

## Acute Skin Necrosis with Compartment Syndrome in the Upper Extremity

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### Abstract

We present the case of a 43-year-old female patient who developed acute blistering and necrosis of the skin of the left upper limb following a routine cannulation. The case highlights the importance of early recognition of compartment syndrome in the non-trauma setting and also introduces the angiosome concept to explain why extensive degloving inevitably results in skin necrosis.

**Keywords:** Compartment syndrome; Angiosome; Split thickness skin graft

### Introduction

Acute limb compartment syndrome (LCS) is a condition in which raised pressure within a closed fascial space reduces capillary perfusion below a level necessary for tissue viability [1]. This condition has been recognised since 1881 when Volkmann first described the contracture that is a common sequel [2].

LCS is a surgical emergency and immediate decompression of the affected compartments is the treatment of choice. It has been shown that an intracompartmental pressure (ICP) of 30 mmHg or more for 6-8 hours can cause irreversible changes to the fascial contents [3]. Early diagnosis is therefore essential and clinicians should have a low index of suspicion of LCS in the acutely painful limb, even in the absence of a traumatic injury.

The following case report highlights the diagnostic challenges and potential complications associated with LCS, and also introduces the concept of skin angiosomes [4,5]. This concept delineates the human body into three-dimensional blocks of tissue fed by specific arterial and venous sources named angiosomes (Figure 1). Adjacent angiosomes are connected by a vast compensatory collateral web, or choke vessels [6]. In cases of extensive degloving of multiple skin angiosomes from the underlying fascia, the compensatory vessel network is insufficient to maintain the viability of the skin.

### Case Report

A 43-year-old female patient with end-stage renal failure was admitted for a myocardial perfusion scan as part of a routine pre-transplant work-up.

She had a medical history of rheumatoid arthritis, systemic lupus erythematosus, septic arthritis, multinodular goitre, osteoporosis, and hypertension.

Her drug history included prednisolone and thyroxine, but she was on no anticoagulants.

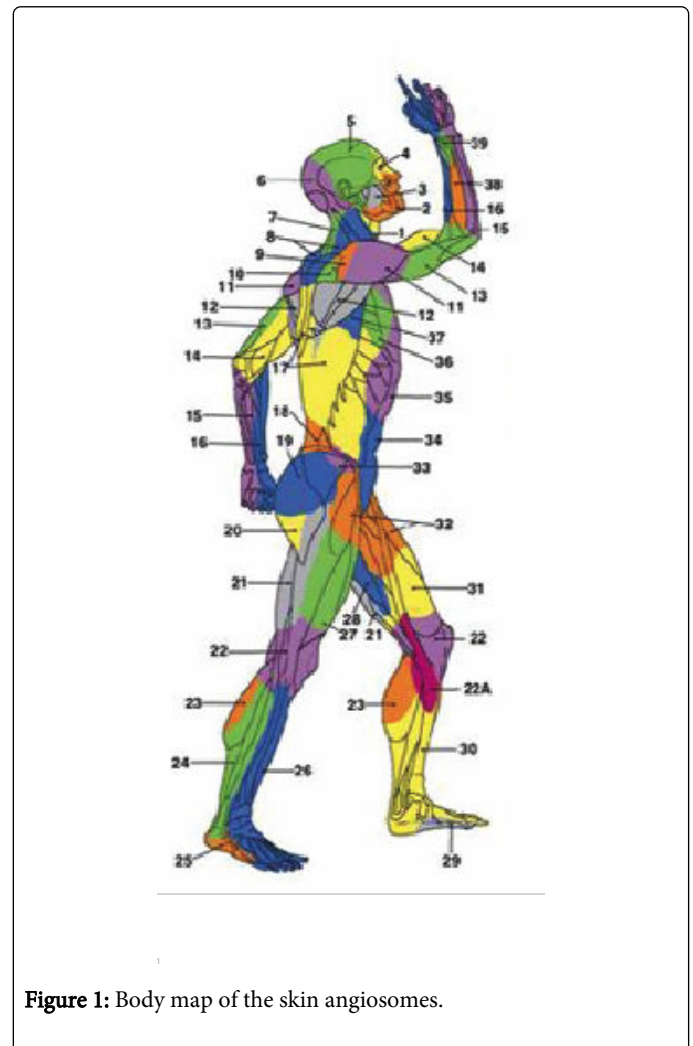


Figure 1: Body map of the skin angiosomes.

A technetium-based medium was injected into her left antecubital fossa and she immediately developed localised pain and swelling.

Within hours, her entire left upper limb was swollen and tense, with large areas of blistering and skin necrosis (Figure 2).



**Figure 2:** Blistering and skin necrosis of the left upper limb.

Initial investigations revealed a lactic acidosis, with a pH of 7.27, and haemoglobin of 4.9g/dL. The patient rapidly decompensated to a peri-arrest state.

The patient was seen by Plastic Surgery who advised urgent exploration of the limb in theatre. Compartment pressure readings of >45mmHg showed acute LCS in all compartments of the arm and forearm. Full-length medial and lateral escharotomy incisions revealed extensive, circumferential, suprafascial haematoma (Figure 3).



**Figure 3:** Extensive, circumferential haematoma after escharotomy.

The haematoma was evacuated and the non-viable skin was excised. The anaemia and acidosis were corrected and the patient rapidly stabilised. Repeat compartment pressures were normal.

The blister fluid grew bacillus cereus, streptococcus mitis, and granulicatella adiacens. Tissue samples showed abundant dermal and subcutaneous haemorrhage.

6 days after the initial debridement, the left upper limb defect was reconstructed with split thickness skin grafts harvested from both thighs. 18 days after the first operation, the patient was discharged with stable wounds.

At 1-month follow-up, the skin grafts were fully healed (Figure 4) and the patient had made a complete recovery from the acute episode.

## Discussion

LCS is a limb and life-threatening condition. In the trauma setting, the diagnosis is easier to make since it is a well-recognised complication of long bone injuries, especially those of the tibia. There are, however, a variety of other causes and the treating clinician should

have a low index of suspicion for LCS in any patient with an acutely painful limb.



**Figure 4:** Healed skin grafts at 1 month. The hand position is postural and does not represent contracture.

In this case, the patient presented a clinical picture that was unique to the treating clinicians. Following a routine cannulation for a myocardial perfusion scan, she rapidly developed extensive blistering of the entire left upper limb. Whilst there was significant uncertainty over the pathophysiology, it was clear that immediate intervention was necessary, as the patient was rapidly progressing towards death.

Measurement of the compartment pressures was invaluable since it confirmed that surgical exploration was required, and explained the progressive lactic acidosis from tissue ischaemia. In theatre, it became clear that the patient had bled profusely from the cannulation site and the subsequent haematoma had degloved the overlying skin from the fascia of the arm and forearm.

Whilst the skin was still attached, significant areas of it were already necrotic. The angiosome concept tells us that a localised area of degloving will not result in skin necrosis because of compensatory circulation from adjacent angiosomes via the choke vessels. When multiple angiosomes are affected, however, this compensatory supply is insufficient to maintain the viability of the skin, as was evidenced in this case.

To our knowledge, an injury of this severity, from a routine cannulation, has not previously been described in the literature.

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