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CASE REPORT

Whole-Process Skin Management Strategies in a 19-Week Pregnant Woman With Acute Skin Failure Induced by Inevitable Abortion: A Case Report

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Abstract: Acute skin failure (ASF) is a critical condition marked by rapid skin deterioration, catastrophic skin dysfunction due to systemic hypoperfusion, often occurring alongside multi-organ failure. This report presents the case of a 37-year-old woman who developed rapid skin necrosis within less than a week and was diagnosed with ASF. The progression of her condition was categorised into three stages: progressive, necrotic and reparation. A comprehensive skin management strategy was applied at each stage, comprising systemic stabilization (antibiotic therapy/fluid resuscitation), stage-targeted local care (protective foam dressings in the progressive stage, chlorhexidine antisepsis in the necrotic stage, and recombinant human epidermal growth factor [rhEGF] gel in the reparation stage), and early rehabilitation (ankle pump exercises)—was applied at each stage, leading to successful healing and discharge on the 42nd day of hospitalisation. This case highlights the effectiveness of a staged approach to ASF management and emphasises the need for further research to establish standardised diagnostic criteria and a pathophysiological understanding of ASF. Thus, this case describes the patient's ASF course (skin appearance and the patient's feelings), together with the care evaluations at each stage and key treatment points. This study aims to improve the medical staff's ability to recognise and treat ASF in critically ill patients.

Keywords: acute skin failure, skin necrosis, progressive stage, necrotic stage, reparation stage, case report

Introduction

Acute Skin Failure (ASF) is a life-threatening condition characterized by rapid, catastrophic skin dysfunction due to systemic hypoperfusion, often occurring alongside multi-organ failure. ASF was first conceptualised in 1991 by La Puma, who proposed that 'the skin is the largest organ of the body; if the heart and lungs and kidneys fail, the skin is likely to fail too.[1] This notion was expanded in 2006 by Langemo, who classified skin failure into acute, chronic and end-stage categories.[2] Acute skin failure is characterised by the death of skin and underlying tissues due to hypoperfusion, often coinciding with multi-organ dysfunction.[2] ASF typically arises from severe systemic insults that trigger hypoperfusion and microvascular thrombosis, most commonly sepsis, disseminated intravascular coagulation (DIC), vasopressor-dependent shock, or multi-organ dysfunction syndrome (MODS). The current treatment strategy prioritizes systemic therapy. Septic shock and DIC were reversed through blood transfusion, anti-infection treatment (with biapenem), and continuous renal replacement therapy (CRRT), which laid the foundation for skin recovery. Meanwhile, the wounds were intervened. However, there are currently no unified diagnostic criteria for ASF, and there are very few descriptive studies on its occurrence worldwide.[3] Although the basic service system has developed rapidly, the quality levels of medical technology and personnel allocation differ.

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Despite advances in healthcare systems, varying levels of understanding about ASF among medical staff have led to misdiagnosis or delayed treatment.[4] This condition, frequently seen in critically ill patients, has been associated with high mortality rates, and survivors often require amputation of the affected extremities.[5–8] This case report presents a pregnant woman who rapidly developed ASF, with skin necrosis occurring within 7 days of admission. The progression of ASF, in this case, was categorised into three distinct stages: progressive, necrotic and reparative. Targeted interventions were implemented at each stage by a multidisciplinary team, resulting in significant recovery.

Case Description

On 2 July 2023, a 19-week pregnant (gravida 4, para 2) healthy woman presented to the obstetrics department with a 2-day history of vaginal discharge and hyperthermia (max. 40°C). An ultrasound examination revealed foetal death. The laboratory report was as follows: neutrophils = 12.28 × 10^9/L, leukocytes = 14.82 × 10^9/L and C-reactive protein = 59.13 mg/L. Upon admission, the patient was diagnosed with (1) premature membrane rupture, (2) inevitable abortion and (3) intrauterine infection. After admission, the patient was administered mifepristone orally to induce labour, along with an intravenous antibiotic infusion, including cefoperazone sodium with sulbactam sodium (3.0 g, twice daily) and levornidazole disodium phosphate (1.0 g, once daily).

On 3 July, a dead foetus was delivered along with the complete placenta. Subsequently, the patient developed severe postpartum haemorrhage and underwent a pelvic artery embolisation. However, the persistence of the postpartum haemorrhage led to a subsequent total abdominal hysterectomy. Five coagulation profile tests were as follows: prothrombin time of 51.9 s, prothrombin international standard ratio value [4.58], activated partial thromboplastin time of 91.1s, thrombin time of 88.5 s and undetectable fibrinogen. She was transferred to the authors' department for further treatment due to disseminated intravascular coagulation (DIC) and postoperative hemodynamic instability.

After being admitted to the intensive care unit (ICU), the patient was sedated and given endotracheal intubation as well as mechanical ventilation. Her vital signs were as follows: blood pressure of 106/68 mmHg (norepinephrine 0.3 ug/kg/min), heart rate at 110 beats/min at 36°C body temperature and clammy skin on the limbs. The laboratory indicators were as follows: neutrophils = 13.38 × 10^9/L, white blood cells = 14.59 × 10^9/L, red blood cell count = 2.81 × 10^{12}/L, haemoglobin = 73 g/L, haematocrit = 23.1% and platelets = 24 × 10^9/L. The five coagulation parameters were as follows: prothrombin time = 21.6 s, international standard ratio of prothrombin = 1.89, fibrinogen = 0.3 g/L and D-dimer >10 mg/L, whereas activated prothrombin time was undetectable. The patient developed a high fever postoperatively, with a peak body temperature of 39°C. *Escherichia coli* was identified through peripheral venous blood and hemoperitoneum cultures. Subsequently, the patient received an intravenous biapenem infusion and mechanical ventilation for approximately 18 days post-admission. However, it was removed as the disease improved.

On the 3rd hospitalisation day (4 July), a larger dosage of vasoactive agents was administered to maintain the target blood pressure (a mean arterial pressure of 70–75 mmHg) with the maximum dose of norepinephrine = 2 μg/kg/min and pituitrin = 2 u/h (for 4 hours). On day 4 of hospitalisation (5 July), her haemodynamics stabilised after a maximum dose of norepinephrine (0.4 μg/kg/min), which was gradually discontinued.

On the 3rd hospitalisation day (July 4), continuous renal replacement therapy (CRRT) was provided (in continuous venovenous haemodiafiltration mode) for approximately 18 days until renal function recovered. At 4:15 p.m. on day 4 of hospitalisation (5 July), the patient's heel skin was red and cold. A few hours later (9:00 a.m. on the 5th admission day), the skin on both sides of the dorsal and soles of the feet showed purpura, erythema and bullae (maximum diameter >8 cm). At 7 a.m. on the 6th admission day, a portion of the patient's toes turned black and necrotic on the back of the feet. Bullae fluid from her feet was examined; however, no bacterial population was detected.

Purpura fulminans and symmetrical gangrene are associated with septic shock and DIC. Purpura fulminans often present as systemic, diffuse and extensive full-layer skin lesions.[9] Symmetrical peripheral gangrene is the necrosis of the entire skin that often involves the muscle layer. The discoloured skin becomes necrosed and eventually needs amputations.[10] However, only foot necrosis and superficial skin were observed in this patient. This is different from the first two types of skin lesions. Additionally, she received vasoactive drugs within the first few days of admission. However, vasopressor drugs causing skin necrosis have also been reported before. A recent review suggested that the key factor in the pathogenesis of symmetrical peripheral gangrene is the natural anticoagulant depletion theory, and skin

necrosis caused by vasopressor drugs is not plausible.[11] The main pathogenic factors causing pressure injuries were subcutaneous pressure and shear force; however, the patient's skin lesions were not under stress. The patient was diagnosed with multiple organ dysfunction syndrome (MODS) and displayed skin hypoperfusion (hypothermia and peripheral perfusion index <0.3). Eventually, she was diagnosed with ASF.

Based on the skin lesion progression, ASF was divided into three stages: progressive, necrotic and reparation (Figure 1). There are no reports on skin progression stages in ASF to date.

Progressive stage (5 days): By the 5th day of admission, the patient developed scattered purpuric rashes on her feet, especially the plantar, instep and toe surfaces of the right foot (Figure 2). Doppler ultrasound confirmed unobstructed blood flow in the lower limbs, and her bilateral dorsal pedis arteries were palpable. A multidisciplinary team initiated treatment, including wound management, CRRT, infection control, respiratory support and rehabilitation.

To stabilise her condition, the team administered blood transfusions, fluid resuscitation and vasopressor drugs. Her organ function was maintained with mechanical ventilation and CRRT. Foot temperature was closely monitored, local

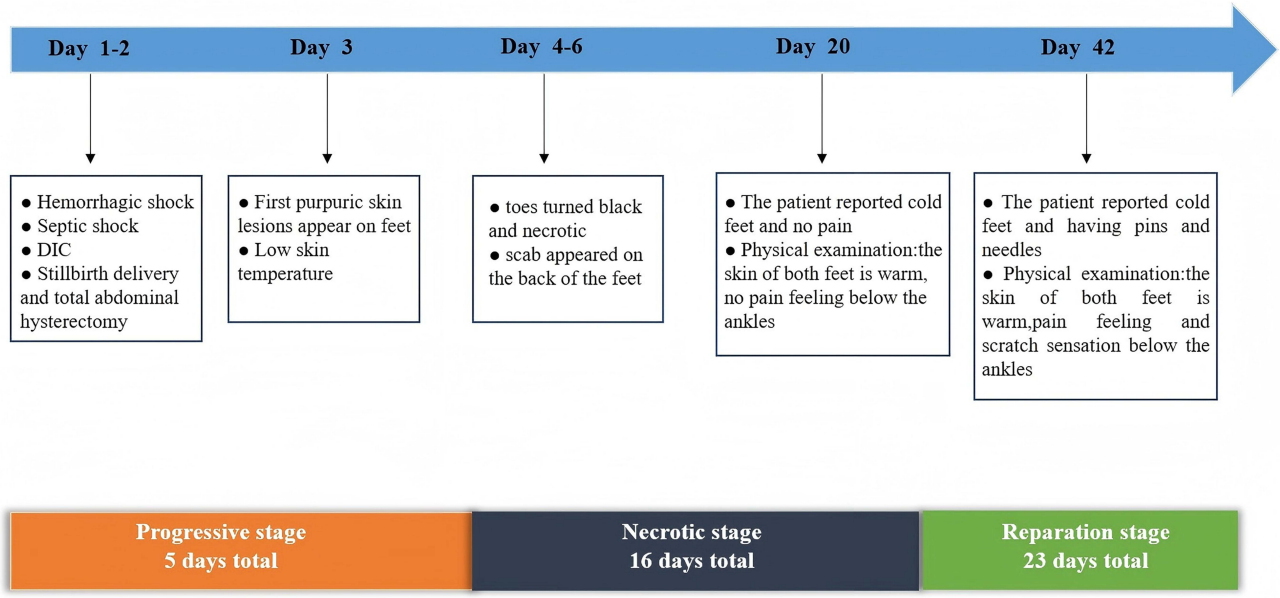

Figure 1 Three stages and key features of ASF. Timeline of the events summarizing the most important moments in the development and management of this patient.

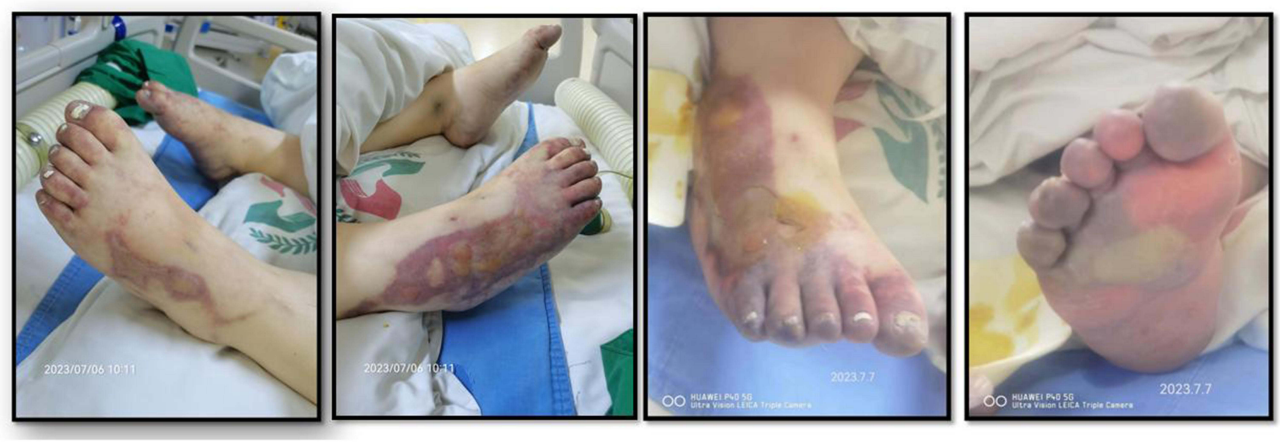

Figure 2 Progressive stage of ASF. Includes erythema, fused into patches, blistering.

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bullae were drained and protective foam dressings were applied. Despite these efforts, the condition of her feet rapidly worsened, progressing to necrosis.

Necrotic stage (16 days): On the 6th admission day, a portion of the patient's plantar, instep and toe surfaces turned black and necrotic. However, her bilateral dorsal pedis arteries were still palpable (Figure 3). The medical team continued daily wound assessments and dressing changes, applying foam dressings and disinfecting the areas with chlorhexidine. The chlorhexidine (0.5% solution) was chosen over alternatives like povidone-iodine due to its broader antimicrobial spectrum against Gram-positive/Gram-negative bacteria, sustained residual activity, and reduced cytotoxicity on viable tissue margins—critical for limiting infection in devitalized skin. As the patient's condition improved, vasopressors were gradually reduced, and her nutritional intake was increased. Moreover, a psychotherapist provided psychological counselling while the patient received bedside companionship from her spouse, thereby promoting emotional adjustments.

Reparation stage (23 days): On the 20th admission day, a physical examination revealed that the wounds on the patient's feet were well-covered by the dressings, and red granulation tissue was visible. At this stage, the attending physicians conducted regular sensory perception assessments and gradually increased the rehabilitation exercises to improve the prognosis (Figure 4). The focus shifted to promoting tissue growth with growth factor gel and increasing nutrient intake. The growth factor gel applied during the reparation stage contained recombinant human epidermal growth factor (rhEGF), selected for its evidence-based efficacy in stimulating angiogenesis and epithelialization in full-thickness wounds. The patient's pain and cold feet persisted; therefore, a rehabilitation therapist introduced ankle pump exercises. After a week, the patient was able to perform these exercises independently.

Post-Acute Care and Outcome

Post-discharge, the nurse provided discharge education and instructed the patient to avoid foot baths before the wounds healed. Additionally, the patient was instructed to report to the community hospital every 3 days for foot dressing and rehabilitation exercises. Furthermore, patient recovery information was obtained through weekly telephone follow-ups. After recovering from haemorrhagic shock and multiple organ failure secondary to septic shock, she was discharged from the ICU on day 42. Furthermore, the skin on her feet had healed well at the 2-month follow-up.

Discussion

This report showed the case of a 37-year-old woman who presented with rapid skin necrosis in <7 days and had ASF. Acute skin failure has been categorised into three stages based on the progression of the skin lesions: progressive, necrotic and reparation. By applying whole-process skin management strategies based on the skin failure progression stage, the skin of the patient's feet healed well, and she was discharged successfully on day 42 of hospitalisation. Thus, this treatment intervention was safe, feasible and beneficial in such cases.

Figure 3 Necrotic stage of ASF. The wounds turned black.

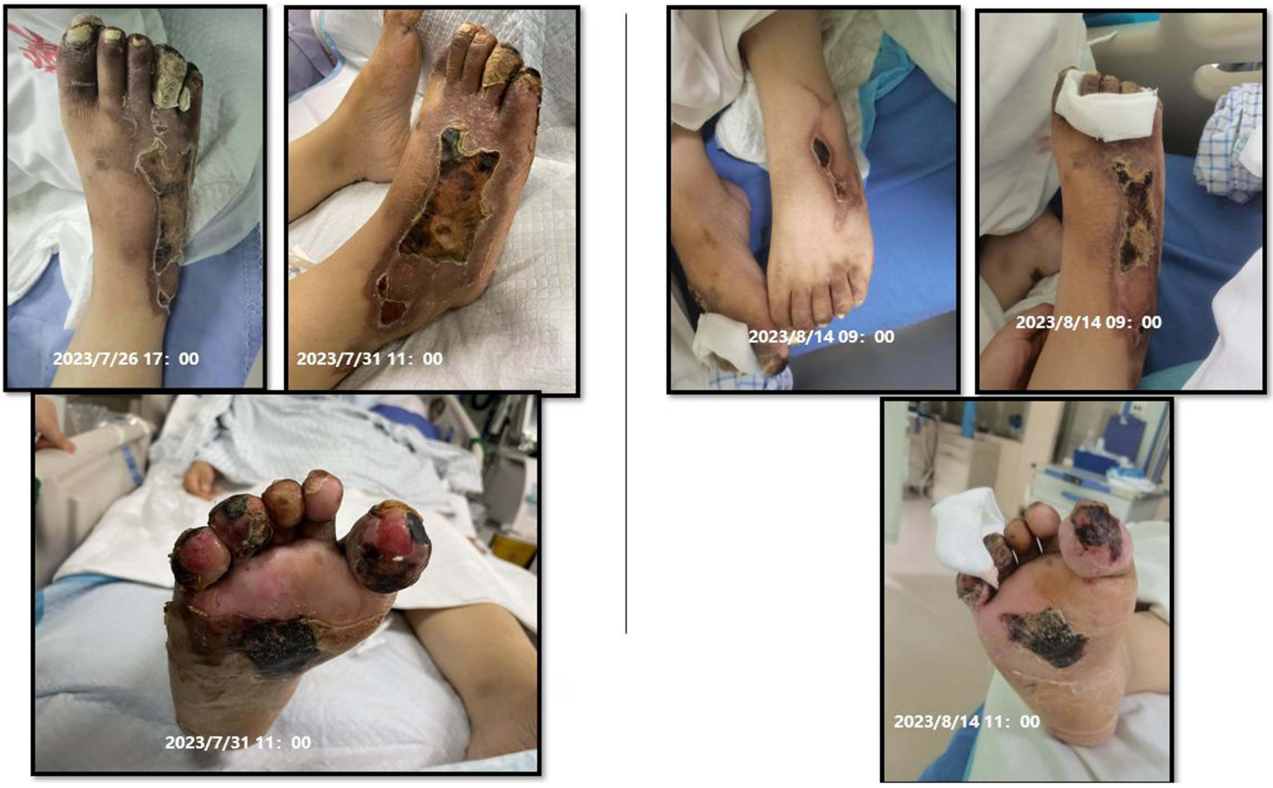

Figure 4 Reparation stage of ASF.

Initially, it was questioned whether studies on ASF should not be conducted, especially after searching relevant studies on skin necrosis,[6] gangrene and purpura fulminans.[8,12,13] Many similarities, including a population similar to the majority of critically ill patients, were found. Skin progression was similar to cutaneous manifestations such as acrocyanosis, purpura, necrosis and gangrene. At present, Langemo's definition of ASF holds more value.[2] Thus, critical care providers have gradually accepted the concept of ASF since its introduction. This is due to its simplicity in teaching healthcare professionals and non-professionals. However, there is a lack of uniform world-wide diagnostic criteria and relevant pathophysiological studies to date.[3] Acute skin failure occurs concurrently with multi-organ failure.[2] However, the skin has no specific diagnostic equipment or laboratory-positive indicators, unlike other organs. Therefore, additional studies are required to explain ASF.

While ASF is not directly caused by pregnancy, obstetric complications can *indirectly* trigger ASF through shared pathophysiological pathways. Pregnancy induces a state of physiological hypercoagulability and immunomodulation, which —when compounded by critical events like septic abortion, postpartum hemorrhage, or DIC creates a high-risk environment for systemic hypoperfusion and microvascular thrombosis. In this case, ASF developed after the patient experienced septic shock and DIC following an inevitable abortion, leading to rapid skin necrosis. Though ASF itself shows no inherent gender predominance, pregnancy-specific conditions (such as, amniotic fluid embolism, preeclampsia-related DIC, or obstetrical sepsis) may disproportionately expose women to ASF-precipitating factors. Documented cases remain exceptionally rare, with sparse literature explicitly linking ASF to pregnancy; most reports describe comparable pathologies like purpura fulminans or symmetrical gangrene in obstetric crises. Thus, pregnancy acts as a catalyst rather than a direct cause, where systemic collapse—not gestation itself—drives ASF. Vigilance for early skin changes in pregnant patients with hemodynamic instability or multi-organ failure is critical, as prompt intervention may mitigate progression.

This case describes the complete progression of ASF from the onset of skin problems to its healing. Additionally, the corresponding treatment methods were incorporated at different times to achieve satisfactory therapeutic effects as per the patient's skin progression. This concludes the authors' proposal for a whole-process skin management strategy.

The key factors that potentiated skin healing in this complex ASF case includes: (1) Early Multidisciplinary Coordination: Daily wound assessments by intensivists, dermatologists, and wound nurses enabled real-time intervention adjustments—particularly critical during the rapid progressive-to-necrotic transition (Days 4–6). (2) Targeted Antiseptic Selection: Chlorhexidine's persistence on necrotic tissue (unlike iodine) created a sustained barrier against *E. coli* biofilm formation, while its low cytotoxicity preserved marginal viable tissue for subsequent regeneration. (3) *Growth Factor Precision*: Recombinant human EGF (rhEGF) gel applied during the reparative stage directly stimulated fibroblast migration and collagen III synthesis, accelerating granulation tissue formation observed by Day 20. (4) Dynamic Rehabilitation Integration: Bedside ankle pump exercises initiated in the necrotic stage (despite pain) improved microcirculation, preventing contractures and enabling active motion by Day 27. (5) Aggressive Metabolic Support: High-protein nutrition (1.5–2 g/kg/day) and micronutrient repletion (zinc, vitamin C) counteracted catabolic losses from CRRT/sepsis. These factors—coupled with strict pressure-offloading—collectively explain the unprecedented limb salvage achieved, providing a template for managing rapid-onset ASF.

Similar to pressure ulcers and incontinence-associated dermatitis,[14,15] ASF is included in the category of dermatological symptomatology. Consequently, based on the staging and grading criteria for pressure injuries and incontinence-associated dermatitis, the patient's skin failure was divided into three stages: progressive, necrotic and reparation. To our knowledge, this is the first descriptive study to outline the staging and progression of ASF both domestically and internationally. This framework can enhance understanding among intensive care professionals. However, managing ASF presents challenges, particularly because the necrotic stage can rapidly follow the progressive stage without a clear time interval. Further research is needed to refine diagnostic criteria, pathophysiological understanding and management strategies for ASF, ensuring more effective care and better patient outcomes.

Conclusion

This case demonstrates that ASF, while rare in obstetric patients, can arise rapidly from septic shock and DIC following pregnancy complications. Critical diagnostic elements included: (1) hemodynamic instability preceding skin changes, (2) symmetrical distal limb necrosis *without* pressure or vascular occlusion, and (3) concurrent multi-organ failure—distinguishing ASF from pressure injuries or isolated gangrene. Our staged management strategy proved pivotal: Progressive stage (Days 1–5): Aggressive systemic stabilization (CRRT, vasopressors, antibiotics) preceded skin interventions. Protective foam dressings mitigated shear stress, but necrosis progressed despite unimpeded arterial flow, underscoring ASF's microvascular pathogenesis. Necrotic stage (Days 6–21): Chlorhexidine antisepsis (selected for biofilm penetration and tissue preservation) prevented infection in devitalized tissue, while psychonutritional support counteracted catabolism. Reparation stage (Days 22–42): rhEGF gel accelerated granulation, and early rehabilitation (ankle pumps) preserved function.

For clinicians, we emphasize that ASF recognition requires vigilance for rapidly progressive symmetrical necrosis in critically ill patients, particularly those with DIC/sepsis. Stage-adapted interventions (systemic rescue → infection control → regenerative rehab) are essential to prevent irreversible tissue loss. This approach, generalizable to ASF beyond pregnancy, fulfills our aim to improve detection and staged management of this underrecognized emergency.

Ethics Approval

This study was conducted in accordance with the Declaration of Helsinki and approved by the ethics committee of The Fourth Hospital of Hebei Medical University (approval number 2024KS190).

Patient Consent Statement

The written informed consent has been provided by the patient to have the case details and any accompanying images published.

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Disclosure

The authors report no conflicts of interest in this work.

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