Case report

Lessons learned in the challenging case of evolving compartment syndrome with atypical presentation

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Accepted 20 October 2019

SUMMARY

We present the rare case of a non-painful compartment syndrome of the forearm in a 35-year old man with a history of intravenous drug abuse, and lessons learned which would have enabled earlier consideration of this limb threatening condition and earlier therapeutic intervention.

On initial assessment in the emergency department, the patient was drowsy, tachycardic and pyrexia. There was no obvious pain outwith expectation, and his forearm was erythematous and swollen. Due to the constellation of symptoms and signs, he was presumed to be septic from a forearm cellulitis and was thus treated for sepsis with intravenous antibiotics under the care of the medical team. As his arm swelling continued, he was then thought to possibly have a vascular issue and was given a therapeutic dose of low molecular weight heparin. He was otherwise pain free throughout and had no evidence of neurological compromise. A subsequent orthopaedic review raised the concern of compartment syndrome, and this, alongside a clinically evolving picture, resulted in a diagnosis of developing compartment syndrome being made. The patient underwent subsequent surgical management with dual incision fasciotomies and definitive wound management with split skin graft under the care of plastic surgery. This is a rare presentation of a masked developing compartment syndrome. This case highlights the importance of considering atypical presentations of compartment syndrome in high risk patients, and the importance of early specialist orthopaedic referral, particularly in those who do not present with typical features in their history and examination. We summarise the lessons learned in this case which would have enabled earlier identification of this limb-threatening diagnosis and could have improved further definitive outcome.

BACKGROUND

Compartment syndrome occurs when pressures within a fascial compartment exceed that of capillary level, leading to restriction of blood flow, associated muscle ischaemia and eventual necrosis. This is a surgical emergency that requires prompt diagnosis and intervention, typically in the form of fasciotomies, to reduce intracompartmental pressures.¹

The classical presentation of compartment syndrome involves the rapid development of severe pain beyond that expected with the injury sustained, with or without associated evidence of neurovascular impairment. The constellation of symptoms

and signs are collectively colloquially known as 'the 6 Ps': pain out of proportion, poikilothermia, paraesthesia, paralysis, pulselessness and pallor.^{1 2}

However, it is important to recognise the subjective nature of compartment syndrome; there are rare descriptions of atypical presentations of compartment syndrome in the literature. In addition to this, adjuncts to clinical diagnosis, such as measuring intracompartmental pressures, may be useful in those with diagnostic uncertainty.

By recognising those at increased risk of atypical features of compartment syndrome through history of presentation and medical history, a more focused use of monitoring can be tailored. This can be effective in diagnosing compartment syndrome promptly and thus instigating appropriate and timely surgical intervention.

This case documents the development of a masked developing compartment syndrome with both atypical symptoms and signs, initially treated as a cellulitis of the forearm.

CASE PRESENTATION

A 35-year-old, known intravenous drug user, was found collapsed and unresponsive at home by his flatmate. Significant medical history included opioid dependence syndrome and previous drug-induced psychosis. The cause and duration of lie were unknown, but a dirty needle was found at the scene by the paramedic crew. Naloxone was administered by his flatmate, and the presumed diagnosis of drug overdose was made as the acute cause of presentation to the emergency department.

On admission, he was drowsy but responsive to voice. The patient denied any drug or alcohol use and reported no other new symptoms. He was tachycardic (115 beats/min) and pyrexia (38.2°C), with an otherwise normal cardiorespiratory and abdominal examination. All limbs proved to be neurologically intact, and he was pain free. Of note, he had a palpable hard firm swelling in the anterior right forearm, which was poorly circumscribed, thought to be chronic induration and soft tissue fibrosis due to repeated injection at this site. Compartments were soft on palpation.

Given the clinical examination and supporting investigations performed, the patient was presumed to have sepsis secondary to forearm cellulitis and was promptly admitted under the acute medical team for intravenous flucloxacillin (1 g four times per day).

Despite continued intravenous antibiotic therapy and fluid resuscitation over the following 24 hours,

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To cite: Ng J, Makaram N, Mitchell S. *BMJ Case Rep* 2019;**12**:e231116. doi:10.1136/bcr-2019-231116

no clinical improvement was observed. The affected forearm remained erythematous, and the swelling remained unchanged. The patient did not report any increase in forearm pain and remained neurologically intact; an orthopaedic review was therefore subsequently sought given the lack of clinical improvement. The medical team at this point administered a therapeutic dose of low molecular weight heparin while awaiting a Doppler ultrasound of the arm, in case of a vascular cause for the swelling.

Upon orthopaedic review, the patient was lucid and still reported no pain. He was found to have a cool right hand, with a capillary refill time of 4 seconds. The radial pulse was palpable, and the right forearm was globally tense. There were no noted injection markings and no pain was elicited on passive flexion. Power was 4/5 in all myotomes at the upper limb, and sensation was intact.

Given the clinical suspicion, a compartment pressure monitor was inserted.

INVESTIGATIONS

On initial assessment, full blood count revealed a mildly raised white cell count ($11.2 \times 10^9/L$) and raised C-reactive protein (13.9 mg/L)

Serum biochemistry revealed a raised D-dimer (1937 $\mu g/mL$) and creatine kinase was elevated at 6942 U/L. The patient was hyperkalaemic with a potassium of 6 mEq/L. Arterial blood gas showed a partially compensated respiratory acidosis with a lactate of 1.3 mmol/L and renal function was normal.

As the D-dimer was raised, the potential diagnosis of venous thromboembolism was considered. However, the patient did not undergo any formal imaging on initial assessment, as it was deemed that this was most likely raised secondary to an infective cause. A therapeutic dose of dalteparin was however administered in the event of thromboembolism and a Doppler ultrasound scan was ultimately carried out after secondary assessment following admission to the medical ward; this proved to be normal.

Upon compartment pressure monitor insertion, a significant delta pressure of 29 mm Hg was observed (<30 mm Hg—high likelihood of compartment syndrome), with an absolute

compartment pressure of 46 mm Hg (normal 0–8 mm Hg). The patient was otherwise clinically stable and remained pain free.

One hour later, compartment pressures were repeated; the delta pressure had increased to 69 mm Hg. Repeat clinical examination revealed the development of median nerve paraesthesia with a repeat creatine kinase of 19 000 U/L.

DIFFERENTIAL DIAGNOSIS

The likely differential diagnosis in this case is illustrated in [table 1](#), with supportive and deterring factors detailed. The confirmatory investigations with their associated findings are additionally illustrated.

TREATMENT

Given the evolving clinical signs and the initial investigative findings of high compartment pressures, with an increasing creatine kinase, the patient was urgently taken to theatre for forearm fasciotomies.

Fasciotomies of the right forearm and concurrent carpal tunnel decompression were performed within 28 hours of presentation. This comprised of a dual incision forearm fasciotomy, with a volar incision used to decompress the superficial and deep anterior compartment of the forearm and a dorsal incision to decompress the posterior compartment of the forearm.

The muscles of the anterior compartment were noted to be bulging and dusk on direct visualisation but appeared to regain their normal flushed appearance following fasciotomy, as can be observed in [figure 1](#). In the dorsal compartment, the extensor digitorum communis and extensor pollicis longus appeared pale and bulging ([figure 1](#)). The carpal tunnel was decompressed through a separate incision. Peripheral circulation was deemed to be intact.

The fasciotomy wounds were left open and dressed. Postoperatively, over the following 8–16 hours, there appeared to be a marked improvement in the patient's symptoms, with normal sensation returning. His clinical observations also normalised, with heart rate returning to normal and fever resolving. In addition to this, postoperative creatine kinase was found to have

Table 1 Differential diagnosis for acute atraumatic forearm swelling and erythema

Differential diagnosis	Supportive factors in history and examination	Deterring factors in history and examination	Investigative findings
Venous thromboembolism	<ul style="list-style-type: none"> ▶ Raised D-dimer ▶ Risk factor of long lie ▶ Swollen and tense forearm 	<ul style="list-style-type: none"> ▶ No evidence of pitting oedema ▶ No localised tenderness along deep venous system ▶ No evidence of collateral superficial veins 	<ul style="list-style-type: none"> ▶ D-dimer -1937 $\mu g/L$ ▶ Ultrasound of the arm showed no evidence of venous thromboembolism
Cellulitis	<ul style="list-style-type: none"> ▶ Associated systemic upset—tachycardia and fever ▶ Potential inoculation injury secondary to intravenous drug injection ▶ Erythema, swollen and tense forearm 	<ul style="list-style-type: none"> ▶ Raised intra-compartment pressures ▶ Evolving neurovascular compromise ▶ Inflammatory markers only marginally raised 	<ul style="list-style-type: none"> ▶ White cell count—$11.2 \times 10^9/L$ ▶ C-reactive protein—13.9 mg/L
Compartment syndrome	<ul style="list-style-type: none"> ▶ Risk factors of lie of unknown duration ▶ Clinically evolving picture despite medical management ▶ Increasing creatine kinase 	<ul style="list-style-type: none"> ▶ No associated pain 	<ul style="list-style-type: none"> ▶ Initial delta pressure—29 mm Hg ▶ Absolute compartment pressure—46 mm Hg ▶ Increasing creatine kinase from 6942 to 19 000 u/L
Haematoma	<ul style="list-style-type: none"> ▶ Known intravenous drug use with associated trauma ▶ Poorly circumscribed swelling in anterior forearm ▶ One-off dose of therapeutic dalteparin 	<ul style="list-style-type: none"> ▶ Initial haemoglobin within normal range 	<ul style="list-style-type: none"> ▶ No clotting sample taken at time of admission ▶ Haemoglobin 140.0 g/L ▶ Compartment pressure raised

Figure 1 Image taken following initial washout postfasciotomy, demonstrating appearance of superficial anterior compartment and posterior.

significantly decreased to 12 035 u/L, and white cell count had normalised to 7.2×10^9 /L.

OUTCOME AND FOLLOW-UP

The patient returned to theatre on a further two occasions, for a second and third look at 48 and 96 hours following the initial procedure. At these intervals, minimal debridement was required as there was not noted to be any further significant muscle necrosis. On the third procedure at 96 hours postoperatively, the fasciotomy wounds were closed at their proximal and distal limits, leaving approximately 7 cm of defect which was unable to be closed primarily, as observed in [figure 2](#). The final creatine kinase postoperatively was found to have significantly improved, returning a value of 3924 u/L.

Five days after his initial fasciotomy, this patient was transferred to a specialist plastic surgery unit to undergo split skin graft, which was performed with no complications. At latest clinic follow-up, his wounds had healed well, and he had regained all neurological function with no deficit in power.

Figure 2 Image of dorsal and volar fasciotomy wound following second washout and attempted primary closure limited due to swelling.

DISCUSSION

Compartment syndrome is a surgical emergency and is caused by increased pressures within compartments bound by fascia; it is typically associated with long bone fractures, burns and prolonged compression.³ The classical signs are cited as intractable pain out of proportion to the injury sustained, poikilothermia, paraesthesia, paralysis, pulselessness and pallor.² Of these, pain is universally considered the most important, reliable and early indicator of a developing compartment syndrome. Emergency fasciotomy is key in relieving pressure within the compartment, as irreversible loss in function can start to develop between 4 and 12 hours postinjury.^{1 4} It is often a condition associated with high-energy injuries and fractures, classically of lower limb diaphyseal fractures.

This case illustrates the rare presentation of a masked developing compartment syndrome, without any significant pain, without associated fracture, in a relatively uncommon anatomical location. As such, it is important to recognise that although pain is an important clinical predictor of compartment syndrome, the absence of pain does not exclude the presence of compartment syndrome. This is particularly the case in patients with an atypical presentation.

Acute compartment syndrome results in decreased tissue perfusion, vascular collapse and cell anoxia.^{5 6} The resultant ischaemia leads to cell necrosis, triggering increased extravasation of inflammatory exudate and thereby increasing compartment pressures further until vast tissue necrosis results.

It is therefore important to recognise those at particular risk of developing compartment syndrome; research has shown that the positive predictive value of the 6Ps associated with compartment syndrome is only 11%–15%.⁷

Typically, those at risk of compartment syndrome can be categorised as follows:

- ▶ Trauma with associated long bone fractures, classically of the lower limb.
- ▶ Upper and lower limb 'crush' trauma without fractures.
- ▶ Prolonged compression.
- ▶ Vascular injury—particularly reperfusion related injuries.
- ▶ Burns.
- ▶ Non-traumatic causes.

Traumatic injuries with closed long bone fractures are most closely correlated with the risk of development of compartment syndrome, with high-energy closed tibial diaphyseal fractures being most commonly cited. Less commonly, prolonged compression associated with long periods of time lying on limbs of the body, as in this presentation, are found to be the precipitant. Evidence suggests that the long period of muscle compression results in ischaemia and the rebound reperfusion following relief of compression triggers a cascading inflammatory reaction; this precipitates further microvascular and tissue necrosis.⁸

In these described scenarios, the cardinal feature that aids diagnosis remains the presence of pain disproportionate to the injury sustained.

More rarely, the presentation of compartment syndrome in the absence of significant pain is protean in its features, and the methodology to ascertain which patients are most at risk remains unknown. The literature is scarce in the presentation of such occult atypical compartment syndromes.

Badhe *et al*⁹ presented four cases of painless compartment syndromes, all following acute traumatic high-energy fractures and all in the lower limb. They were all competent sensate young men, as our patient was. In contrast, our patient unusually

developed compartment syndrome in the upper limb, after a low trauma mechanism.

Lee *et al*¹⁰ reported a retrospective case series of five patients who met inclusion criteria that deemed their symptoms to be consistent with compartment syndrome, with no significant presentation of pain. Two cases had lower limb involvement and three had upper arm involvement following supracondylar fractures. Two patients had significantly limited communication due to autism, and all developed compartment syndrome following acute traumatic injury. None here were observed in adults or were present in the forearm, and all followed obvious traumatic injury, unlike our case.

Tang *et al*¹¹ reported a case of a 62-year-old woman who underwent right total knee arthroplasty for osteoarthritis. The patient remained on continuous epidural anaesthesia in the postoperative period for pain control, and shortly after the procedure, her

prosthesis dislocated and required urgent reduction. She then developed compartment syndrome diagnosed from motor weakness, swelling and increased compartment pressures.

In the UK, the 'gold standard' guideline for diagnosis and management of suspected compartment syndrome has been published by the British Orthopaedic Association Standards for Trauma (BOAST).¹²

BOAST guidelines highlight the importance of accurate documentation in those at high risk of compartment syndrome; a clear history must be taken to ascertain the true nature of the injury and regular monitoring must be carried out to look for a developing clinical picture.

The described management algorithm for compartment syndrome as per BOAST guidelines is summarised in figure 3.

On immediate assessment following the suspicion of compartment syndrome, it is important to release any circumferential

Figure 3 Algorithm for diagnosis and management of patient with suspected compartment syndrome.

dressings and moderately elevate the limb. Given the dependent nature of delta pressure on diastolic blood pressure, it is also important to maintain normal blood pressure and regularly monitor for an evolving clinical picture.

A complete fasciotomy is the therapeutic intervention required in those with a clinical diagnosis of compartment syndrome and involves full-length incisions that release all compartments in the affected anatomical region.¹³

Timely diagnosis and subsequent fasciotomy reduces risk of progressive compartment syndrome producing significant muscle necrosis and necessitating salvage amputation. The rate of limb loss, as a consequence of compartment syndrome, is reported as up to 13%.¹⁴

Despite being vital in preventing irreversible muscle damage, fasciotomies are not without risk; these procedures carry morbidity and adults can suffer from short-term complications such as wound infection and need for blood transfusions. In this particular case, the patient required two further looks and washouts after his initial fasciotomy.¹⁵ Patients undergoing fasciotomies should return to theatre 48 hours postoperatively for further washout and debridement of necrotic tissues.

BOAST also recommends the employment of intracompartmental pressure monitoring. This can be of particular use in those with diagnostic uncertainty.¹²

Intracompartmental pressure monitoring can differ between departments, ranging from a regular 18-gauge needle with a similar set up to an arterial line to a Stryker needle (side portal needle) and a slit catheter. What is important to note is that there is a variation in compartment pressures between different injuries and significant variability within normal compartments.¹⁶

A relationship has been shown between the distance from the fracture site to the intracompartmental pressure.¹⁷ Mubarak *et al* showing that while 89% of the highest compartmental pressure readings were at the fracture site, 5% of pressures measured 5 cm distal to the fracture were of the highest values.¹⁸

The normal value of intracompartmental pressure is between 0 and 8 mm Hg in adults¹⁹; various in vivo studies have shown that at 30 mm Hg, the fascia is maximally stretched and adequate blood flow to the muscle tissue can no longer be maintained.^{18 20 21}

The concept of delta pressure for the diagnosis of compartment syndrome was first introduced by Whitesides *et al*.²² They observed that ischaemia begins when pressure rises to within 10–30 mm Hg of the diastolic blood pressure.

McQueen and Court-Brown further postulated that absolute compartment pressure is an unreliable indication of the need for fasciotomy. Indeed, a growing body of evidence has gathered over the last two decades to support this view; tissue viability is dependent on adequate perfusion and the blood flow within the microcirculation is dependent on both tissue and venous pressures.²³

The delta pressure is the difference in pressure between the diastolic blood pressure and the intracompartmental pressure; this emphasises the importance of maintaining normotension in patients with suspected compartment syndrome. Those with a lower diastolic blood pressure are therefore at increased risk of developing compartment syndrome. McQueen and Court-Brown concluded that in a study of 116 patients, 30 mm Hg was the threshold delta pressure that led to 'no missed cases, unnecessary fasciotomies or significant complications', and this is widely accepted as the threshold diagnostic delta pressure for compartment syndrome.²³

It remains that the clinical picture and constellation of clinical signs are the most important determining factors regarding the

decision to proceed to fasciotomy. Further investigation should not preclude or delay timely fasciotomy.

We recognise that not every centre has the facility to measure intracompartmental pressures however intracompartmental pressure monitoring can be a useful diagnostic adjunct, where possible, in those cases presenting with atypical symptoms or where there is diagnostic uncertainty. This particular presentation included a myriad of ambiguous symptoms and signs initially, and in particular, the absence of pain and presence of fever and erythema confounded initial diagnosis. Thus, the use of compartment pressure monitoring in this scenario was useful and deemed worthwhile in supporting our diagnosis.

It is important to also note the features in this particular patient which increased the likelihood of compartment syndrome. This was a case of an intravenous drug user who had likely injected prior to symptoms arising. Additionally, his history was consistent with that of a patient who had sustained a long lie likely from being obtunded secondary to effects of intravenous drug use. Thus, this patient was likely to be a poor historian and his ability to perceive pain would have perhaps been impaired. These key features of the patient's history should raise the clinician's suspicion for compartment syndrome on initial presentation.

An important lesson that should be taken away from this case is the importance of being vigilant for compartment syndrome as a differential in such patients who are likely to be under the influence of narcotics, who have a history of an unknown duration of lie and who present with a swollen limb. It is also particularly important in these patients to recognise that pain perception can be significantly altered due to narcotic use. In such atypical cases, where the classic diagnostic symptom of pain outwith proportion is unreliable, the clinician must be on the alert and look for secondary signs of compartment syndrome, including soft tissue swelling and tense fascial compartments. This constellation of symptoms and signs in such at risk individuals should prompt urgent orthopaedic referral.

A further explanation that could account for the lack of pain initially, but a worsening set of measured compartment pressures, is the high likelihood that this was a developing case of compartment syndrome post-presentation in concurrence with an initial cellulitis. This would account for the progressively abnormal pressures and an evolving picture which grew more consistent with a classical compartment syndrome following initial presentation, resulting in fasciotomies.

Wounds postfasciotomy must be carefully monitored to ensure control of swelling, minimisation of skin retraction and appropriate recovery time for injured tissues. Given these potential complications, closure is typically not possible at initial setting, and many patients undergo multiple procedures to achieve gradual closure.²⁴ Indeed, in our patient, two subsequent procedures were required for further irrigation and debridements to be performed with subsequent assessment of wound management definitively. Interestingly, Bengezi *et al* reported a series of 12 fasciotomy wounds, in which simple elevation of the limbs allowed for successful primary closure within 5 days.²⁵ This highlights the significant role tissue oedema can play on the outcome of wounds postfasciotomy.

Fasciotomy wounds may heal via secondary intention, split thickness skin grafting, vacuum-assisted closure or dermal apposition.²⁶ The method chosen is dependent on the individual case and how well the patient may cope with the associated healing times/complications and potential need for further procedures. Infection rate postfasciotomy has been reported to be up to 38%,¹⁴ hence, the requirement for thorough debridement and multiple episodes of irrigation are invaluable. Given the risk

Table 2 A summary of definitive wound management options following fasciotomy for compartment syndrome

Method of healing	Description	Advantages	Disadvantages
Secondary intention	Skin contraction is used to heal the wound	<ul style="list-style-type: none"> ► Suitable for those who can manage wounds without supervision ► Reduction in additional scars ► Reduction in hospital stay 	<ul style="list-style-type: none"> ► Healing can take up to 3–4 months ► Normalisation of scar contour can take up to 4 years ► Not suitable for larger wounds
Split thickness skin grafting	This uses a skin graft from a donor site	<ul style="list-style-type: none"> ► Technique is tension free ► Suitable for large wounds that require closing 	<ul style="list-style-type: none"> ► Requires another operation ► Associated infection risk with another operation ► Extension of stay in hospital ► Chance of graft failure ► Cosmetic and functional problems including donor site morbidity
Vacuum assisted closure	This uses negative pressure to use elastic properties of skin	<ul style="list-style-type: none"> ► Reduces oedema ► Promotes blood flow ► Increases tissue granulation rate 	<ul style="list-style-type: none"> ► Requires regular dressing changes ► Expensive ► May actually fail to provide skin edge approximation
Dermal apposition	A load is placed on the margin to gradually increase skin length and gradually allows for delayed primary closure	<ul style="list-style-type: none"> ► Shoelace technique does not require additional equipment ► Reduced time for closure ► Less expensive 	<ul style="list-style-type: none"> ► Cannot be started until oedema settles ► Constant tension created from devices can lead to recurrence of compartment syndrome and skin necrosis ► Repeated procedures required

of infection described, it is important to select the most appropriate definitive wound management method to provide optimal coverage and closure.

A summary of definitive wound management strategies post-fasciotomy is illustrated in [table 2](#).

Given the importance of wound management and achieving definitive wound coverage postfasciotomy, a multidisciplinary approach is crucial, with a need of early involvement of plastic surgery. The availability of early clinical photography intraoperatively following initial fasciotomy, debridement and throughout further procedures is also important, as this can prove invaluable in aiding later management.

As with many patients postfasciotomy, our patient underwent split thickness skin grafting; this was most suitable given the large area required for wound management and the social history of the patient.

Learning points

- Compartment syndrome is a surgical emergency that requires rapid diagnosis and treatment.
- Not all patients with compartment syndrome present with the classical '6Ps': pain out of proportion, poikilothermia, paraesthesia, paralysis, pulselessness and pallor.
- Although pain is a key diagnostic aid, the absence of pain does not exclude the presence of compartment syndrome in an otherwise suspicious clinical context.
- The clinician should be vigilant of patients who have a history of narcotic use or substance misuse where pain perception may be altered, and history may be poor.
- Clinicians should be aware of developing compartment syndromes, and the absence of symptoms and signs on initial presentation does not preclude the subsequent development of compartment syndrome in the clinical course.
- There must be a low threshold for compartment monitoring to enable timely and effective surgical intervention (fasciotomy) in those high-risk patients to prevent irreversible disability and even amputation.

CONCLUSION

Compartment syndrome is a surgical emergency that requires rapid diagnosis and surgical intervention. While the key diagnostic aid is pain, both disproportionate to the injury sustained and on passive stretch of the affected compartment, it is important to recognise that not all patients present with such classical symptoms. The absence of pain does not therefore exclude compartment syndrome in patients who may be at risk and who have a suggestive history. Occult asymptomatic compartment syndrome may occur in non-competent, distracted or insensate patients. This case illustrates a rare occult developing compartment syndrome in a sensate patient with the absence of pain.

In patients at risk, a high index of clinical suspicion is required, and there must be a low threshold for using diagnostic adjuncts such as continuous compartment pressure monitoring, frequent documented clinical examination and blood creatine kinase monitoring.

Clinicians should be aware of rare atypical presentations of occult developing compartment syndrome, and individuals who may be at risk should be highlighted, with focused clinical assessments performed regularly. Diagnostic adjuncts should be employed at a much lower threshold in these patients to facilitate early diagnosis and timely management with emergency surgical fasciotomy. Following diagnosis, an early multidisciplinary approach with involvement of plastic surgery and clinical photography is paramount in aiding further definitive management following fasciotomy.

Contributors JN, NM and SM were involved in data collection, literature review, drafting and finalising the manuscript.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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