotics were given after Citrobacter koseri was isolated. Limb salvage was achieved, with short-term follow-up focused on wound progression, anticoagulation and neurologic rehabilitation. This case highlights venous-driven, limb-wide ACS and provides a practical approach when swelling spans the thigh and lower leg.

Keywords: Compartment syndromes; Fasciotomy; Venous thrombosis; Lower extremity; Iliofemoral vein

Introduction

Acute compartment syndrome is a time-critical surgical emergency in which elevated intercompartmental pressure impairs perfusion and rapidly threatens limb viability[1,2]. The leg is the most frequently involved site and population estimates suggest an incidence of about 7.3 per 100,000 in men and 0.7 per 100,000 in women[2]. High-energy fractures, particularly of the tibia, are common precipitants and timely fasciotomy is central to limb salvage[3,4]. When the examination is equivocal, intercompartmental pressure assessment can support decision-making, with a commonly cited differential threshold near 30 mm Hg, but clinical judgment remains paramount[1-3].

Beyond fracture and high-energy trauma, acute compartment syndrome can follow vascular injury, ischemia-reperfusion, prolonged compression and, more rarely, iliofemoral deep venous thrombosis with severe venous hypertension, sometimes presenting as phlegmasia cerulea dolens[5-7]. In such presentations, rapidly progressive swelling, pain out of proportion and evolving neurologic deficit may precede overt ischemic skin changes; management should prioritize timely decompression rather than extended imaging work-ups that risk delay[2,3].

We report a rare scenario requiring decompression of all three thigh compartments and all four leg compartments for limb salvage. Seven-compartment lower-limb releases have been described only in isolated case reports, underscoring the importance of early recognition and comprehensive fasciotomy when swelling spans both thigh and leg[8]. This report details the clinical presentation, rationale for broad decompression, perioperative decisions including anticoagulation and wound strategy and short-term outcomes.

Case Presentation

A 40-year-old man was referred from a private clinic for rapidly progressive right lower-extremity swelling and pain after a soft-tissue injury to the thigh. Over several days he developed marked edema, increasing tension in the thigh and leg and worsening pain despite analgesia. On arrival he was visibly cyanotic in the affected limb. Neurologic examination demonstrated a complete foot drop with 0/5 ankle dorsiflexion strength and inability to actively flex the ankle. Distal pulses were palpable and capillary refill was delayed **(Figure 1)**.

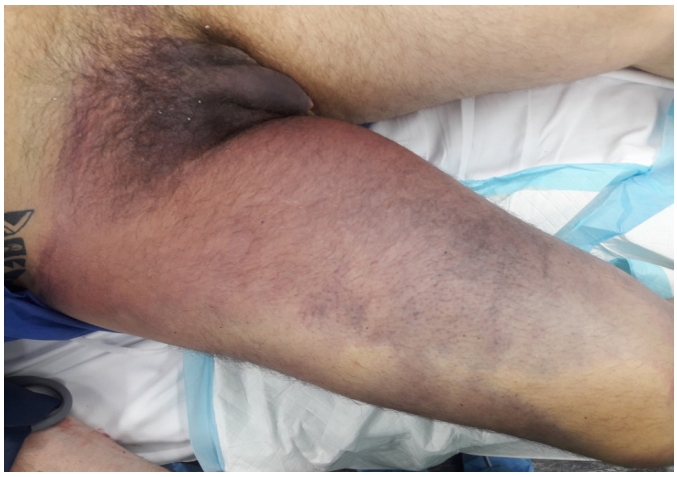

Figure 1: Preoperative discoloration of the right thigh and groin.

Marked purple-red discoloration extends from the distal thigh through the groin to the right hip, consistent with extensive venous congestion due to iliofemoral thrombosis and evolving compartment syndrome involving the proximal compartments.

Bedside duplex ultrasonography of the right lower extremity showed an occlusive femoral deep venous thrombosis with suspected proximal extension into the iliac system. Given the clinical picture of escalating pain, tense compartments spanning the thigh and leg and evolving neurologic deficit, acute compartment syndrome was diagnosed and the patient was taken emergently to the operating room **(Figure 4)**.

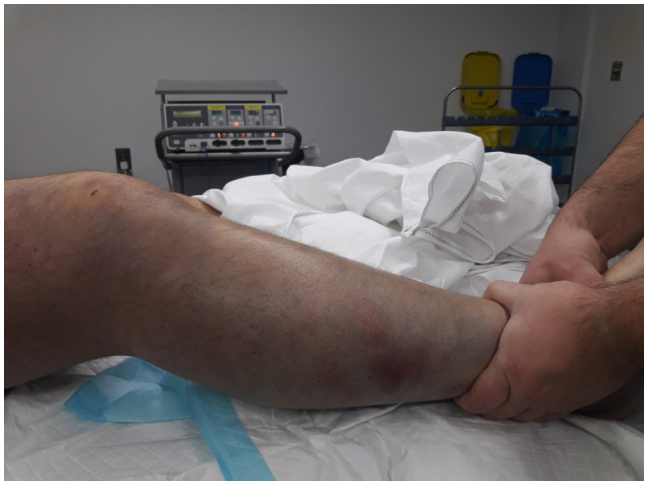

Figure 2: Clinical appearance of the right lower limb prior to fasciotomy.

The surgeon demonstrates pallor and diffuse purple discoloration of the tense, edematous leg. The limb was firm to palpation with absent distal pulses, consistent with acute compartment syndrome and severe venous hypertension.

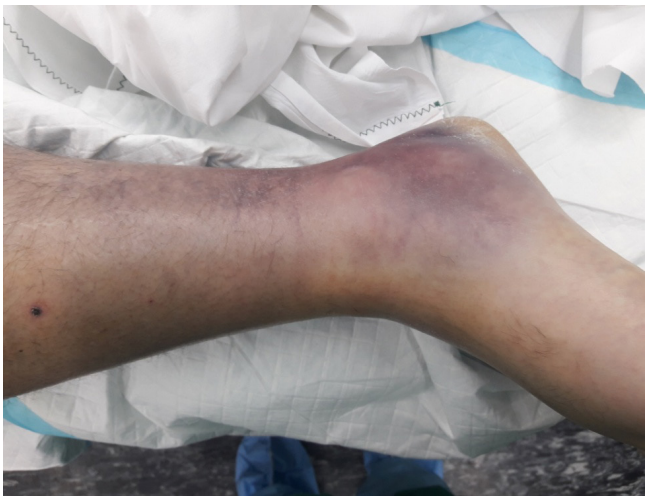

Figure 3: Right ankle showing severe dependent edema and ecchymosis.

Marked swelling and purple discoloration are evident around the ankle and distal leg, consistent with dependent venous congestion and ischemic changes prior to decompression.

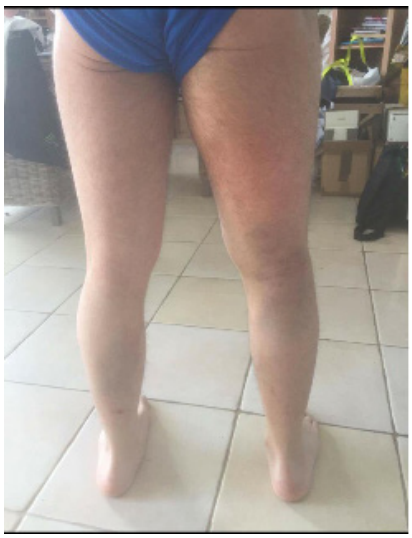

Figure 4: Posterior view of bilateral lower limbs.

Diffuse erythema and swelling of the right buttock, thigh, and lower leg are visible compared to the contralateral limb, reflecting extensive soft-tissue involvement secondary to iliofemoral outflow obstruction.

Fasciotomy was performed to decompress all seven compartments of the involved limb. The three thigh compartments were released using a lateral approach with extension as needed to ensure full decompression. The four compartments of the leg were released using a standard two-incision technique, ensuring thorough decompression of the deep posterior compartment. Intraoperatively, there was tense fascia with marked intramuscular swelling and hematoma; evacuation of large clot burdens was required. Multiple specimens were sent for culture and sensitivity **(Figure 5)**. Wounds were left open and two drains were placed. Negative-pressure wound therapy and staged closure were planned.

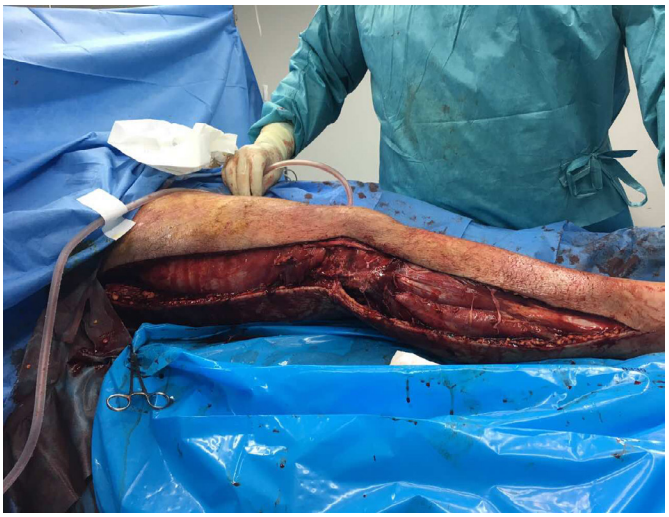

Figure 5: Intraoperative view of the right-sided fasciotomy.

Comprehensive decompression was achieved through a continuous lateral incision extending from the greater trochanter to the external malleolus, allowing release of all seven compartments. The photograph demonstrates exposure of underlying fascia and musculature under high tension along the full length of the limb.

Postoperatively, targeted wound management was provided. Tissue culture later grew Citrobacter koserii and antibiotic therapy was tailored to the organism's susceptibilities based on the antibiogram. Vascular surgery was consulted for thrombosis management, with systemic anticoagulation initiated once haemostasis was secured. The remainder of the postoperative course was uncomplicated.

Past medical history included systemic lupus erythematosus, chronic myeloid leukaemia, a prior venous thromboembolism and a previous methicillin-resistant Staphylococcus aureus infection of the upper extremity. The patient also reported exogenous testosterone use.

His past medical history includes systemic lupus erythematosus with concomitant arthritis, myositis, neutropenia, nephritis, cardiomyopathy, Raynaud's syndrome, alopecia, mouth ulcers, chronic myeloid leukaemia, deep vein thrombosis of the upper extremity, MRSA. He also used testosterone injections.

Key laboratory findings on presentation were notable for marked leucocytosis with neutrophilia, severe hyponatremia, mild hyperkalaemia, elevated transaminases, markedly increased lactate dehydrogenase, elevated CK-MB, very high C-reactive protein and an extreme creatine phosphokinase elevation consistent with rhabdomyolysis.

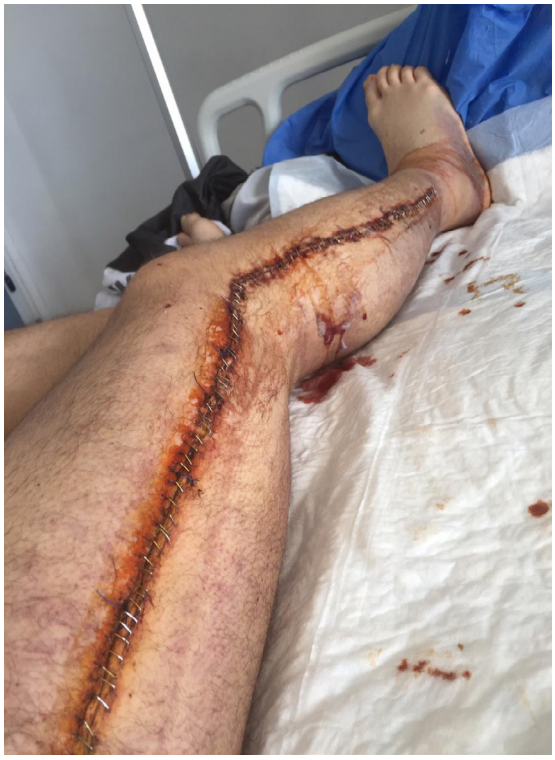

Figure 6: Post-fasciotomy closure of the right lower limb.

Stapled skin closure following staged wound management extends from the greater trochanter to the external malleolus along the lateral aspect of the limb. The wound surface was treated with povidone-iodine to reduce microbial contamination before dressing application.

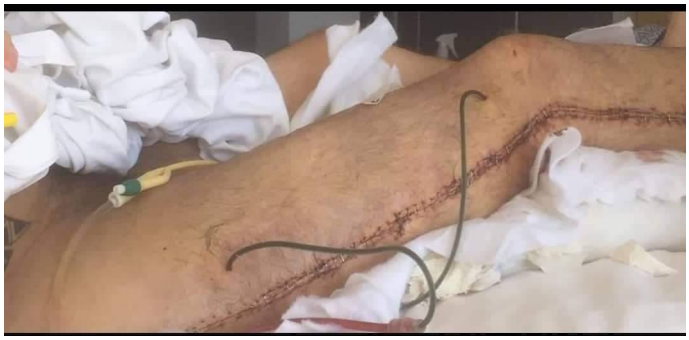

Figure 7: Placement of postoperative drains in the right lower leg.

Two closed-suction drains were positioned anteriorly in the proximal thigh following decompression extending from the greater trochanter to the external malleolus. Drain placement was performed to prevent postoperative fluid accumulation, reduce soft-tissue tension in the proximal compartments and facilitate wound healing during the early postoperative period.

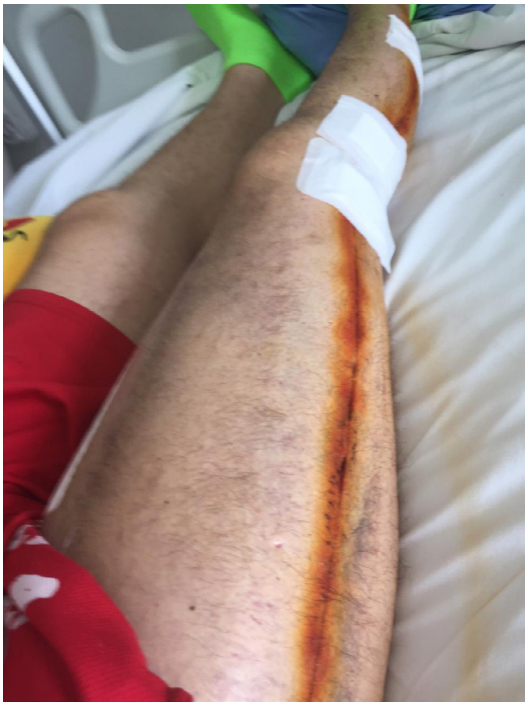

Figure 8: Follow-up appearance after staple removal.

Postoperative healing is evident along the previous fasciotomy line extending from the thigh to the ankle. Re-epithelialization and soft-tissue recovery are visible, with decreased swelling and restored contour of the right lower limb.

Representative laboratory values included:

- WBC: 24,220
- Neutrophils: 19,180
- Lymphocytes: 2,330
- Monocytes: 2.690
- Hemoglobin: 7.4
- Urea: 91
- Creatinine: 2.87
- Sodium: 121
- Potassum: 5.20
- SGPT: 123
- SGOT: 301
- Lactate Dehydrenase: 1,579
- CK-MB: 105.2
- CRP: 96.24
- CPK 12,613

Anatomy and operative considerations

The lower limb contains three thigh compartments (anterior, medial, posterior) and four lower leg compartments (anterior, lateral, superficial posterior, deep posterior). In diffuse swelling from iliofemoral venous thrombosis, pressure can rise across contiguous segments, so selective releases risk leaving ischemic muscle behind. The surgeons therefore planned comprehensive decompression from thigh to foot, with special attention to the deep posterior compartment of the leg and the medial thigh, which are commonly under-released in extensive edema.

Thigh

The surgeons used a lateral incision to decompress the anterior and posterior compartments and extended the exposure proximally or distally as needed to ensure full release. The medial compartment was assessed independently to avoid missing adductor compartment hypertension while protecting the femoral vessels and nerve. When fascia over the adductors remained tight, a separate medial release was performed.

Lower leg

A standard two-incision approach was used for the lower leg. Through the anterolateral incision, the surgeons released the anterior and lateral compartments while identifying and protecting the superficial peroneal nerve. Through a posteromedial incision, they released the superficial posterior compartment and then opened the deep posterior compartment along the posteromedial tibia. The soleus bridge was divided, the fascia over tibialis posterior was opened along its length and free excursion of flexor hallucis longus was confirmed.

Verification and postoperative strategy

At each level, the team confirmed complete decompression by direct visualization of muscle herniation, palpation of softened compartments and reassessment of tibial and peroneal nerve function. Wounds were left open with negative-pressure therapy, with a planned return to the operating room within 24 to 72 hours for reassessment and debridement as needed. Staged closure was achieved using dermatotraction or delayed primary closure when tissues permitted and anticoagulation decisions were coordinated with soft-tissue status and haemostasis.

Discussion

How the patient's comorbidities likely converged to cause seven-compartment ACS

This presentation can be explained by several prothrombotic influences acting together to produce a large iliofemoral deep venous thrombosis, marked venous hypertension and rapid limb swelling from thigh to foot. Systemic lupus erythematosus, chronic myeloid leukemia with possible treatment effects, a prior venous thromboembolism and exogenous testosterone likely increased clot formation and promoted proximal propagation once thrombosis began. High venous pressures accelerate interstitial fluid accumulation, lymphatic clearance is overwhelmed and compartment pressures rise in closed fascial spaces. As perfusion pressure falls, muscle and nerve become ischemic in parallel compartments, which fits the need for comprehensive seven-compartment decompression in this case[6,9,10].

Systemic lupus erythematosus and antiphospholipid pathways

People with SLE have higher venous thromboembolism rates, especially when antiphospholipid antibodies are present. Mechanistically, antiphospholipid antibodies activate endothelium and platelets through beta-2 glycoprotein I complexes, promote neutrophil extracellular traps and amplify complement, which increases tissue factor expression and thrombin generation. These changes favor rapid formation and extension of fibrin-rich thrombi in the iliac and femoral veins, raising venous pressure throughout the limb. In an SLE patient with a large iliofemoral DVT, early testing for lupus anticoagulant, anticardiolipin and anti-beta-2 glycoprotein I antibodies is useful because confirmed antiphospholipid

syndrome can alter intensity and duration of anticoagulation and supports closer surveillance for recurrence[11-17].

Chronic myeloid leukaemia and therapy-related risk

Cancer increases thrombotic risk through inflammation, procoagulant microparticles and altered blood counts. In CML, baseline venous risk is lower than in other myeloproliferative neoplasms, but treatment can modify that risk. Tyrosine kinase inhibitors have different vascular profiles. Ponatinib and to a lesser extent nilotinib and dasatinib, are linked to higher rates of arterial events and some venous thrombosis compared with imatinib. Endothelial stress, metabolic effects and platelet reactivity may all tilt the balance toward thrombosis and facilitate proximal clot propagation. If this patient was receiving a higher-risk TKI, that exposure could have compounded the SLE or antiphospholipid contribution and intensified venous outflow obstruction[18-21].

Exogenous testosterone as an amplifier

Observational reports connect testosterone therapy with venous thromboembolism, although meta-analyses show mixed results and the cardiovascular outcomes data are reassuring when therapy is appropriately indicated. Testosterone can raise haematocrit and viscosity and may influence platelet function and coagulation proteins. In a patient who already has strong prothrombotic drivers such as SLE or active malignancy, these changes can lower the threshold for thrombosis and support a larger thrombus burden once clotting begins. During the acute event, it is reasonable to reassess the necessity and timing of therapy while anticoagulation and wound management are being coordinated[22-25].

Effect of prior venous thromboembolism and proximal clot location

A previous DVT raises the risk of recurrence, particularly after proximal events. Residual venous obstruction and valve damage reduce venous reserve and make any new thrombus more likely to generate large pressure gradients. When a recurrent event involves the iliac and femoral segments, venous hypertension extends through both thigh and leg, accelerating edema and broadening the risk of compartment syndrome across multiple anatomical compartments[26].

How these risks combine to trigger compartment syndrome

The unifying pathway begins with iliofemoral outflow obstruction that drives venous hydrostatic pressure upward. Interstitial edema accelerates, intercompartmental pressure increases and capillary flow falls. Perfusion pressure can be understood simply as arterial pressure minus intercompartmental pressure. When those gradient narrows, tissue oxygen delivery drops and ischemia advances. Severe cases also develop secondary impairment of arterial inflow, a pattern similar to phlegmasia cerulea dolens. This sequence explains simultaneous compromise of thigh and leg compartments and supports early, comprehensive decompression for limb salvage[6,9,10].

Conclusion

This case describes acute compartment syndrome involving all three thigh compartments and all four compartments of the lower leg in the setting of extensive iliofemoral deep venous thrombosis. The presentation is best understood as the product of several prothrombotic influences acting together, including systemic lupus erythematosus, chronic myeloid leukaemia with possible treatment effects, a prior venous thromboembolism and exogenous testosterone. The resulting venous hypertension produced rapid, limb-wide edema and a critical fall in perfusion pressure across multiple compartments at once.

Management focused on timely recognition and decisive, comprehensive decompression. The surgical team planned release of all seven compartments from the outset, with special attention to the deep posterior compartment of the lower leg and the medial thigh, verified complete decompression intraoperatively and used staged wound care with a planned second look. Anticoagulation was coordinated with soft-tissue status and culture results guided antibiotic selection. This approach reflects a practical strategy when swelling spans the thigh and lower leg and neurologic deficits are evolving.

The clinical implications are straightforward. First, when a patient with strong thrombotic risk presents with large-territory iliofemoral thrombosis and tense swelling, clinicians should reassess compartments frequently and prioritize decompression over extended diagnostic workups when the examination is convincing. Second, when the anatomic extent of swelling is broad, proactive seven-compartment planning reduces the risk of under-release. Third, evaluation for antiphospholipid antibodies in systemic lupus erythematosus, documentation of tyrosine kinase inhibitor exposure in chronic myeloid leukaemia and reconsideration of exogenous testosterone can refine long-term anticoagulation and recurrence prevention. Finally, early rehabilitation and close follow-up are essential given the risk of persistent neurologic deficits.

Taken together, the case emphasizes that venous-driven compartment syndrome can be as time-critical as fracture-related disease. Recognizing the comorbidity stack, committing to comprehensive decompression when indicated and aligning anticoagulation with wound strategy are the key steps that support limb salvage and functional recovery.

Declarations

Ethics approval was not required for this case report. The patient provided informed consent to participate and anonymized medical data were collected and analysed for educational and publication purposes. All data supporting the findings of this case are contained within the manuscript and no additional datasets were generated. The author declares no conflicts of interest and received no external funding for this work. All contributions, including data analysis, literature review and manuscript preparation, were undertaken by the author.

Written consent for publication of anonymized clinical details and images was obtained from the patient through the Orthopaedic Surgery Department of Paphos General Hospital. The case was first presented at an orthopaedic conference and afterward the anonymized clinical information and relevant explanations were provided to the author for preparation of this report. This information was already in the public domain prior to submission of the manuscript for publication.

Acknowledgments

I extend my sincere thanks to the Orthopaedic Surgery Department of Paphos General Hospital for their generous support in developing this case report. Their detailed insights into the clinical background, surgical rationale and procedural

approach greatly deepened my understanding of complex compartment syndrome management. I am especially grateful for their provision of operative photographs, laboratory data and imaging studies, which enriched the clinical narrative and educational value of this manuscript. Their commitment to transparency and teaching exemplifies the collaborative spirit that advances clinical practice.

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