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Case Report

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Posted Date: September 30th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10769867/v1>

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Additional Declarations: No competing interests reported.

Successful non-grafted second-intention healing of a massive gluteal soft-tissue wound complicating septic shock with secondary hemophagocytic lymphohistiocytosis in a patient with type 2 diabetes: a case report

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Abstract

Background:

A furuncle of the gluteal region, aggravated by scratching in a patient with poorly controlled type 2 diabetes, may progress to necrotizing soft-tissue infection, septic shock, and life-threatening immune dysregulation. We report an unusual case in which a seemingly trivial furuncle precipitated septic shock with secondary hemophagocytic lymphohistiocytosis (HLH) and multi-organ dysfunction, complicated by an 18 x 8 cm full-thickness gluteal wound adjacent to the anus, yet healed completely without skin grafting.

Case presentation:

A 53-year-old Han Chinese man with a one-month history of type 2 diabetes and a 15-year history of hypertension scratched a right gluteal furuncle on September 13, 2024. Within days he developed septic shock with profound pancytopenia (white blood cell count $1.25 \times 10^9/L$, absolute neutrophil count 0.13, platelets $11 \times 10^9/L$, hemoglobin 89 g/L), peak procalcitonin 25.3 ng/mL and interleukin-6 > 5000 pg/mL, and cardiac, hepatic, and renal dysfunction. He fulfilled six of seven applicable HLH-2004 criteria (fever, splenomegaly, cytopenias, hypertriglyceridemia with hypofibrinogenemia, elevated soluble interleukin-2 receptor at 3027 U/mL, and ferritin > 500 ng/mL; bone marrow showed no hemophagocytosis). Targeted gene sequencing (tGS) of wound secretions identified methicillin-resistant *Staphylococcus aureus*, *Klebsiella pneumoniae*, *Stenotrophomonas maltophilia*, *Enterococcus faecium*, *Pseudomonas aeruginosa*, and *Candida* species. After resuscitation, broad-spectrum antimicrobials, granulocyte colony-stimulating factor, and dexamethasone for HLH, surgical debridement on day 20 disclosed an 18 x 8 cm full-thickness necrotic wound with honeycomb-like fat necrosis and copious pus; vacuum-assisted closure (VSD, 15 x 5 cm foam) drained substantial pus but could not cover the entire wound. Subsequent twice-daily debridement with topical zinc sulfadiazine cream, then an antimicrobial ointment and a chlorhexidine-based antimicrobial ointment, progressively yielded granulation tissue, marginal epithelial advancement, and wound contraction. The patient was discharged on day 53 with a 15 x 5 cm contracting wound and continued daily self-dressing changes. Serial photography documented progressive re-epithelialization; the patient reported complete healing by March 2025, achieving near-scarless second-intention healing without skin grafting. At 16-month follow-up (June 2026), the previously massive wound had healed by spontaneous epithelialization without skin grafting, leaving an elongated atrophic scar that was flat, without hypertrophic elevation, depression, ulceration, sinus tract, erythema, or exudate, with hypopigmentation over the scar and hyperpigmentation of the surrounding skin, and without scar contracture.

Conclusions:

A minor diabetic furuncle can trigger septic shock with secondary HLH and a massive perianal wound. When systemic stabilization, source control through debridement and negative-pressure therapy, and disciplined sequential topical wound care are combined, even an extensive contaminated wound adjacent to the anus may heal by second intention without skin grafting. The traditional Chinese medicine-based ointment used here (propolis, *Ganoderma*, *Angelica sinensis*, mulberry bark, and *Sophora flavescens* in a beeswax-sesame oil base) maintained a moist wound environment, promoted autolytic debridement of necrotic tissue, and supported granulation growth, contributing importantly to the healing process.

Keywords: Septic shock; Hemophagocytic lymphohistiocytosis; Diabetic soft-tissue infection; Wound healing; Negative-pressure wound therapy; Methicillin-resistant *Staphylococcus aureus*; Case report

Background

Type 2 diabetes mellitus predisposes patients to skin and soft-tissue infections through impaired neutrophil function, microvascular compromise, and delayed wound healing [1]. A furuncle that is manipulated or scratched can disseminate into necrotizing soft-tissue infection and bacteremia, occasionally progressing to septic shock and multi-organ dysfunction [2]. In a subset of patients, the exuberant inflammatory response to infection triggers secondary hemophagocytic lymphohistiocytosis (HLH), a syndrome of pathologic immune activation defined by the HLH-2004 criteria and associated with high mortality when superimposed on sepsis [3,4].

Managing large soft-tissue wounds in patients with concurrent sepsis, HLH, cytopenias, and immunosuppressive therapy is challenging. The wound is often contaminated by polymicrobial flora, located in anatomically unfavorable sites, and aggravated by immunoparesis. Vacuum-assisted closure (VSD/NPWT) is established for complex wounds, yet wounds exceeding the dimensions of available foam, or adjacent to contaminating orifices such as the anus, may require supplementary or alternative strategies [5,6].

We present a case in which a scratched gluteal furuncle in a patient with newly diagnosed type 2 diabetes evolved into septic shock with secondary HLH and multi-organ dysfunction, complicated by an 18 x 8 cm full-thickness gluteal wound adjacent to the anus. Through systemic stabilization, sequential debridement, negative-pressure therapy, and disciplined daily topical wound care, the wound achieved complete re-epithelialization by second intention without skin grafting. Serial photography over approximately five months documented the entire wound trajectory. Written informed consent for publication was obtained from the patient, and this report follows the CARE guidelines [7].

Case presentation

Initial presentation and diagnosis

A 53-year-old Han Chinese man (height 170 cm, weight 85 kg, body mass index 29.4 kg/m²), a decorator with a 15-year paint-exposure history, had newly diagnosed type 2 diabetes mellitus of one month's duration treated with metformin, and a 15-year history of hypertension treated with nifedipine with good control. His mother had diabetes. On September 13, 2024, he scratched a right gluteal furuncle. Over the following days the right buttock became swollen and painful without overt redness, and he did not seek care. On September 23, worsening swelling with erythema and low-grade fever (peak 37.8 C) prompted presentation to a local hospital, where elevated inflammatory markers, pancytopenia, and acute kidney injury (creatinine 314 umol/L, blood pressure 87/61 mmHg) were found; empirical antibiotics did not improve his condition. On September 25 he presented to our emergency department,

where white blood cell count was $1.64 \times 10^9/L$, platelets $18 \times 10^9/L$, hemoglobin 95 g/L, procalcitonin 25.3 ng/mL, interleukin-6 > 5000 pg/mL, creatinine 328.7 $\mu\text{mol/L}$, NT-proBNP 7308 pg/mL, and fibrinogen 9.43 g/L; pelvic CT suggested possible hematoma around the left common femoral artery, and bedside ultrasound showed right gluteal soft-tissue edema. Empirical linezolid plus piperacillin-tazobactam was initiated at the emergency department; piperacillin-tazobactam proved insufficient for the polymicrobial burden, and although linezolid provided appropriate anti-staphylococcal coverage (subsequently validated by the MRSA identification), its clinical benefit had not yet become evident during this brief pre-transfer period.

On admission to the Department of Tropical Medicine (September 26, 2024), he was acutely ill: temperature 37.8 C, pulse 113/min, respiratory rate 40/min, blood pressure 132/90 mmHg, with scleral icterus. The right buttock showed a 14 x 13 cm swollen area with a central 6 x 5 cm purple-black suspected necrotic eschar; a 15 x 5 cm irregular purplish ecchymosis was present on the left proximal thigh. Laboratory testing (Table 1) revealed profound pancytopenia (white blood cell count $1.25 \times 10^9/L$, absolute neutrophil count 0.13, platelets $11 \times 10^9/L$, hemoglobin 89 g/L), markedly elevated inflammatory markers, coagulopathy (D-dimer 11,651 ng/mL, prothrombin time 22.5 s, fibrinogen 8.32 g/L), metabolic derangement (albumin 23.2 g/L, total bilirubin 145.1 $\mu\text{mol/L}$, creatinine 198.6 $\mu\text{mol/L}$, NT-proBNP 10,374 pg/mL), and arterial lactate 3.91 mmol/L. Chest computed tomography showed bilateral pneumonia; echocardiography showed an ejection fraction of 60%. Blood cultures grew Gram-positive cocci. Stool rapid on-site evaluation revealed abundant Gram-negative bacilli and fungal elements. The initial diagnoses were septic shock, gluteal soft-tissue infection, pneumonia, cholecystitis with cholelithiasis, infectious diarrhea, acute agranulocytosis, thrombocytopenia, anemia, heart failure, hepatic and renal dysfunction, gastrointestinal bleeding, and hypoproteinemia.

Septic shock management and recognition of secondary HLH

The patient was managed in a monitored bed with continuous electrocardiography, oxygen, and blood glucose monitoring. Notably, inflammatory markers had already begun to decline on the empirical linezolid-piperacillin/tazobactam regimen, suggesting the anti-staphylococcal component was beginning to take effect; however, because the platelet count was critically low (nadir platelets $5 \times 10^9/L$ on September 29) and linezolid carries myelosuppressive risk, antibiotics were changed to teicoplanin (400 mg every 12 h) plus cefoperazone-sulbactam (3 g); because stool rapid on-site evaluation and microscopy revealed abundant *Candida* spores and hyphae consistent with *Candida* enteritis following dysbiosis, oral amphotericin B was added for intestinal luminal *Candida* decolonization (oral amphotericin B is negligibly absorbed and acts within the gut lumen). Additional supportive measures included albumin supplementation, diuresis, acid suppression, granulocyte colony-

stimulating factor (G-CSF), and insulin for glycemic control; platelet transfusions were administered for critical thrombocytopenia. Over the first week inflammatory markers continued to decline (procalcitonin 14.1 to 2.19 ng/mL) and diarrhea improved, but the patient remained critically ill with ongoing fever, falling fibrinogen, and mild splenomegaly.

Given persistent fever, declining fibrinogen (8.32 to 1.73 g/L by October 8), mild splenomegaly (bedside ultrasound: spleen 12.6 x 4.7 cm), elevated triglycerides (2.51 mmol/L on October 2), ferritin > 500 ng/mL, and markedly elevated soluble interleukin-2 receptor (sCD25, 3027 U/mL; reference 223-710 U/mL), secondary HLH was suspected. Bone marrow aspiration on October 2 showed active proliferation with reduced erythroid lineage and scattered platelets but no hemophagocytosis. He fulfilled six of seven applicable HLH-2004 criteria (fever, splenomegaly, cytopenias affecting at least two lineages, hypertriglyceridemia and/or hypofibrinogenemia, elevated sCD25, and ferritin $\geq$ 500 ng/mL; bone marrow hemophagocytosis was absent, which does not exclude the diagnosis) [3]. Dexamethasone was initiated (10 mg daily, tapered to 5 mg then switched to oral methylprednisolone) and G-CSF continued; blood counts gradually recovered, with platelets reaching $188 \times 10^9/L$ by October 8.

Wound source control: debridement and negative-pressure therapy

By October 3, the right gluteal lesion had progressed to an 18 x 8 cm area of full-thickness skin necrosis, black and indurated, with surrounding cellulitis (Figure 1A-C). Under local anesthesia at the bedside, escharotomy and debridement were performed: the black eschar was excised, revealing necrotic fat with honeycomb-like interstices from which copious pus drained (Figure 1D, E). A vacuum-assisted closure (VSD) negative-pressure dressing (15 x 5 x 1 cm foam) was applied for continuous suction and irrigation, with attention to perianal soiling and airtight seal (Figure 1F). Because the 18 x 8 cm wound exceeded the foam dimensions, part of the wound could not be covered, and partial air leakage led to early conversion to open care.

Figure 1. Gluteal lesion and surgical debridement on October 3, 2024



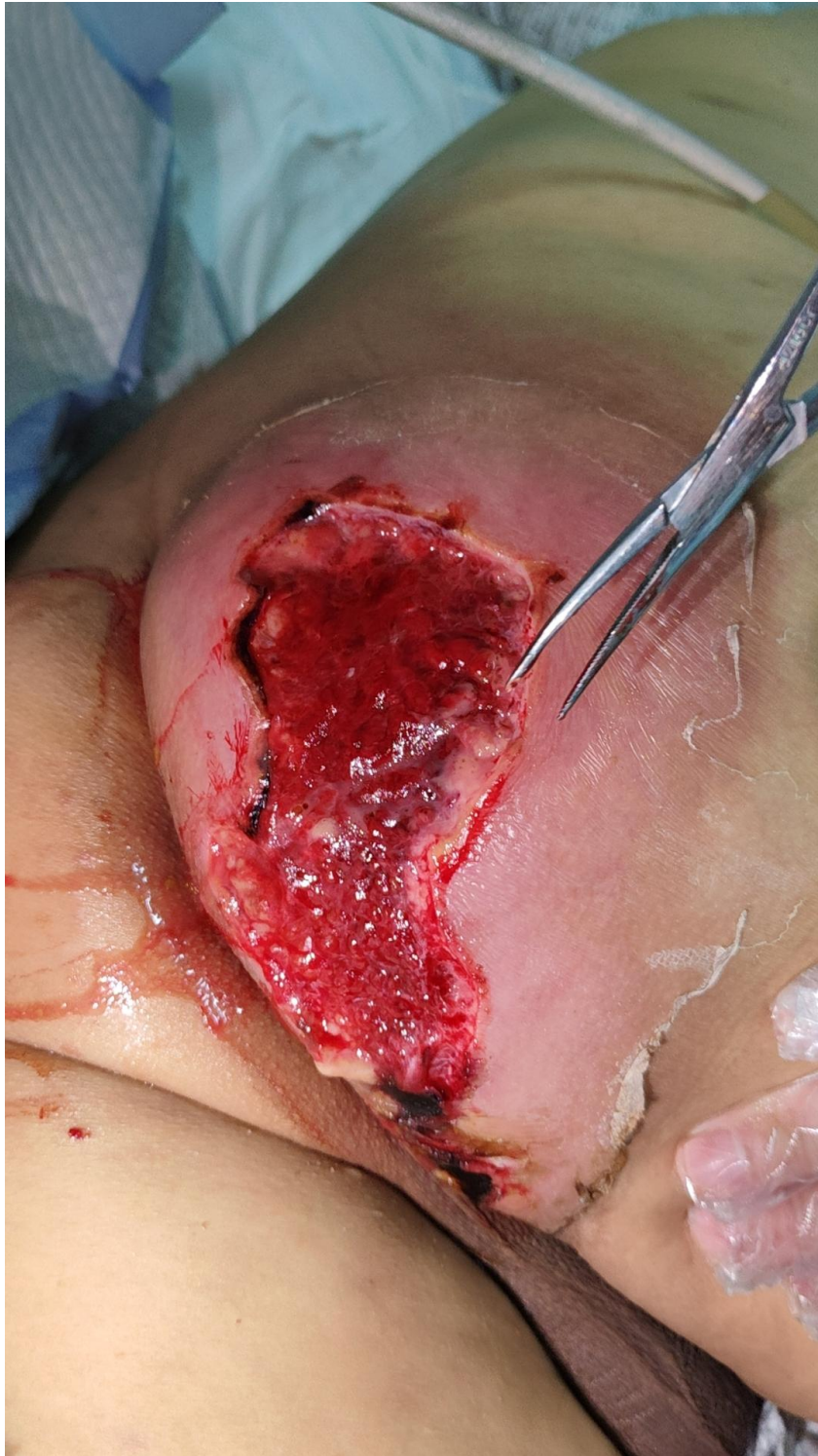
(A) Preoperative appearance of the black necrotic eschar



(B) Partial eschar removal



(C) Debridement in progress



(D) Wound bed after eschar removal, before irrigation, showing necrotic fat



(E) Wound after irrigation, revealing honeycomb-like necrotic fat and purulent drainage



(F) Pus aspirated after vacuum-assisted closure (VSD) negative-pressure application

Figure 1. Gluteal lesion and surgical debridement on October 3, 2024. (A) Preoperative appearance of the black necrotic eschar. (B) Partial eschar removal. (C) Debridement in progress. (D) Wound bed after eschar removal, before irrigation, showing necrotic fat. (E) Wound after irrigation, revealing honeycomb-like necrotic fat and purulent drainage. (F) Pus aspirated after vacuum-assisted closure (VSD) negative-pressure application. Used with patient consent.

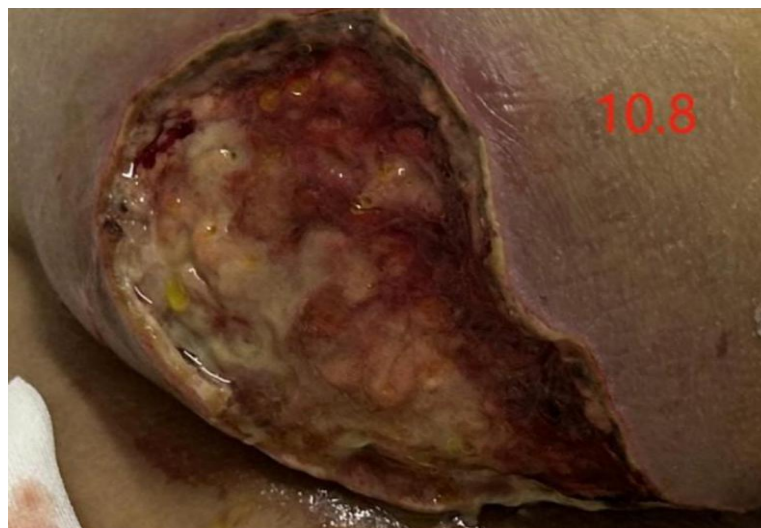
On October 8, repeat debridement was required for ongoing purulence. Pathogen identification relied on targeted gene sequencing (tGS), a metagenomic approach based on a third-generation sequencing platform applied directly to wound secretions, which identified *Staphylococcus aureus*, *Stenotrophomonas maltophilia*, *Candida* species, and *Enterococcus faecium*, while concurrent bacterial culture grew *Klebsiella pneumoniae* and (on a later sample) *Pseudomonas aeruginosa*; the *S. aureus* isolate was subsequently confirmed as methicillin-resistant (MRSA). Antibiotics were escalated to imipenem-cilastatin (1 g every 6 h), tigecycline (50 mg every 12 h), and fluconazole; oral amphotericin B, already begun for intestinal *Candida*, was continued. The VSD was removed on October 10 when the wound

showed purulent exudate and a dark-brown soft slough overlying newly forming pink granulation tissue; management was converted to twice-daily bedside debridement and dressing changes.

Sequential topical wound care and wound evolution

From October 10 onward, wound care was performed approximately twice daily and comprised hydrogen peroxide and normal saline cleansing, povidone-iodine disinfection, debridement of necrotic tissue, and topical antisepsis. Between October 7 and 10, zinc sulfadiazine cream was applied but was associated with increasing purulence and slough (Figure 2A). On October 11, the topical regimen was switched to a traditional Chinese medicine-based ointment, Shangdi Rulekang (Hunan Xiongshi Pharmaceutical Co., Ltd., China), composed of propolis, Ganoderma (lingzhi), Angelica sinensis (danggui), mulberry bark (Cortex Mori), and Sophora flavescens (kushen) in a beeswax and sesame oil base (detailed in the Discussion), applied as an oil-impregnated gauze dressing. Within one day, fresh granulation tissue increased markedly with less necrotic material and a cleaner wound bed (Figure 2B).

Figure 2. Early wound bed evolution under topical antisepsis (October 8-29, 2024)



(A) October 8: purulent exudate and slough during zinc sulfadiazine therapy



(B) October 12: increased granulation and cleaner bed after switching to the Shangdi Rulekang ointment dressing



(C) October 18: thickening granulation and contracting margins



(D) October 29: marginal filiform epithelial projections advancing across the wound, with pigmentation and early re-epithelialization

Figure 2. Early wound bed evolution under topical antisepsis (October 8-29, 2024). (A) October 8: purulent

exudate and slough during zinc sulfadiazine therapy. (B) October 12: increased granulation and cleaner bed after switching to the Shangdi Rulekang ointment dressing. (C) October 18: thickening granulation and contracting margins. (D) October 29: marginal filiform epithelial projections advancing across the wound, with pigmentation and early re-epithelialization.

Serial photography thereafter documented the wound trajectory (Figures 2-3; the complete series of 121 dated photographs spanning approximately five months is provided in Additional file 1). Through late October, granulation thickened, the wound margins contracted, and characteristic filiform projections were observed extending from the skin edges across the wound surface; these projections thickened over consecutive days and were accompanied by marginal pigmentation and advancing epithelialization (Figure 2C-D). On October 30, after the Shangdi ointment stock was exhausted, the topical agent was changed to a chlorhexidine-based antimicrobial ointment (Bake; Quanyu-He-Yi-Gao Ba-Wei-Ke Shi-Hao antibacterial ointment), maintaining a clean, contracting wound. Between November 10 and 11, the family, citing another patient's anecdotal experience, briefly applied eggshell membrane for two days; the wound remained clean with a white surface layer and filiform marginal growth, and the membrane was discontinued. The Shangdi ointment was resumed and continued. During the hospital course the patient also developed a rhinovirus upper-respiratory infection (confirmed by throat-swab tGS) attributed to steroid-related immunosuppression, and a gout flare that resolved with analgesics, urate-lowering therapy, and brief corticosteroids. He was discharged on November 18, 2024 (day 53) with a 15 x 5 cm contracting wound with healthy granulation and clean margins, on oral antidiabetic, antihypertensive, urate-lowering, and neurotrophic medications, with instructions for twice-daily self-dressing changes and weekly blood-count monitoring.

After discharge, the patient continued daily self-administered dressing changes at home, documented by serial photography (Figure 3; Additional file 1). Progressive re-epithelialization, continued marginal contraction, and advancing thin sheets of new epithelium were observed. Topical agents were discontinued on February 8, 2025; the wound remained stable without medication through February 16, 2025, and the patient reported complete healing by March 2025, achieving near-scarless second-intention healing without skin grafting. At long-term follow-up on June 24, 2026 (approximately 16 months after healing), the previously massive wound had epithelialized spontaneously without skin grafting; the area showed an elongated atrophic scar that was flat, without hypertrophic elevation, depression, ulceration, sinus tract, erythema, or exudate, with hypopigmentation over the scar and hyperpigmentation of the surrounding skin, and no scar contracture (Figure 4). The patient remains well, with normal uric acid, satisfactory glycemic and blood pressure control on oral medication, and full unrestricted functional activity.

Figure 3. Post-discharge healing trajectory (November 19, 2024 - February 16, 2025)



(A) November 19, 2024: after dressing change, first days of home care



(B) December 5, 2024: progressive contraction and re-epithelialization



(C) January 2, 2025: continued marginal advancement



(D) January 24, 2025: continued contraction



(E) February 8, 2025: last day of topical agents



(F) February 16, 2025: stable wound without medication (last photographed date)

Figure 3. Post-discharge healing trajectory (November 19, 2024 - February 16, 2025). Representative milestones from daily home dressing changes showing progressive contraction and re-epithelialization; the wound remained

stable after topical agents were discontinued on February 8, 2025, with the last photographed date on February 16, 2025. The complete dated photographic series is provided in Additional file 1.

Figure 4. Long-term follow-up at 16 months (June 24, 2026)



(A) Overview of the healed area



(B) Close-up view

Figure 4. Long-term follow-up at 16 months (June 24, 2026). The previously massive wound healed by spontaneous epithelialization without skin grafting, leaving an elongated atrophic scar that is flat, without hypertrophic elevation, depression, ulceration, sinus tract, erythema, or exudate; hypopigmentation over the scar with hyperpigmentation of the surrounding skin, and no scar contracture. Used with patient consent.

Outcome

The patient survived septic shock with secondary HLH and multi-organ dysfunction and achieved complete wound healing by second intention over approximately five months without skin grafting. Hospital stay was 53 days. Final diagnoses included septic shock (MRSA), polymicrobial soft-tissue infection (MRSA, *K. pneumoniae*, *S. maltophilia*, *C. albicans*) with a giant gluteal wound, secondary HLH, agranulocytosis, anemia, hepatic/renal/cardiac

dysfunction, gastrointestinal bleeding, rhinovirus respiratory infection, anal fissure, type 2 diabetes, gout flare, peripheral neuropathy, and hypertension.

Table 1

Table 1. Selected laboratory values during the first two weeks. H, high; L, low; WBC, white blood cell count; ANC, absolute neutrophil count; IL-6, interleukin-6; PT, prothrombin time; sCD25, soluble interleukin-2 receptor; NT-proBNP, N-terminal pro-B-type natriuretic peptide; ED, emergency department; HLH, hemophagocytic lymphohistiocytosis.

Parameter	Sep 25 (ED)	Sep 26 (admission)	Oct 2 (HLH work-up)	Oct 8 (debridement)	Reference
WBC ($\times 10^9/L$)	1.64 L	1.25 L	2.9 L	9.4	3.5-9.5
ANC	—	0.13 L	—	—	1.8-6.3
Platelets ($\times 10^9/L$)	18 L	11 (nadir 5 on Sep 29)	121	188	125-350
Hemoglobin (g/L)	95 L	89 L	87 L	—	130-175
Procalcitonin (ng/mL)	25.3 H	14.1 H	1.15 H	0.308 H	<0.05
IL-6 (pg/mL)	>5000 H	—	119 H	14.4 H	<7
D-dimer (ng/mL)	2951 H	11651 H	12155 H	2389 H	<500
PT (s)	17.2 H	22.5 H	15.2 H	13.0 H	9.4-12.5
Fibrinogen (g/L)	9.43 H	8.32 H	—	1.73 L	2.0-4.0
Triglycerides (mmol/L)	—	—	2.51 H	—	<1.7
Ferritin (ng/mL)	—	—	>500 H	—	30-400
sCD25 (U/mL)	—	—	3027 H	—	223-710
Albumin (g/L)	24.2 L	23.2 L	—	—	40-55
Total bilirubin ($\mu\text{mol/L}$)	69.9 H	145.1 H	48.5 H	—	5.1-28.0
Creatinine ($\mu\text{mol/L}$)	328.7 H	198.6 H	—	—	57-97
NT-proBNP (pg/mL)	7308 H	10374 H	1314 H	—	<125
Lactate (mmol/L)	2.8 H	3.91 H	—	—	0.5-1.6

Discussion

This case underscores how a seemingly trivial diabetic furuncle can precipitate a catastrophic chain: necrotizing soft-tissue infection, septic shock, secondary HLH, multi-organ dysfunction, and an extensive perianal wound. Several features merit discussion.

First, the concurrence of septic shock and secondary HLH substantially worsens prognosis. Secondary HLH complicating sepsis is easily overlooked because fever, cytopenias, and hyperferritinemia overlap with severe sepsis itself [3,4]. In this patient, falling fibrinogen (rather than the rising fibrinogen typical of sepsis), splenomegaly, hypertriglyceridemia, and a markedly elevated sCD25 were decisive; bone marrow hemophagocytosis was absent, illustrating that a negative marrow does not exclude HLH [3]. Prompt recognition enabled corticosteroid therapy with G-CSF support, coinciding with count recovery. Clinicians managing severe soft-tissue sepsis with disproportionate cytopenias, falling fibrinogen, or hepatosplenomegaly should actively screen for HLH-2004 criteria, including sCD25 and ferritin [4].

Second, source control had to contend with polymicrobial infection (MRSA, *K. pneumoniae*, *S. maltophilia*, and *C. albicans*) in an immunocompromised host. Sequential antimicrobial escalation guided by cultures eventually controlled infection; the combination of MRSA-directed agents, broad Gram-negative coverage, and oral amphotericin B for intestinal *Candida* decolonization reflected the polymicrobial nature of necrotizing diabetic soft-tissue infections complicated by dysbiosis [1,2]. Notably, infection control and wound improvement paralleled HLH-directed immunomodulation and count recovery, suggesting that restoring immune competence was as important as antimicrobial coverage.

Third, the wound itself posed a dilemma. At 18 x 8 cm, full-thickness, and adjacent to the anus, it was a high-contamination, difficult-to-graft wound in a catabolic, immunosuppressed patient. Although in this case the wound area exceeded the dimensions of the available vacuum-assisted closure (VSD) foam, and the perianal location was prone to seal failure, the early negative-pressure drainage played a decisive salvage role: it promptly evacuated the large volume of pus from the deep peri-wound soft tissues, providing timely decompression and drainage of what was effectively a soft-tissue high-pressure abscess, and thereby interrupted the ongoing entry of septic products into the circulation. This early source control was a critical step in halting septic shock. VSD/NPWT is established for complex wounds [5,6], and randomized trials in diabetic foot ulcers have shown NPWT to be superior to advanced moist wound therapy for wound healing [24,25]; early NPWT intervention in trauma and complex wounds has been advocated to reduce infection and promote granulation [26]. Once the deep purulent burden had been drained and the acute septic phase controlled, the device-dependent limitations of NPWT in this oversized, periorificial wound became apparent — partial exposure and air leakage — and management was pragmatically converted to open daily care: repeated sharp debridement, antiseptic cleansing, and sequential topical antiseptics. This sequence illustrates that NPWT's greatest value in such cases may lie in the early salvage drainage of deep septic collections, after which its technical limitations in oversized or periorificial wounds warrant a structured fallback rather than persistence.

Fourth, the traditional Chinese medicine (TCM)-based topical ointment used in this case

warrants detailed description, as it plausibly contributed substantially to the observed healing trajectory. Shangdi Rulekang (Hunan Xiongshi Pharmaceutical Co., Ltd., China) is an ointment developed for pressure-ulcer care, composed of propolis, Ganoderma, Angelica sinensis (danggui), mulberry bark (Cortex Mori), and Sophora flavescens (kushen), among other herbal ingredients, with beeswax and sesame oil jointly serving as the base; its described actions include tissue repair, antimicrobial activity, regulation of wound acid-base and moisture balance, and promotion of granulation and vascular formation, with a fine, easily absorbed texture that is gentle and non-irritating. Applied to the ulcerated wound, the oleaginous base forms a yellow, semi-transparent protective film that shields and moistens the wound surface, reducing opportunities for bacterial contamination, in keeping with the moist-wound-healing principle [17,18]. Through hydration of necrotic tissue, the ointment promotes liquefaction of slough, which loses its adherence to underlying viable tissue and detaches spontaneously at dressing changes, leaving little residue; this autolytic debridement reduces the wound bacterial load while sparing normal tissue and avoiding the repetitive trauma of forced mechanical removal. The preparation keeps the wound consistently moist throughout the phase of necrotic-tissue clearance, minimizes external irritation, and is described as enhancing local microcirculation — properties that together favor rapid granulation growth at the wound base. The individual constituents have documented pharmacology directly relevant to wound repair: propolis has well-characterized antimicrobial and wound-healing properties [33] and has been shown to improve wound healing in a rodent model of diabetes [34]; Angelica sinensis promotes wound healing through keratinocyte and fibroblast activation [35]; Ganoderma lucidum polysaccharides accelerate refractory wound healing [36]; and the lavandulylated flavonoids of Sophora flavescens possess direct antibacterial activity [37]. The broader role of medicinal plants in wound care has been extensively reviewed [38]. In this case, the switch from zinc sulfadiazine cream to this ointment on October 11 was followed within one day by visibly increased granulation and a cleaner wound bed (Figure 2B), and the ointment remained the mainstay of topical care thereafter — a temporal relationship that, together with the constituent pharmacology, supports a contributory therapeutic role. The preparation is registered in China as a Class II disinfection product and randomized trial evidence for this specific product is not yet available; we therefore present its role as a well-supported clinical observation rather than established therapy. The other topical agents were as follows: zinc sulfadiazine cream, a licensed topical antimicrobial, was used between October 7 and 10, during which purulence transiently increased; and Bake, a chlorhexidine-based antimicrobial ointment, was substituted between October 30 and November 12 when the Rulekang stock was exhausted, with the wound remaining clean and contracting throughout. Anecdotal use of eggshell membrane by the family was brief (two days) and is reported transparently as a patient-driven action, not a recommended intervention.

Fifth, the wound-healing biology observed in this case merits detailed description, as the

serial photographs captured a complete second-intention healing sequence in a full-thickness (dermal) wound, and the observed phases are consistent with the established biology of wound repair [8]. Wound healing proceeds through overlapping phases of inflammation, proliferation, and maturation/remodeling [8,9]. In this patient, several recognizable, temporally distinct phenomena were documented. (1) Granulation without central epithelium: after debridement the wound bed filled with granulation tissue while the surface remained devoid of epithelium — a phase we describe as 'granulation growing but no skin growing' — in which only granulation tissue proliferated and no epithelialization occurred over the central wound. Granulation tissue formation involves fibroblast proliferation, extracellular matrix deposition, and neovascularization, with fibroblasts differentiating into contractile myofibroblasts expressing alpha-smooth muscle actin (alpha-SMA), the cells responsible for wound contraction [10,11]. (2) Mucoid filiform projections from the margins transforming into 'flesh columns': at the wound edges, delicate, mucus-like filiform strands first appeared, extending from the surrounding skin toward the wound center; over consecutive days these gradually transformed into stouter, columnar projections ('flesh columns') that served as subcutaneous growth points (anchors) from which advancing epidermis could extend. (3) Epithelial crawling over the granulation: new epidermis did not arise *de novo* within the wound center but migrated inward from the margins, advancing over the granulation bed and relying on the marginal columns as a scaffold. This is consistent with the established mechanism of re-epithelialization, in which keratinocytes at the wound edge undergo phenotype conversion, proliferate, and migrate across the wound surface to restore the epithelial barrier [12,13]; mechanical cues transduced by ion channels such as PIEZO1 regulate keratinocyte migration during this process [14]. Thus granulation and epithelialization were spatially and temporally separated: granulation built the bed, while the margins supplied the epithelium. (4) Color evolution of the advancing edge: the earliest crawling epithelium appeared pale yellow and, as it matured over days, transitioned to white — a change plausibly reflecting progressive keratinization and reduction of the underlying vascular granulation showing through thin, immature epidermis, with the pale-yellow hue corresponding to parakeratotic/immature keratinocytes and the white hue to mature, fully keratinized stratified squamous epithelium. (5) Contraction and re-epithelialization: as marginal epithelium advanced and myofibroblast-mediated contraction reduced the wound area, the defect progressively closed without grafting; transforming growth factor-beta (TGF-beta) is a key driver of both fibroblast-mediated collagen-matrix contraction and myofibroblast differentiation [11,15], and tenascin, an extracellular matrix glycoprotein induced in healing wounds, supports cell migration during this phase [16].

Sixth, the case underscores that moist healing is essential for full-thickness dermal wounds of this size. The principle that occlusive, moist environments accelerate re-epithelialization by preventing desiccation of the denuded dermis and allowing unimpeded keratinocyte migration is well established [17,18]; controlled moisture vapor transmission promotes both re-

epithelialization and wound contraction [19], and occlusive dressings have been associated with more rapid healing and better scar quality than gauze in randomized comparisons [20,21]. If the wound surface is allowed to desiccate, a dry crust (eschar) forms and the underlying tissue progressively necroses, converting a healable wound into a deeper defect [17,22]. The oily (oleaginous) ointment used here served three functions that, in combination, plausibly contributed to the favorable outcome: (i) it maintained wound moisture by forming an occlusive film that prevented desiccation, consistent with the documented benefit of occlusive/moist-environment dressings [17,18,20]; (ii) its oily vehicle created an environment inhospitable to bacterial proliferation, providing a degree of topical antisepsis — relevant because biofilm-associated bacterial colonization impairs wound healing [23] — without the cytotoxicity of harsher antiseptics on proliferating keratinocytes; and (iii) its non-irritant, bland nature avoided chemical stimulation of the wound bed, thereby preventing exuberant or disordered granulation (hypergranulation) that can impede epithelial crawling. Notably, the TCM-based ointment combined this occlusive oleaginous base with herbal constituents whose wound-repair pharmacology is increasingly documented [33-38], so that its vehicle-level and constituent-level actions acted in concert — a synergy consistent with the rapid granulation response observed after its introduction.

Seventh, this case carries a broader translational implication. The decision not to skin-graft was prospective: because the wound margins demonstrated sustained epithelial advancement and contraction, the treating team predicted that second-intention healing was achievable and did not recommend grafting. Some patients with large full-thickness wounds — including certain burn wounds — may therefore not require skin grafting and can achieve complete healing by second intention when conditions are optimized. This does not negate the role of grafting, which remains indicated for wounds that cannot close in a reasonable time [8] and which carries its own morbidity and failure risk in contaminated, immunocompromised, catabolic patients; rather, it demonstrates that under meticulous moist wound care with bland occlusive antisepsis, marginal epithelial crawling supported by granulation-bed flesh columns can re-epithelialize even an 18 x 8 cm defect. Wound chronicity and biofilm burden are key determinants of healing failure [23,27], and standardized assessment tools can monitor healing progress [28]; in this case, the favorable systemic trajectory (infection control and immune recovery) likely reduced the chronicity and biofilm factors that otherwise impair healing [29]. The convergence of several favorable factors — adequate granulation, sustained marginal epithelial advancement, a consistently moist and bacteria-suppressed environment, non-irritant topical care preventing hypergranulation, and patient adherence to daily dressing changes — collectively enabled the near-scarless outcome. Removing any one of these elements would likely have impaired healing; their combination, rather than any single intervention, produced the complete result.

Limitations include the single-patient, retrospective nature; the lack of quantitative

wound-area measurement; and the absence of histopathologic confirmation of the filiform projections, whose interpretation as advancing epithelium rests on longitudinal visual observation rather than biopsy. Nonetheless, the unusually complete serial photographic record (121 dated images spanning approximately five months, supplemented by intra-debridement photographs on October 3 and a 16-month follow-up photograph in June 2026; Additional file 1) provides strong visual evidence of both the wound trajectory and the durable long-term outcome.

Conclusions

A minor diabetic furuncle can trigger septic shock with secondary HLH, multi-organ dysfunction, and a massive perianal wound. When HLH is recognized and treated, polymicrobial infection controlled with culture-guided antimicrobials, and source control achieved through debridement with negative-pressure therapy supplemented by disciplined sequential topical wound care, even an 18 x 8 cm contaminated wound adjacent to the anus may heal completely by second intention without skin grafting. The TCM-based ointment — through its moist-environment maintenance, autolytic debridement of necrotic tissue, barrier protection, and granulation-supporting constituents — contributed importantly to this outcome. This case supports proactive HLH screening in severe soft-tissue sepsis and demonstrates the feasibility of non-grafted healing for selected extensive wounds under meticulous care.

Abbreviations

CT: computed tomography; G-CSF: granulocyte colony-stimulating factor; HLH: hemophagocytic lymphohistiocytosis; IL-6: interleukin-6; MRSA: methicillin-resistant *Staphylococcus aureus*; NPWT: negative-pressure wound therapy; NT-proBNP: N-terminal pro-B-type natriuretic peptide; PCT: prolactin; sCD25: soluble interleukin-2 receptor; tGS: targeted gene sequencing (third-generation sequencing-based metagenomic pathogen detection); VSD: vacuum sealing drainage.

Declarations

Ethics approval and consent to participate: This case report was conducted in accordance with the Declaration of Helsinki and was approved by the Medical Ethics Committee of Chinese PLA General Hospital Hainan Hospital (approval no. S2026-18-02), under the framework of the approved research project “Study on nursing intervention strategies and efficacy evaluation of negative-pressure drainage for skin and soft-tissue infections in hot and humid environments” (grant no. 2025HNYH-PY-04). Written informed consent for participation and publication was obtained from the patient.

Consent for publication: Written informed consent for publication of clinical details and

photographs was obtained from the patient. A copy of the consent form is held by the corresponding author and available on request.

Availability of data and materials: The datasets (de-identified medical records and photographs) supporting this article are available from the corresponding author on reasonable request, subject to patient consent. The complete serial photographic record is provided in Additional file 1.

Competing interests: The authors declare that they have no competing interests.

Funding: This work was supported by the Nursing Research Support Project of Chinese PLA General Hospital Hainan Hospital (grant no. 2025HNYH-PY-04, "Study on nursing intervention strategies and efficacy evaluation of negative-pressure drainage for skin and soft-tissue infections in hot and humid environments"). The funder had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Authors' contributions: SY, FS, and ZYW contributed equally as co-first authors. HLL and PJ contributed equally as co-corresponding authors. SY: conceptualization, methodology, writing – original draft, writing – review & editing, validation; performed the majority of wound dressings, managed the patient's clinical treatment, and curated the clinical images. FS: data curation (image organization), investigation (assisting with wound-care procedures), and contributed to reconstruction of the wound-care timeline. ZYW, FZY, and WJL: investigation, data curation (clinical image acquisition and archiving), and participation in daily clinical management of the patient. PJ: funding acquisition, project administration, obtained ethical approval, provision of critical resources for advanced wound dressings, and writing – review & editing (critical revision of the nursing-protocol section). HLL: supervision, overall clinical guidance, and final approval of the version to be published. All authors read and approved the final manuscript.

Acknowledgements: The authors thank the patient and his family for consent and cooperation, and the nursing staff for meticulous bedside wound care.

Additional material

Additional file 1 (separate documents, provided at submission): Complete serial photographic record of the gluteal wound. A chronological compilation of 121 dated photographs (September 10, 2024 to February 16, 2025) documenting the entire wound evolution from initial furuncle, through necrosis, debridement, negative-pressure therapy, granulation, marginal epithelial advancement, contraction, to near-complete re-epithelialization, plus a long-term follow-up photograph at 16 months (June 24, 2026). Each image is captioned with its date and the topical agent in use. Used with patient consent.

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