Case Report

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Angiosomal Vascular Occlusions, Deep-Tissue Pressure Injuries, and Competing Theories: A Case Report

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ABSTRACT

Compression of the soft tissue between a support surface and a bony prominence has long been the accepted primary mechanism of pressure injury (PrI) formation, with the belief that said compression leads to capillary occlusion, ischemia, and tissue necrosis. This explanation presupposes an "outside-in" pathophysiologic process of tissue damage originating at the local capillary level. Despite advances in prevention protocols, there remains a stubbornly consistent incidence of severe PrIs including deep-tissue injuries, the latter usually evolving into stage 4 PrIs with exposed bone or tendon. This article presents just such a perioperative case with the aim of providing further evidence that these more severe PrIs may result from ischemic insults of a named vessel within specific vascular territories (labeled as angiosomes). Pressure is indeed a factor in the formation of severe PrIs, but these authors postulate that the occlusion occurred at the level of a named artery proximal to the lesion. This vascular event was likely attributable to low mean arterial pressure. The authors suggest that the terminology proposed three decades ago to call both deep-tissue injuries and stage 4 PrIs "vascular occlusion pressure injuries" should be the topic of further research and expert consensus.

KEYWORDS: angiosome, deep-tissue injury, pressure injury, pressure ulcer, vascular occlusion, wound care

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INTRODUCTION

The US National Pressure Injury Advisory Panel (NPIAP) is a professional organization committed to preventing pressure injuries (PrIs). A PrI can present as intact (unbroken) skin or an open wound, and the etiology is considered to be the result of localized intense and prolonged pressure alone or in combination with shear forces, usually over a bony prominence, causing damage to the skin and/or underlying soft tissue.[1] Compression of the soft tissue between a support surface and a bony prominence has long been the accepted primary mechanism for PrI formation, with the belief that said compression leads to capillary occlusion, ischemia, and tissue necrosis.[2,3] This explanation presupposes an "outside-in" pathophysiologic process of tissue damage originating at the local capillary level.

The NPIAP has recently revised its classification/staging system for PrIs based on observable tissue damage.[1] Although numbered, the stages do not represent a linear progression; rather, the stages refer only to the depth of tissue affected based on visual inspection. This descriptive system is not based on biopsy findings, angiography, or diagnostic imaging; therefore, the proposed pathophysiologic mechanisms remain speculative.[4]

The risk factors associated with severe PrIs, particularly in critically ill patients, are well established and include hypotension, anemia, hypoalbuminemia, hypoxia, use of vasopressors, known vascular disease, reduced cardiac output, and hemodynamic instability.[5–13] It has been suggested that some PrIs may be "immune to prevention."[14] According to the NPIAP, a PrI is rendered "unavoidable" only in certain clinical situations such as hemodynamic instability, shock, impaired tissue oxygenation and perfusion, and whenever lifesaving interventions take precedence.[15] Because *unavoidability* is a payment-related term that refers to whether appropriate preventive interventions were in place, it is more accurate to say that some severe PrIs are medically "unpreventable."

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This clinical quandary raises the question of whether current prevention protocols are directed at the right mechanism/risk factors in certain situations. For example, providers know that the risk factors listed above really matter; however, there are no scientifically defined parameters to guide treatment decisions. Current prevention strategies are to offload tissue to reduce local compression, which is largely accomplished with repositioning efforts,[15,16] and to use support surfaces that assist with pressure redistribution. Although clearly beneficial for preventing less severe PrIs, there is limited evidence supporting the use of pressure redistribution surfaces and frequent repositioning to prevent severe PrIs.[17–19] Using the logic that all PrIs are caused by local tissue compression, the most severe PrIs (eg, stage 4) are considered the result of poor care delivery, on the assumption that tissue injury should have been readily apparent at an earlier stage.

The high cost and prevalence of hospital-acquired PrIs, in combination with the policy of denying federal or state reimbursement for the additional inpatient costs incurred in treating them, have led to a nationwide focus on preventive measures.[20,21] These include but are not limited to frequent repositioning/turning of patients, improved support surfaces, optimal nutrition, early mobilization, prevention of skin maceration, elevation of heels when supine, and use of foam dressings to protect against friction and shearing.[1,21] When implemented consistently and as a "bundle" of interventions, the result has been a demonstrable decrease in facility-acquired stage 1 and 2 PrIs.[17,22]

Despite these advances, there remains a stubbornly consistent incidence of severe PrIs, including deep-tissue injuries (DTIs), the latter usually evolving into stage 4 PrIs with exposed bone or tendon.[23,24] In fact, studies show an increase in intraoperative PrIs that has thus far defied explanation.[25,26] Also unexplained is the fact that severe PrIs exhibit a highly predictable and repeatable anatomic pattern of tissue necrosis—frequently not directly over a bony prominence—a puzzling fact given the NPIAP definition of a PrI.

In 2019, *Advances in Wound Care* published a case report of a healthy 22-year-old who developed intraoperative PrIs (stage 1) on areas not exposed to external pressure.[4] This case study suggests that the mechanism for PrI formation in some cases may be that of a transient occlusion of the blood vessel supplying the affected vascular territory (angiosome). An angiosome is a three-dimensional block of skin, subcutaneous tissue, fascia, and muscle supplied by a named (source) artery and vein. The entire human body comprises more than 40 such angiosomes. Given the absence of direct pressure, the intraoperative event described had to have occurred proximal to the area of visible PrI and been caused by an occlusion of the vessel(s) supplying those tissues, resulting in an ischemia-reperfusion injury to the downstream tissues that recovered before tissue necrosis occurred.[4] This explanation presupposes an "inside-out" pathophysiologic process for some PrIs of hypoperfusion to the deep tissues in a vascular territory. (Table 1 summarizes variables related to hypoperfusion.)

A similar mechanism may account for stage 4 PrIs, except the vascular event is not recoverable and thereby results in necrosis of the tissues supplied by the affected vessel(s) (Figure 1). Although iconoclastic, this idea is not new. In the past, there was general acknowledgment that the most severe PrIs developed in the deep tissues and evolved outward.[27,28] In contrast, less severe PrIs, which involve only the top layers of the skin, likely result from the outside-in processes affected by moisture, friction, and shear. When the current staging system aggregated superficial and deep lesions into one descriptive classification, the concept of the outside-in etiology became the dominant pathophysiologic mechanism for all PrIs.[27,29,30] However, more than three decades ago, visionary clinician Roberta Abruzzese suggested that severe decubitus ulcers be termed "vascular occlusion ulcers."[31] It seems likely that she observed their relationship to vascular anatomy.

Presented in this article is a recent perioperative case with a DTI that evolved into a stage 4 PrI. The aim here is to provide further evidence that these more severe PrIs may result from ischemic insults of a named vessel within specific angiosomes. In fact, these authors believe that the visual correlation between named vascular territories and the anatomic distribution of most stage 4 PrIs is nothing short of astounding. Through the lens of this case report, the authors will review the risk factors associated with severe PrIs to explain alternative PrI etiology

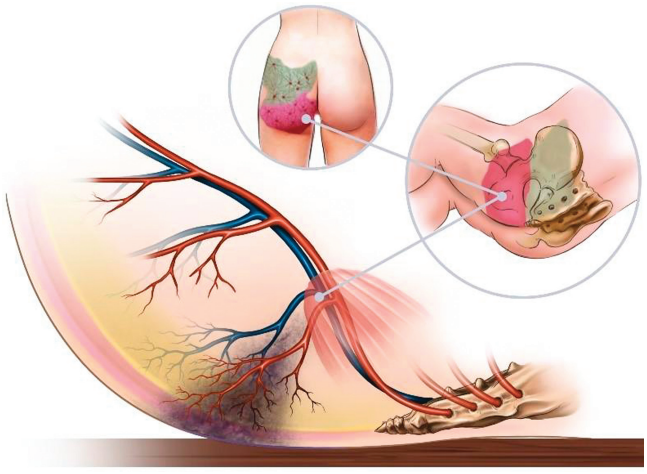
Table 1. FACTORS ASSOCIATED WITH HYPOPERFUSION AND SEVERE PRESSURE INJURY POTENTIAL

Measure	PrI Risk Mechanism
Vital signs: BP (systolic, diastolic, and mean arterial pressure), peripheral capillary oxygen saturation (Spo_2)	BP and Spo_2 are essential for delivery of oxygen-rich blood to tissues and reduce associated risk for PrI formation[9,12-16,29]
Vasopressor infusion: norepinephrine, vasopressin, dopamine, phenylephrine, epinephrine	Vasopressors stimulate α- and/or α- and β-receptors to varying degrees resulting in peripheral vasoconstriction with associated risk for PrI[9,12]
Oxygen-carrying capacity: hemoglobin, hematocrit	Anemia is associated with PrIs, likely because of diminished oxygen-carrying capacity[60]
Serum albumin	Low albumin reflects decreased colloid osmotic pressure, increasing PrI risk[8,10]

Abbreviation: PrI, pressure injury.

Figure 1. "INSIDE-OUT" ETIOLOGY

This illustration demonstrates the way in which severe pressure injuries can develop in soft tissues that are not directly over a bony prominence. Note that either the arterial or venous supply to the buttock (eg, superior gluteal artery or vein, etc) could be compressed by the weight of the body at the choke point where they perforate the muscle fascia. This would result in an "inside-out" vascular ischemic event that would involve the entire angiosome and extend from the muscle to the skin.



that could positively impact future prevention strategies. The patient provided his written informed consent for the educational use of his photographs, and the reporting of this case adheres to the Declaration of Helsinki.

CASE REPORT

A 46-year-old obese (body mass index, 42 kg/m^2) White man with a history of coronary artery disease and left ventricular dysfunction was admitted for an elective coronary artery bypass graft. He was a former smoker with a history of myocardial infarction, hypertension, hyperlipidemia, obstructive sleep apnea (continuous positive airway pressure therapy), ischemic cardiomyopathy, and anemia of chronic disease.

His surgery under general anesthesia lasted 5 hours 20 minutes, during which time he was supine with his arms tucked and secured to arm boards and his pressure points padded. The patient's mean arterial pressure (MAP) was less than 60 mm Hg for much of the procedure, but did not drop below 50 mm Hg.

Immediately following surgery, he was transferred to the ICU where 30 minutes after arrival he became hypotensive (78/44 mm Hg). Intravenous norepinephrine was initiated and continued for approximately 12 hours. He remained hypotensive for 6 hours, with a systolic BP less than 80 mm Hg and a diastolic BP less than 60 mm Hg. Of note, his serum albumin at admission was only 2.8 mg/dL. The following day, his arterial pressure ranged from 59/46 to 71/53 mm Hg. He had acute blood-loss anemia but did not have cardiopulmonary decompensation.

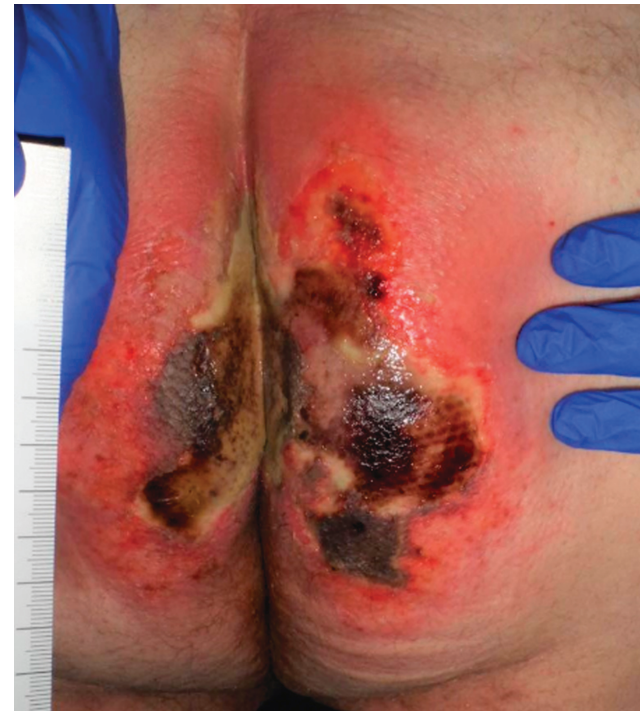
On postoperative day (POD) 2, nursing assessment noted purple discoloration over his buttock cheeks. On POD 4, a large unstageable PrI of the buttocks was documented. He was discharged on POD 5 and referred to the outpatient wound center.

During the initial outpatient evaluation on POD 7, the patient had a very large DTI (11.3 × 8.9 cm) involving both buttock cheeks on either side of the gluteal cleft (Figure 2), as well as breakdown of his sternal incision warranting immediate readmission. He first underwent operative debridement of the sternum and placement of negative-pressure wound therapy (NPWT) to the chest. On POD 16, the wound management team and plastic surgery decided to surgically debride the extensive and evolving DTI to reduce the risk of bacterial colonization of necrotic tissue (inevitable when the wound is near the rectum) that could in turn impact the open sternal wound.

During DTI debridement, bilateral hematomas were noted beneath the gluteus maximus muscles. The largest surface area of buttock tissue loss was near the skin, with the zone of tissue loss narrowing dramatically at the deepest aspect of the wound in the same tissue plane as the hematomas (cone-shaped). There was also a rim of violaceus but viable tissue around the necrotic zone (Figure 3). After aggressive operative debridement of these tunnels, the parasacral ligaments became visible.

Figure 2. LARGE DEEP-TISSUE INJURY

A very large deep tissue injury measuring 11.3 × 8.9 × 0.1 cm and surrounded by erythema was observed on both buttock cheeks of the case patient on postoperative day 5.



The now stage 4 PrI underwent NPWT with instillation for 13 weeks until periwound skin irritation and wound odor necessitated its discontinuation, at which time the wound measured 5.4 × 3.2 × 1.5 cm (Figure 4). Tunneling remained visible superior to the gluteal cleft, proximal to the majority of tissue loss, and adjacent to the sacroiliac ligaments. The deeper of the two tunnels (on the left) measured approximately 2.4 cm deep, persisted for 12 more weeks (Figure 5), and was the last area of the wound to close.

Magnetic resonance imaging of the buttock showed no evidence of osteomyelitis or abscess. Moist wound care was continued until the wound closed completely by secondary intention, 15 weeks after discontinuation of NPWT and approximately 6.5 months after his original cardiac surgery (Figure 6). The patient remained insensate over the buttock cheeks, indicating that sensory nerves were affected. The chest wound also healed by secondary intention after discontinuation of NPWT. Neither wound required a surgical flap, and 1 year later, the wounds remained closed.

DISCUSSION

In the case study presented here, the distribution of the evolving DTI and subsequent necrosis are not logically explained by the prevailing pathophysiologic mechanism of local capillary compression.[1] The soft tissues of the buttock on either side of the gluteal cleft do not overlie any bony structure. Although PrIs over the sacral bone itself might be explained by the prevailing mechanism of capillary occlusion from local pressure, full-thickness necrosis of the skin, subcutaneous tissue, fat, and muscles of the fleshy buttocks cannot be explained by this mechanism. So what, then, happened?

The patient developed what appeared to be a severe PrI of the fatty tissue of the buttock following an extended period of hypotension. Normal MAP is between 65 and 110 mm Hg. A value of 50 mm Hg is low enough to cause ischemic injury to tissues and organs[6] and was likely the cause of his PrI. To increase MAP, vasopressors can accentuate the body's innate value judgment regarding blood-flow redistribution during hypotensive episodes, shunting blood away from the skin to vital organs such as the brain and kidneys.[32] Vasopressors are known to result in ischemia to peripheral tissues such as the digits and may also be associated with medically unpreventable PrIs.[6]

More than 200 significant risk factors of PrI development have been identified.[33] However, few of the PrI risk factors common to critically ill patients are addressed in prevention protocols.[9] For example, among critical care patients, one of the strongest predictors of PrI development is hypotension, a factor not addressed by PrI prevention protocols.[6,10–13]

Ischemia-Reperfusion Injury and the Angiosomal Hypothesis of PrI Formation

It has been suggested that the necrosis observed in stage 4 PrIs is indicative of an irreversible ischemia-reperfusion (or reoxygenation) injury.[34] Reperfusion injury is the tissue damage caused when blood supply returns to tissue after a period of time of ischemia or lack of oxygen. The absence of oxygen and nutrients from blood during the ischemic period creates a condition in which the restoration of circulation results in inflammation and oxidative damage through the induction of oxidative stress rather than (or along with) restoration of normal function.

Restoring blood supply to ischemic tissue is essential; unfortunately, reperfusion of ischemic tissues is associated with microvascular injury, inflammation, and oxidative damage.[35] Although ischemia-reperfusion injury has not been conclusively studied in the context of human PrI, it is a logical explanation for the microcirculatory damage that occurs when a previously ischemic area of the skin or underlying tissue is exposed to oxygen-rich blood.[36–38]

Figure 3. SACRAL PRESSURE INJURY FOLLOWING SURGICAL DEBRIDEMENT

The injury is pictured immediately following surgical debridement on postoperative day 18. The largest area of the wound comprised its more superficial aspect, whereas the deeper part of the wound was smaller, with the two areas forming a noticeable cone shape. A rim of violaceous tissue surrounded the lesion.

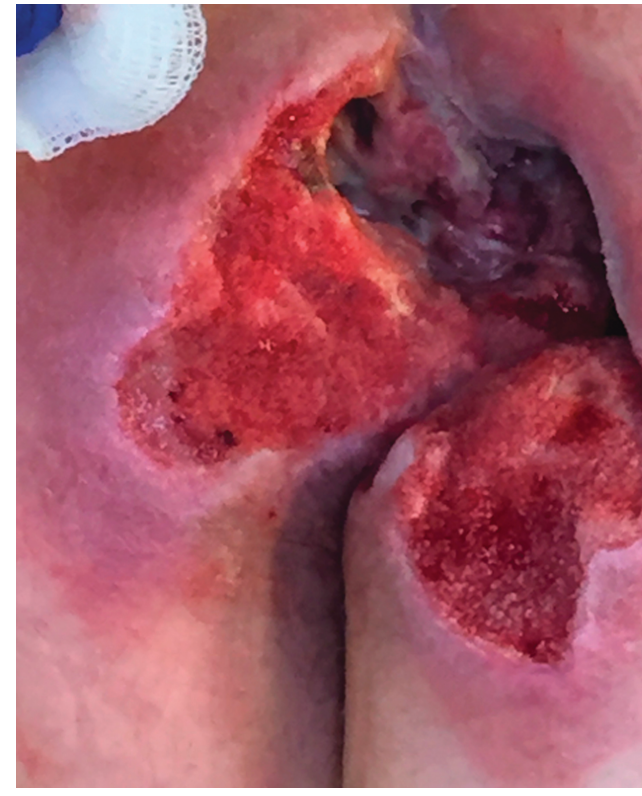
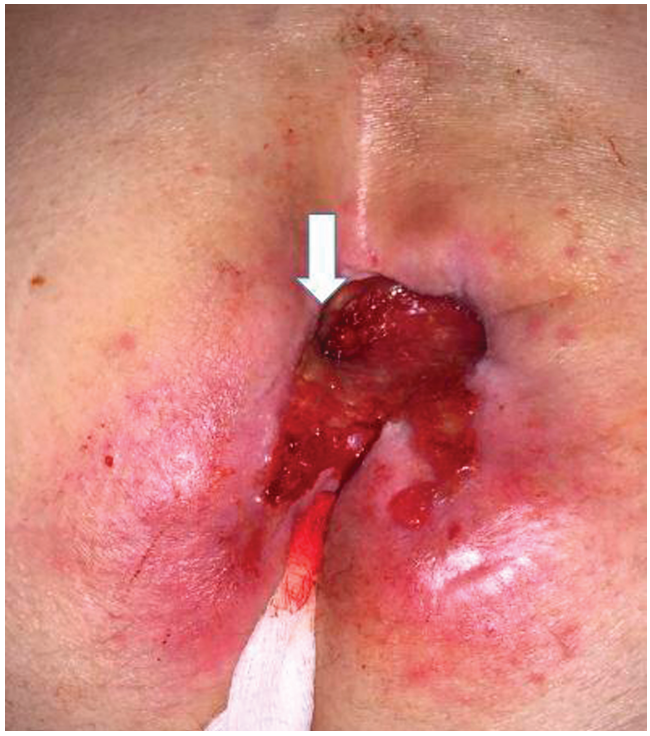


Figure 4. AFTER 13 WEEKS OF NEGATIVE-PRESSURE WOUND THERAPY

The sacral wound is pictured here after 13 weeks of negative-pressure wound therapy, which was stopped because of heavy tissue colonization. At this time, the wound measured 5.4 × 3.2 × 1.5 cm. The white arrow points to a 2.4-cm tunnel beside the sacroiliac ligament.



This patient's stage 4 PrI followed the bilateral anatomic distribution of the perforating vessels of the superior gluteal artery. The distribution of tissue necrosis makes angiosomal ischemia a logical explanation. The authors begin by observing that in this article's case, the lesions over the buttock follow the distribution of named vessels in an angiosomal pattern. As noted above, an angiosome is a three-dimensional block of tissue supplied by a main source artery and its accompanying vein(s).[4,39,40] In most cases, the angiosome extends from the bone all the way to the skin.[4,39] The cutaneous perforators pierce the deep fascia near where they are anchored to the bone or intramuscular septa and then flow toward the skin. In most angiosomes, the surface area of the cutaneous tissue supplied by the vessel is large, whereas the deep tissue region supplied is small. The entire body is covered with a patchwork quilt of these composite blocks of skin, bone, muscle, and other soft tissues neatly fitted together. The sides of these three-dimensional blocks (the junctional zones) are linked by arterial and venous anastomoses, usually within the muscles of the deep tissue such that if the main source artery or vein is occluded, the muscle acts as a bypass shunt.[4,39]

The importance of angiosomes in the pathophysiology of lower-extremity ulceration and their treatment via revascularization is well established.[41–51] Among patients with peripheral arterial disease, when a main source artery is occluded, ischemia of the related angiosome results.[46] In these patients, angiosome-guided revascularization has demonstrated significantly faster and higher wound healing rates and improved limb salvage outcomes.[46–51] In patients with peripheral ischemia, authors recommend that the angiosome concept should always be applied intraoperatively to preserve the blood supply.[45]

In this perioperative case, both buttock cheeks are angiosomes of the superior gluteal arteries (SGAs) and the parasacral arteries,[52] whereas the ischial area is supplied by the inferior gluteal arteries. Based on the above anatomic description and the hematomas under the gluteus muscle, it is possible that the PrI resulted from an occlusion of the bilateral superior gluteal veins (SGVs) with subsequent ischemia of the associated angiosomes. Because angiosomes normally extend from the bone to the skin, tissue loss in the same distribution as the vessel is expected, with the largest surface area of tissue affected at the skin surface and the smallest surface area in the deep tissue. Further, as the skin is supplied by fasciocutaneous vessels passing through the muscle and subcutaneous tissue, if the skin becomes necrotic, it does so because the underlying subcutaneous tissue and muscle are already dead (an inside-out type of necrosis). The junctional zone may or may not have a sharp demarcation, because the muscle of the adjacent angiosome can act as a shunt. However, findings will differ depending on many other factors, such as whether the event began with occlusion of the artery or vein or a general low flow state, the overall health of the surrounding tissue, and whether there were multiple short periods of ischemia or a single long one.

Figure 5. PARTIALLY CLOSED SACRAL WOUND

This depicts the partially closed sacral wound with the tunnel along the sacrum still unhealed.

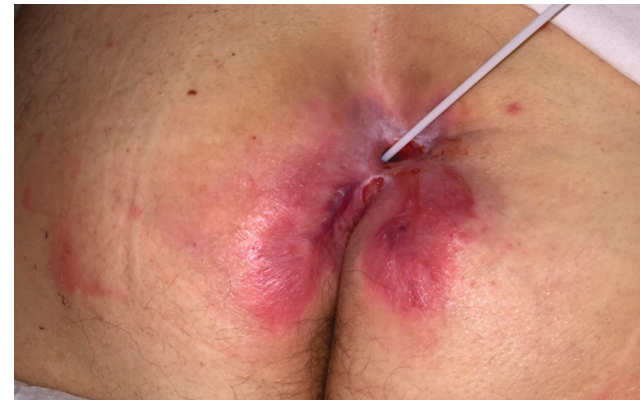
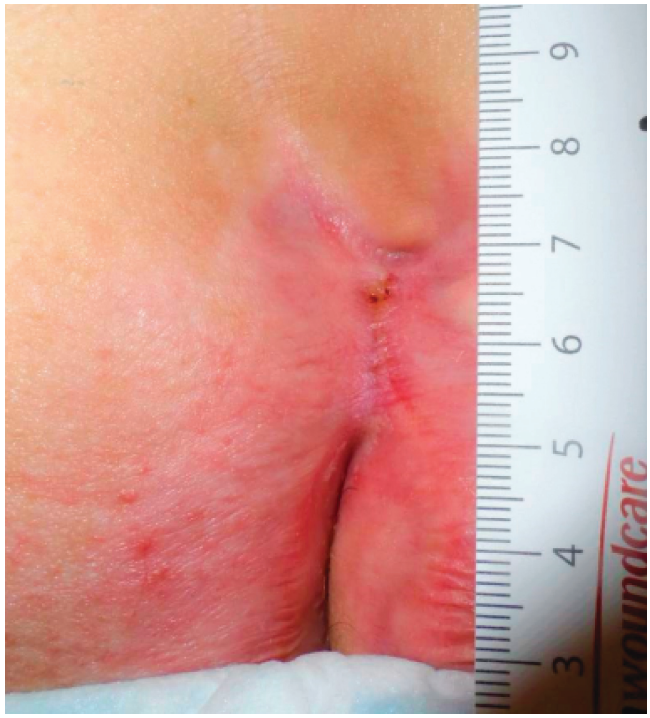


Figure 6. COMPLETELY HEALED SACRAL WOUND

The sacral wound was completely healed following 15 weeks of moist wound care approximately 6.5 months after the patient's original cardiac surgery.



This case provides confirmation that the pathophysiologic process in at least some stage 4 PrIs is not local pressure and does not occur at the capillary level, but rather is a type of vascular compromise resulting in tissue necrosis in the anatomic distribution of a named vessel, which is exactly what is seen with most stage 4 PrIs if viewed from this perspective. Pressure injuries commonly occur in the distribution of the inferior gluteal artery (ischial ulcers) or lumbar arteries (sacral ulcers).

If DTIs and stage 4 PrIs represent the infarction of a named vessel within its associated angiosome, then efforts should focus on understanding the "series of unfortunate events" that precede it. Consider that the SGA exits the greater sciatic foramen above the piriformis muscle and divides into superficial and deep branches to supply the gluteus muscles.[52] There is a choke point as it divides, exiting the fascia near the tight sacroiliac ligaments. Given this case patient's low albumin and the common presence of hypoalbuminemia in similar cases, it is possible the problem began as a venous obstruction at the choke point where the vessels pierce the deep fascia, accentuated by a low oncotic pressure and extravascular leakage from the most distal vessels. The weight of the patient's body against the bed could have obstructed the return of venous blood through the SGV at the choke point, leading to the hemorrhages under the gluteus observed at the time of debridement. One could also postulate that the trend toward permissive hypotension and a nationwide reduction in the use of blood products might contribute to the development of DTIs and stage 4 PrIs.[53,54] If so, even if the MAP cannot be improved, raising serum albumin and/or hemoglobin via transfusion might sustain flow within the SGA (or SGV) and decrease the likelihood of buttock DTI. As already observed, the risk factors for stage 4 PrIs are largely cardiovascular.

Although it is true that "pressure" is integral to this event, tissue ischemia does not originate at the local capillary level, but rather at the regional macrovascular level. The vascular event does not occur directly over the zone of tissue necrosis but proximal to it, where the vessels of the angiosome originate. Case in point, photographs demonstrate a deep defect at the superior pole of the gluteal cleft, proximal to the PrI and some distance from the original DTI that presented on the buttock cheeks (Figure 3).

Readers may ask, why do all at-risk patients not develop PrIs if ideal care situations remain an exception rather than the rule? Why did the groundbreaking TURN study by Bergstrom and colleagues[55] not demonstrate an increase in PrIs among high-risk patients turned every 4 hours rather than every 2? The determining factor for tissue ischemia may be whether the mean arterial (or venous) pressure within the relevant angiosomes or venosomes is sufficient to prevent the occlusion of flow through an anatomic choke point. The current protocol of placing a foam pad over the sacrum (which was done for this case patient) may prevent superficial skin damage from friction and shearing but not a DTI from vascular occlusion originating deep in the muscle. Further, it is not possible to prevent the weight of the patient's own body from occluding blood flow through an anatomic choke point if the body weighs enough and the intravascular pressure is low enough.

CONCLUSIONS

As the dominant pathophysiologic mechanism suggests, some PrIs are likely caused by capillary occlusion from pressure at the bony interface.[2] Pressure is indeed a factor in the formation of severe PrIs, but these authors postulate that the occlusion occurs at the level of a named artery proximal to the lesion. In the perioperative case presented here, this vascular event was likely attributable to low MAP. Angiosomal ischemia would explain the "inside-out" pathophysiologic process often observed in severe PrIs, as well as the three-dimensional "cone" of tissue loss that is also a common feature. This hypothesis explains the anatomic distribution of PrIs that is familiar to wound care practitioners, but which has not been seen heretofore through an anatomic lens.

If the angiosomal hypothesis has merit, it has significant implications for the creation of better PrI prevention protocols, particularly among critically ill patients. If vascular ischemia is the final common pathway for tissue destruction, critical care PrI mitigation protocols should target potentially modifiable risk factors such as hemodynamic instability, hypotension, hypoalbuminemia, hypoxia, anemia, and reduced cardiac output, with a combined emphasis on preserving skin perfusion in critical care patients.[10,12] Because the management of these physiologic factors is normally the purview of the medical team, the impact of such an approach to PrI prevention would be to—at last—obtain the full engagement of physicians in partnership with the nursing staff. This pathophysiologic mechanism may help the discipline define the "medically unpreventable" PrI, perhaps reducing meritless litigation against healthcare providers and institutions that occur with critical illness.

It is the hope of these authors that individuals who research PrI formation will explore this concept. In the meantime, and until additional data are available among critically ill patients, closer attention should be given to the modifiable factors that affect angiosomal perfusion. There is an urgent need to address this multibillion-dollar-per-year problem, which is associated with significant human suffering. The time is right for a new hypothesis focused on the vascular pathophysiologic mechanism of DTIs and stage 4 PrIs, although it is not a new concept. These authors endorse adoption of the terminology proposed three decades ago to call both DTIs and stage 4 PrIs "vascular occlusion pressure injuries." ●

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