Deep Tissue Injury with Acute Myonecrosis: A Case Report and Review of the Literature

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Authors' Contributions

All authors contributed equally both to the writing and editing of this manuscript.

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Disclosures

The authors have no disclosures, financial or otherwise.

Consent

Written informed consent has been obtained by the patient for submission of this manuscript.

Ethical Statement

This research was conducted in accordance with the ethical standards set by our institutional review board. All participants provided informed consent before their inclusion in the study, ensuring that they were fully aware of the study's purpose, procedures, risks, and benefits.

The confidentiality and anonymity of participants were strictly maintained throughout the research process. Data collected was securely stored and only accessible to the research team.

Human & Animal Rights

No human or animal experiment was conducted as part of this review study.

Conflict of Interest

The authors and their immediate family members have no conflict of interest to declare. No financial support was received in the process of this study.

ABSTRACT

Pressure-related injuries are a well-known complication of immobility and most frequently present as pressure sores. However, rarer presentations of such injuries can pose diagnostic challenges, delaying appropriate intervention. This paper presents an atypical case of deep tissue pressure injury manifesting as acute myonecrosis following a period of immobility. Initially, inflammatory changes in the subcutaneous and deeper tissues of the thigh were interpreted as infection, though this eventually evolved into deep tissue necrosis on follow-up imaging. Acute myonecrosis is an uncommon form of deep tissue injury, with a different initial clinical course and imaging features to classical pressure injuries. This case underscores the importance of maintaining a high index of suspicion for such atypical presentations of pressure injuries, potentially reducing morbidity and mortality associated with these challenging wounds.

CASE REPORT

BACKGROUND

Pressure-related injuries can present in atypically, complicating their diagnosis and management. This case report helps to delineate the unique imaging characteristics and clinical features of this condition, and subsequently aid in early recognition and intervention, reducing the risk of subsequent complications.

CASE REPORT

A 58-year-old lady was admitted to the hospital after she was found lying immobile on the floor for two days. The patient claimed to have suffered a fall and could not get up. No precipitating cause for the fall could be found after a detailed history from the patient and her family. Initial physical examination was mostly unyielding save for a fever. No significant neurological deficit was elicited, and there was no point tenderness to suggest a fracture. No skin abnormalities were noted either. First-line laboratory investigations in the emergency department showed normal inflammatory markers but raised creatinine kinase of 2182 units/L. Screening radiographs were negative for fractures.

Radiological Findings

A subsequent computed tomography (CT) scan of the abdomen and pelvis showed no localising source of sepsis but detected inflammatory changes in the imaged proximal right thigh in the form of soft tissue swelling and oedema (Figure 1).

A magnetic resonance imaging (MRI) scan of the right thigh, performed two days post-admission, showed regions of rim-enhancement in the right posterolateral proximal and mid thigh, with mild surrounding oedema in the subcutaneous tissue and muscle (Figure 2-4). Based on the imaging findings, the patient was referred to orthopaedic surgery and started on intravenous antibiotics for presumed infective cellulitis and myositis. However, skin induration and erythema, which were previously absent, developed over the next few days (Figure 5), followed shortly by frank skin ulceration (Figure 6).

A follow-up MRI of the right thigh, requested by the primary team one month later, indicated progression of fat necrosis at the affected regions, with deep subcutaneous ulceration and exposure of the underlying musculature. Post-contrast sequence revealed muscle rim-enhancement extending down to the periosteal surface and showing positive "stipple sign" consistent with acute myonecrosis (Figure 7-9).

The patient was eventually diagnosed with deep tissue pressure injury at both sites and was managed conservatively via intravenous antibiotics and regular wound care. A repeat MRI one month later showed resolution of the rim enhancement in the thigh muscles and overall stability of other superficial tissue changes (Figure 10,11). Her wounds remained stable throughout her 2-month hospital stay, before being discharged to step-down care.

DISCUSSION

Etiology & Pathology

Cutaneous pressure ulcers are a well-documented complication of immobility. The National Pressure Ulcer Advisory Panel (NPUAP) has developed a widely-used six-stage grading system of such pressure injuries [1,2] (Figure 12) – while stages I-IV reflect the usual "top-down" pathophysiology of a pressure ulcer (and stage V an unstageable pressure ulcer), the latest revision incorporates a sixth stage, *Deep Tissue Injury* (DTI), that reflects a slightly different pathological process that could be in keeping with the changes manifested in this patient.

DTI represents a less common form of pressure injury wherein the changes occur in the deeper tissues rather than the skin itself. Traditionally, this initially manifests as a darker discolouration (e.g. "purple" or ecchymotic) [3]. Acute myonecrosis is a less commonly detailed subset of DTI, primarily involving skeletal muscle [4].

Based on pathological examination of rat models, muscle injury has been postulated to be the result of direct deformation and ischaemia that usually occurs in areas of compression between two rigid surfaces [5,6]. The former is a more rapid process that results in direct myocyte destruction, while the latter is a slower process (~2-4h) associated with blood vessel and lymphatic occlusion, followed by an accumulation of cellular waste products and a decrease in the local pH. Both these factors are exacerbated by impaired lymphatic drainage and reperfusion injury. In our case, both wounds occurred over bony prominences; stress distribution modelling [7] also shows peak principle compression forces in the muscles adjacent to the bone, particularly with additional rigid objects in the region which cause significantly increased stress forces and resultant injury. As such, the more superior gluteal wound may have been more extensive due to increased compression forces against a pointed rigid surface (gluteal tuberosity), with a resultant increased weight/stress on the gluteal region compared to the mid-thigh during the patient's immobilization period.

Clinical & Imaging Findings

We present an atypical case of deep tissue pressure injury that manifested as acute myonecrosis, with the mechanism of injury in this case being prolonged immobilisation in a fixed position. The initial impression was that of infection (cellulitis, myositis) due to the patient's active sepsis and inflammatory changes in the right thigh which was initially presumed to be the source of this sepsis. The initial impression of a rim-enhancing abscess was unlikely due to the lack of fluid-like T2w hyperintense signal. Only later did the subsequent MRI studies demonstrate the presence of muscle necrosis. The skin changes manifested only a few days later.

Two categories of acute myonecrosis have been described on imaging [8,9]. Type 1 myonecrosis manifests as muscle oedema with hyperintense T2 weighted signal and contrast enhancement,

which is suggestive of reversible injury. On the other hand, type 2 myonecrosis, which is more pertinent to our case, classically presents as heterogeneous T1 and T2 weighted changes with rim enhancement with the “stipple sign” [9,10], which is seen as foci of punctate/linear enhancement within the area of rim enhancement. This rim-enhancement has been theorised to be related to central necrotic tissue with surrounding hyperaemia in the hyperacute setting [8] and may persist in the acute phase due to the formation of granulation tissue. There may be tissue loss as well if there was nonviable tissue. Similar findings have also been demonstrated in rat models, with increased T2 weighted signal in the affected musculature (postcontrast changes are not as well-delineated due to various technical factors [5,6]. In the chronic phase, there should be eventual resolution of the rim enhancement due to healing. Our patient did not initially exhibit any superficial changes, with the classical imaging signs of the rim enhancement only becoming evident much later.

Calcific myonecrosis is a rarely reported complication of this condition (not observed in our patient) [11], described as a progressively enlarging mass that appears on imaging as plaque-like calcifications occurring in the region of prior trauma/myonecrosis. This complication has rather characteristic imaging findings, though the index of suspicion for the primary traumatic incident needs to be high.

Differential diagnosis

Acute myonecrosis, which is usually trauma or pressure-related, is characterized by inflammatory changes (e.g. soft tissue fat stranding and T2 weighted hyperintensity), and may show the characteristic "stipple sign" on MRI.

A pertinent differential diagnosis on imaging is that of infection (particularly in our case), which may show a broad spectrum of imaging findings, ranging from soft tissue edema, as seen in cellulitis, to rim-enhancing collections.

Of course, classical pressure injuries may be a differential in later stages of disease, though changes would conversely extend from the skin downward (unlike acute myonecrosis, which originates in the muscles), and typically begin with skin changes.

Diabetic myonecrosis shares imaging similarities with traumatic or pressure-related myonecrosis but differs in its cause, being a spontaneous event linked to underlying diabetes.

Ecchymotic skin changes, which are a presenting sign of classical deep tissue pressure injury, has a broad variety of differential diagnoses, including arterial/venous insufficiency, post-traumatic changes like a Morel-Lavallee lesion and necrotising fasciitis [12].

MANAGEMENT

Management of such deep tissue injuries aligns with the standard management of all pressure injuries [3], including repositioning and offloading. Patients should be turned regularly where possible, with the use of soft dressings and air mattresses to complement these nursing measures. Once skin ulceration has occurred, routine surgical and nonsurgical measures should be implemented to aid wound healing.

SUMMARY

This case underscores the spectrum and temporal progression of MR imaging findings in DTI, of which acute myonecrosis is a relatively less frequently encountered subset. We present an acute case of pressure injury, with initial pathology manifesting as inflammatory changes in the deep tissues of the thigh that progressed on to the classical rim enhancement seen in type 2 myonecrosis. Both clinicians and radiologists should be cognizant of this important subset of pressure injuries, maintaining a heightened suspicion for DTI and acute myonecrosis in the presence of a history of prolonged immobility. Early recognition is crucial to enable the initiation of preventative measures and early management.

TEACHING POINT

Acute myonecrosis is a relatively rare form of pressure-related injury with an atypical presentation, wherein subcutaneous/deeper pressure-related inflammatory changes with a lack of cutaneous changes can confound the diagnostic picture on imaging. A high index of suspicion should be maintained to reduce the morbidity and mortality associated with this challenging diagnosis.

QUESTIONS

1. Which of these findings is incorrectly matched with its NPUAP classification?

A. Stage 1: Intact skin with a localized area of non-blanchable erythema

B. Stage 2: Partial-thickness loss of skin with exposed dermis

C. Stage 3: Full-thickness skin loss with exposed adipose tissue

D. Stage 4: Full-thickness skin loss with exposed muscle but with a large eschar on the wound base (applies)

E. Deep Tissue Injury: Darker discolouration of the skin or ecchymosis with intact skin

Correct Answer: D. Full-thickness skin loss with exposed muscle but with a large eschar on the wound base

Stage 4 involves full-thickness skin and tissue loss, with exposure of bone, tendon, or muscle. The key distinction for Stage 4 is the exposure of these deeper structures, which indicates severe injury. The description above is incomplete for staging as there is a large eschar in the wound base which precludes further assessment of the deeper structures, which would instead class it as a stage 5 injury.

[Fig 12… Grade IV ulcer – Full thickness tissue loss, extending to the muscle/tendon/bones]

2. Which of the following is a classical sign of acute myonecrosis on MRI?

A. "Halo" sign

B. "Stipple" sign (applies)

C. "Sunburst" pattern
D. "Soap bubble" pattern
E. "Apple core" pattern
Correct Answer: B. "Stipple" sign

The “stipple” sign is identified as a classical sign of acute myonecrosis on MRI and is characterized by foci of punctate or linear enhancement within the area of rim enhancement, which is indicative of type 2 acute myonecrosis. The “halo” sign can indicate a variety of conditions depending on the imaging modality and body part, with perivascular inflammation in giant cell arteritis being one of them. A “sunburst” pattern of periosteal reaction is a malignant pattern seen in bone tumours. A “soap bubble" pattern is seen in various lesions, including bony lesions. The “apple core” pattern is seen in stenosing bowel tumours.

[Type 2 myonecrosis classically presents as heterogeneous T1 and T2 weighted changes with rim enhancement with the “stipple sign” [5], which is seen as foci of punctate/linear enhancement within the area of rim enhancement.]

3. Which of the following aetiologies is associated with acute myonecrosis?
A. Necrosis of skin and adipose tissue
B. Overproduction of myoglobin
C. Impaired calcium homeostasis
D. Overproduction of myoglobin
E. Myocyte destruction, impaired lymphatic drainage and reperfusion injury (applies)
Correct Answer: E. Myocyte destruction, impaired lymphatic drainage and reperfusion injury

The correct answer, E, encapsulates the primary pathophysiological mechanisms, of which the two main contributory factors are direct deformation leading to myocyte destruction, and ischemia resulting from blood vessel and lymphatic occlusion. This ischemic process is further exacerbated by an accumulation of cellular waste products and a decrease in the local pH, primarily due to impaired lymphatic drainage. Additionally, upon reperfusion, the injured tissue may suffer further damage—a phenomenon known as reperfusion injury.

The options A, B, C, and D, such as necrosis of skin and adipose tissue, overproduction of myoglobin, and impaired calcium homeostasis, while relevant to various pathological conditions, are not specifically relevant to this condition.

[Based on pathological examination of rat models, muscle injury has been postulated to be the result of direct deformation and ischaemia that usually occurs in areas of compression between two rigid surfaces (6,7). The former is a more rapid process that results in direct myocyte destruction, …]

4. In the context of acute myonecrosis, what does the process of rim enhancement on MRI best indicate?
A. The presence of a benign tumor
B. The boundary between healthy and necrotic muscle tissue (applies)
C. The accumulation of adipose tissue
D. The regeneration of muscle fibers
E. The presence of a vascular malformation
Correct Answer: B. The boundary between healthy and necrotic muscle tissue

Rim enhancement occurs due contrast accumulation at the interface between viable and non-viable tissue, thereby highlighting the extent of tissue damage. The contrast agent does not penetrate the necrotic tissue due to the lack of blood flow, but it does accumulate in the vascularized tissue surrounding the necrosis, creating a distinct "rim" of enhancement on the imaging. This feature aids in the delineation of the affected area, providing critical information on the extent and severity of myonecrosis.

The options A, C, D, and E suggest other potential causes or features that might be observed on MRI for various conditions but do not specifically pertain to the characteristic imaging findings of acute myonecrosis.

[This rim-enhancement has been theorised to be related to central necrotic tissue with surrounding hyperaemia in the hyperacute setting [6], and may persist in the acute phase due to the formation of granulation tissue.]

5. Which of the following is not a possible complication of acute myonecrosis?
A. Skin breakdown
B. Osteomyelitis
C. Calcific myonecrosis
D. Compartment syndrome
E. Malignant transformation (applies)
Correct answer: E. Malignant transformation

Acute myonecrosis is a potentially serious condition that can lead to several complications if not promptly and adequately addressed. These include:

A. Skin breakdown - This can occur as a consequence of underlying tissue damage and necrosis, leading to compromised skin integrity.

B. Osteomyelitis - Infection can spread from necrotic muscle tissue to adjacent bones, leading to osteomyelitis, an infection of the bone.

C. Calcific myonecrosis - This is a late complication of muscle necrosis where calcification occurs within the necrotic muscle, often many years after the initial injury or insult.

D. Compartment syndrome - This is an acute condition that can arise from the swelling and increased pressure within a muscle compartment, which can itself be a consequence of acute myonecrosis.

The option E does not align with the natural progression and potential severity of this condition.

[Calcific myonecrosis is a rarely reported complication of this condition…, described as a progressively enlarging mass that appears on imaging as plaque-like calcifications occurring in the region of prior trauma/myonecrosis.]

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FIGURES

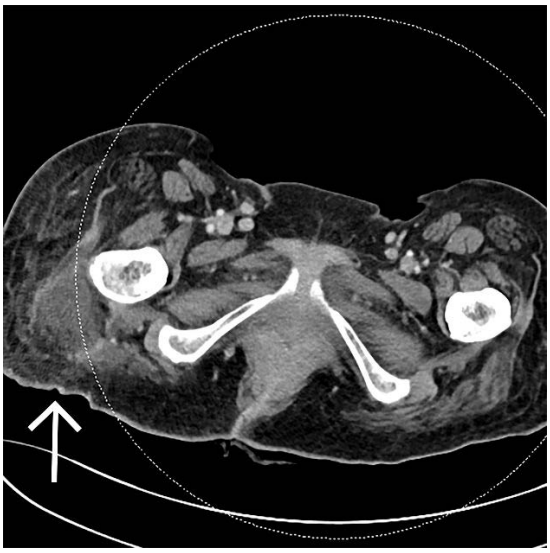

Figure 1: CT of the abdomen and pelvis of our 58-year-old female patient on admission, which shows partially imaged inflammatory changes of the right thigh (white arrow).

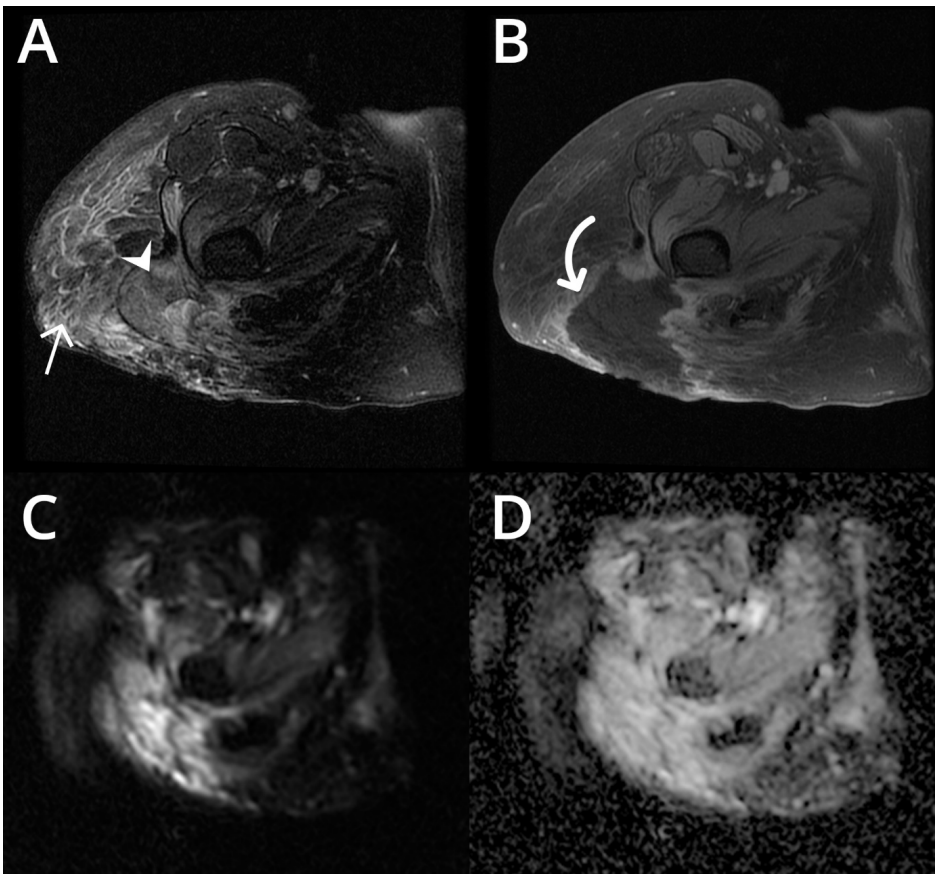

Figure 2: Initial MRI of the right hip performed two days after admission – (a) T2-fat saturated, (b) post-contrast T1-fat saturated, (c) DWI and (d) ADC sections, show subcutaneous oedema over the gluteal region (straight arrow), with oedema of the muscles (gluteus maximus, arrowhead) and a geographic region of central non-enhancement with surrounding rim enhancement in the gluteus maximus muscle (curved arrow). Changes extend to the underlying femur/gluteal tuberosity. There is no significant restricted diffusion to suggest purulent contents.

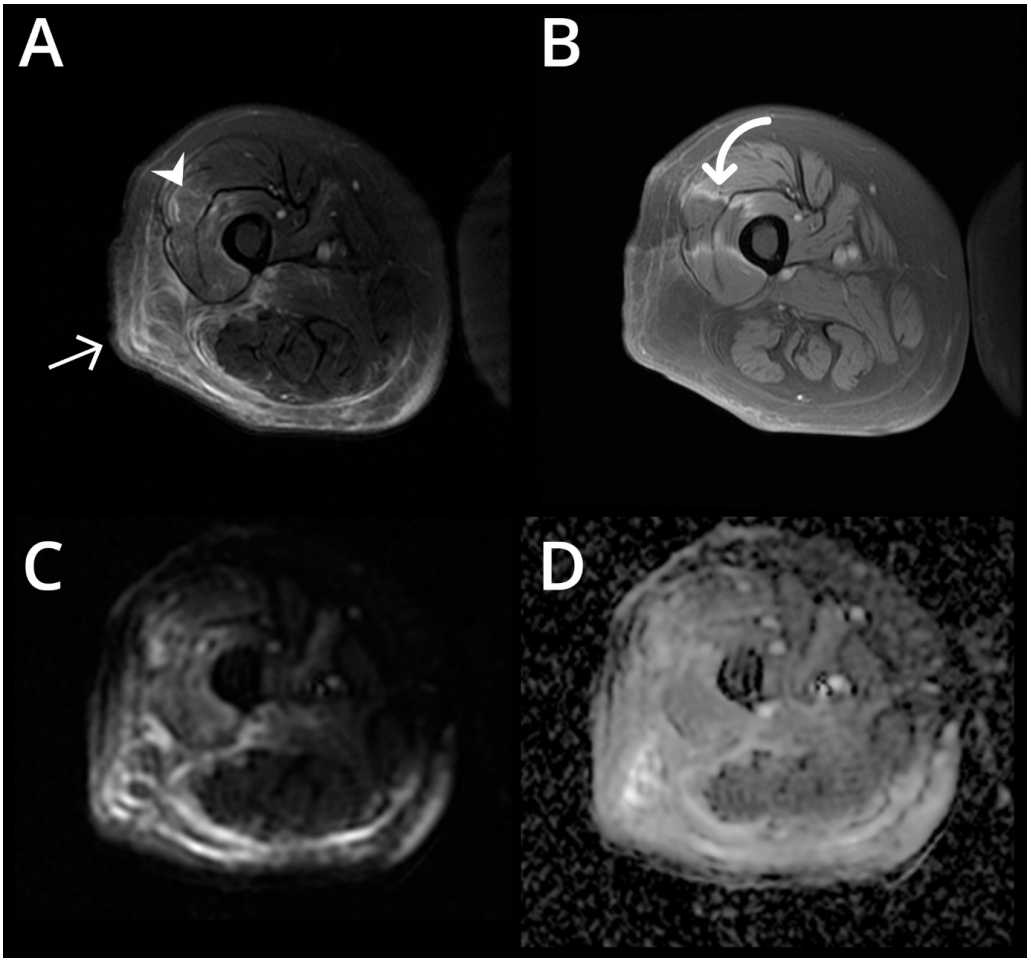

Figure 3: Initial MRI of the right thigh performed two days after admission – (a) T2-fat saturated, (b) post-contrast T1-fat saturated, (c) DWI and (d) ADC sections, showing marked subcutaneous oedema (arrow) and mild muscular oedema (arrowhead, of the rectus femoris muscle) of the lateral right thigh, suggestive of cellulitis and myositis respectively. Post-contrast administration, there was rim enhancement of a region laterally (curved arrow), but no corresponding T2w hyperintensity or diffusion restriction to suggest a collection. There is no evidence of osteomyelitis.

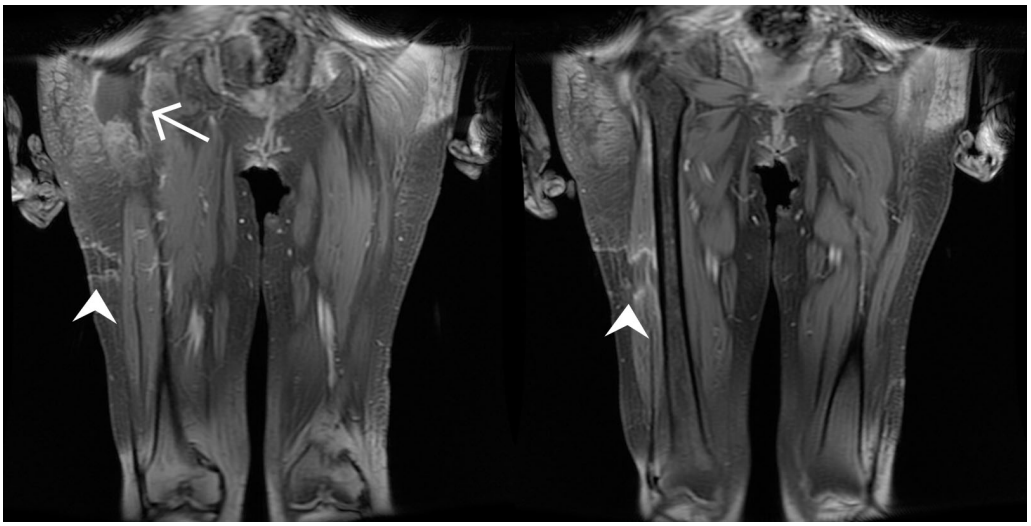

Figure 4: Initial MRI of the right thigh (coronal sections), showing the relative positions of the proximal (straight arrow) and mid-thigh (arrowhead) lesions.

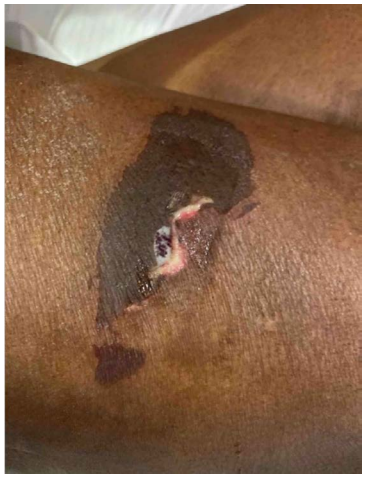

Figure 5: Induration, discoloration and slight erythema with largely intact skin (save for a small wound) at the mid-thigh on day 3 of admission.

Figure 6: Early ulcerative changes in the same area on day 10 of admission.

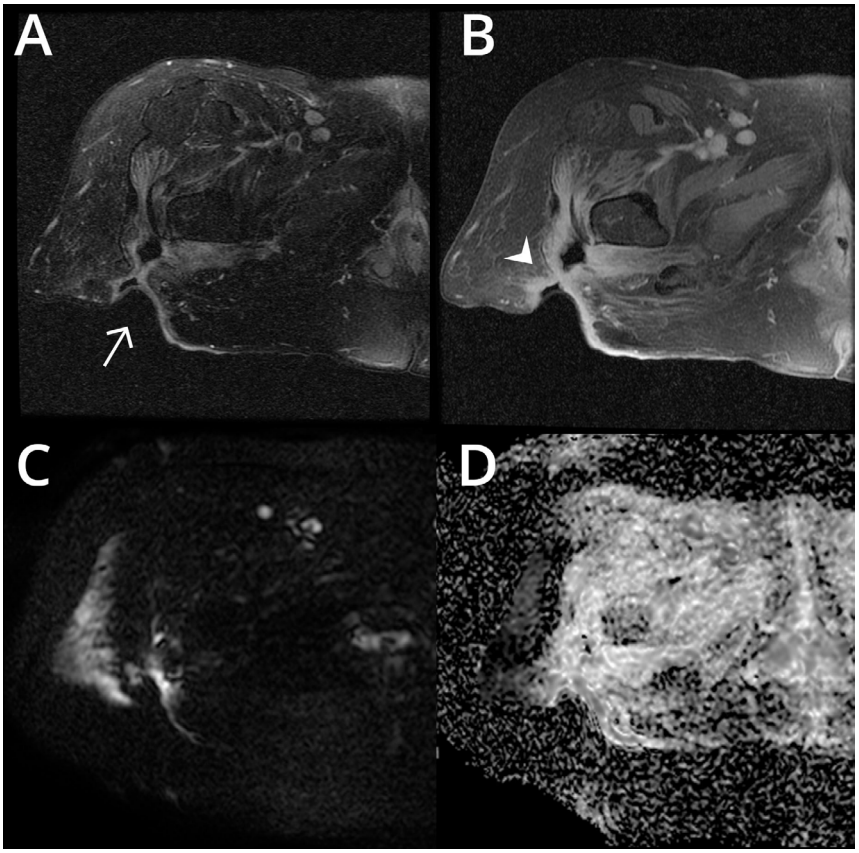

Figure 7: Follow-up MRI of the right thigh performed one month after admission – (a) T2-fat saturated, (b) post-contrast T1-fat saturated, (c) DWI and (d) ADC sections, wherein the previous area of central non-enhancement and restricted diffusion now shows, an ulcer (straight arrow) with extensive, deep soft tissue necrosis and tissue loss. There is peripheral enhancement around the wound edges (arrowhead) which is likely due to granulation tissue. No collection is detected.

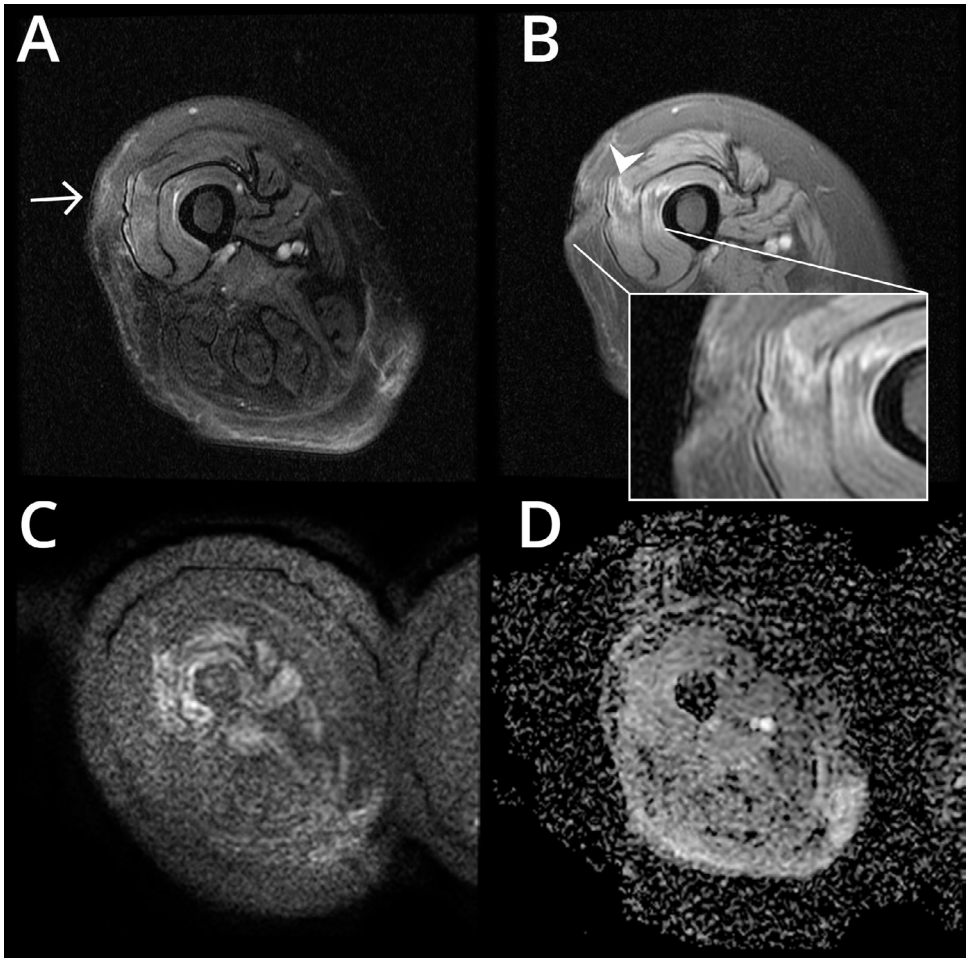

Figure 8: Follow-up MRI of the right thigh performed one month after admission – (a) T2-fat saturated, (b) post-contrast T1-fat saturated, (c) DWI and (d) ADC sections show that there is now ulceration at the site of the previously noted injury (arrow), with exposure of the underlying musculature. There was persistent rim-enhancement of the muscle, with presence of the stipple sign (zoomed up area, arrowheads). No restricted diffusion is currently noted.

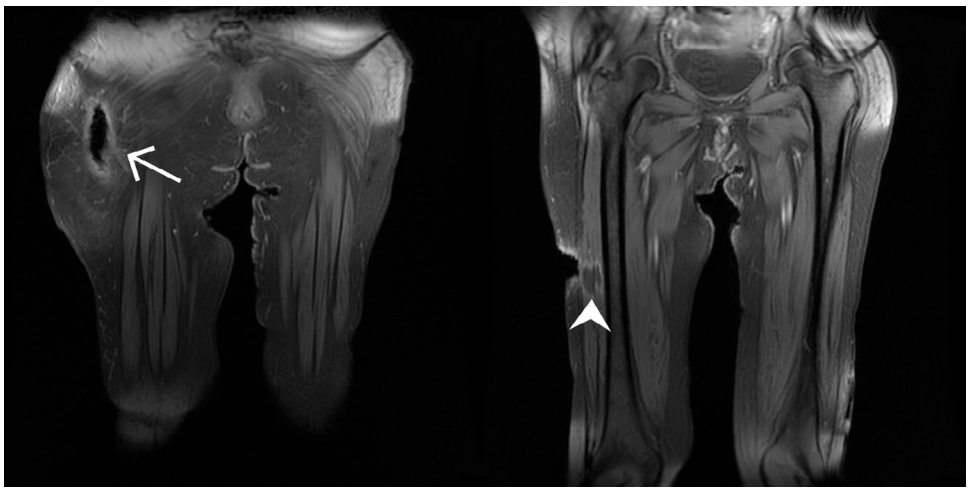

Figure 9: Coronal post-contrast MRI of the right femur, showing temporal progression of the proximal thigh (straight arrow) and mid-thigh (arrowhead) lesions when compared to the findings in Fig 2c.

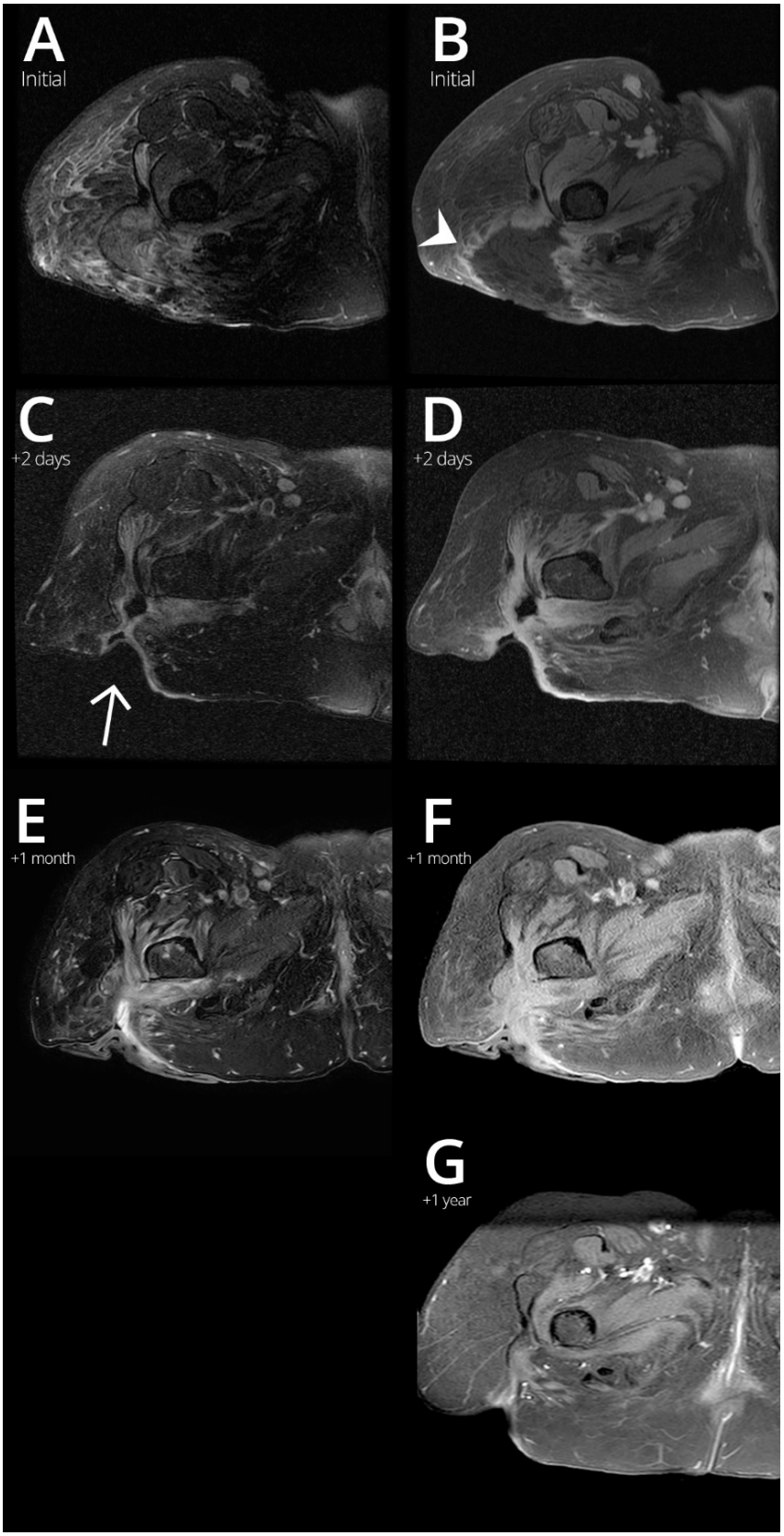

Figure 10: Temporal illustration of the progress of the proximal femoral myonecrosis on MRI – (a) T2 fat-saturated & (b) T1 post-contrast MRI performed two days after admission, (c) T2 fat-saturated & (d) T1 post-contrast MRI performed a month after admission, (e) T2 fat-saturated & (f) T1 post-contrast MRI performed two months after admission, and (g) T1 post-contrast MRI pelvis performed a year after admission. These demonstrate the initial lack of superficial changes but large area of rim enhancement (arrowhead), with subsequent soft tissue ulceration that healed progressively.

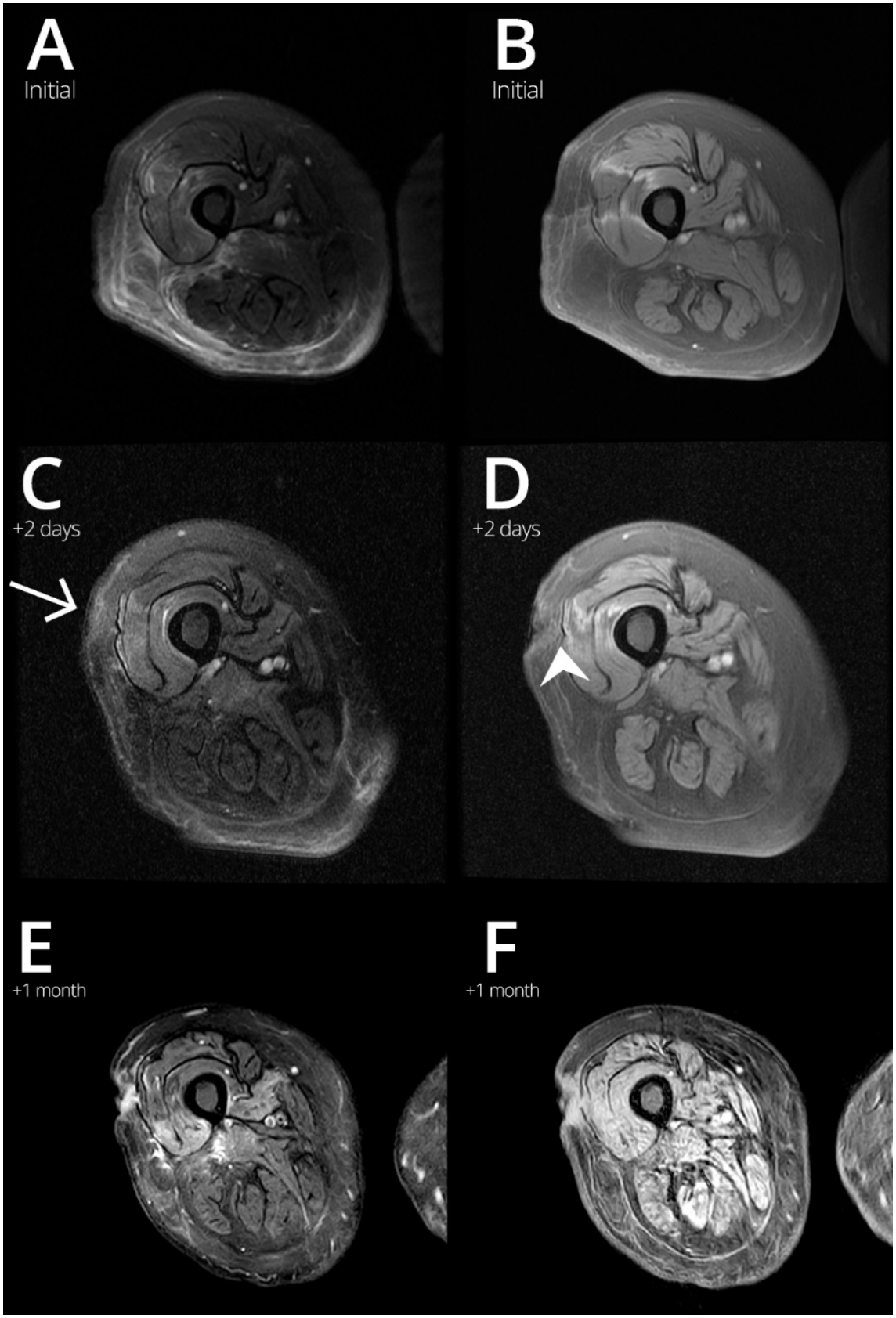

Figure 11: Temporal illustration of the progress of the mid-femoral myonecrosis on MRI – (a) T2 fat-saturated & (b) T1 post-contrast MRI performed two days after admission, (c) T2 fat-saturated & (d) T1 post-contrast MRI performed a month after admission, and (e) T2 fat-saturated & (f) T1 post-contrast MRI performed two months after admission. Again, these demonstrate the lack of superficial changes initially but eventually showed an area of rim enhancement with the stipple sign (arrowhead, see also figure 5b).

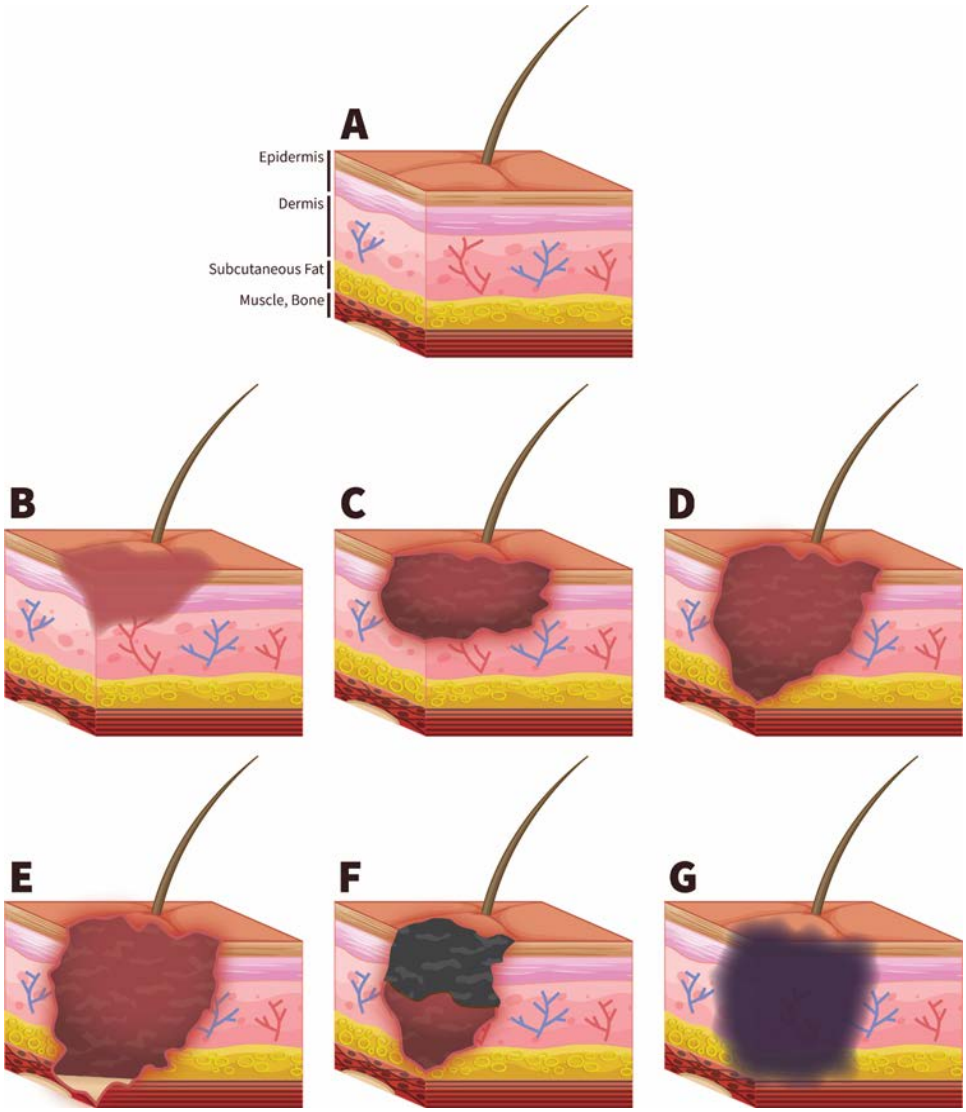

Figure 12: Stages of pressure ulcers. A. Normal skin; B. Grade I ulcer – Nonblanchable erythema; C. Grade II ulcer – Partial thickness skin loss, extending to the subcutaneous tissues; D. Grade III ulcer – Full thickness skin loss, extending to the subcutaneous adipose tissue; E. Grade IV ulcer – Full thickness tissue loss, extending to the muscle/tendon/bones; F. Unstageable ulcer – Base is covered by slough/necrotic tissue; G. Deep tissue injury
Source: Adapted from the NPUAP guidelines [2]

SUMMARY TABLE

Aetiology	Myocyte (as opposed to cutaneous) deformation and ischaemia, exacerbated by impaired lymphatic drainage (from the pressure) and reperfusion injury
Incidence	The incidence of this specific condition is not well-elucidated, but the incidence of general pressure injuries was reported as 4.7% of all inpatients [13]
Gender Ratio	Risk factors are more widely studied, particularly given the widely-acknowledged aetiology of the parent process; a prior study showed a slight male predominance (M:F 3:2) [9]
Age Predilection	Generally elderly; the average age of patients with general pressure injuries was reported as 70.4 years [13]
Risk factors	Acute myonecrosis is a specific subset of pressure injury, as such the same risk factors apply, including immobility and hospitalisation
Treatment	Nonsurgical (e.g. wound dressings) and surgical (e.g. debridement)
Prognosis	The prognosis of traumatic myonecrosis itself is not as well-studied, but the prognosis of deep tissue pressure injuries in general can proceed rapidly to life or limb threatening complications if not promptly treated [3]
Findings on Imaging	Rim enhancement of affected musculature, with the “stipple sign”, or foci of punctate/linear enhancement within this area of rim enhancement Increased T2 signal

DIFFERENTIAL TABLE

Differential Diagnosis	Imaging Features
Acute myonecrosis (traumatic/pressure-related)	Inflammatory changes on CT/MR (fat stranding, T2-hyperintensity), with the classical “stipple sign” on MR.
Infection	Wide variety of imaging findings, ranging from soft tissue oedema (e.g. in cellulitis) to rim-enhancing collections. Oedema can present on CT as fat stranding, and on MR as increased T2 signal. Collections can also present as foci of rim-enhancement in the soft tissues.
Classical pressure injury	T2-hyperintensity and a cutaneous wound, though the changes would extend from the skin down as opposed to originating in the muscles.
Diabetic myonecrosis	This has a similar imaging appearance to traumatic/pressure-related myonecrosis, though it has a different aetiology (spontaneous myonecrosis related to underlying diabetes)

KEYWORDS

Myonecrosis; Pressure Ulcer; Magnetic Resonance Imaging; Deep Tissue Pressure Injury; Stipple Sign

ABBREVIATIONS

CT = COMPUTED TOMOGRAPHY
MRI = MAGNETIC RESONANCE IMAGING
NPUAP = NATIONAL PRESSURE ULCER ADVISORY PANEL
DTI = DEEP TISSUE INJURY

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