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(CASE REPORT)

Scalp necrosis following hair transplantation successfully managed with conservative wound care and adjunctive photobiomodulation: A case report

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Abstract

Hair transplantation is generally safe, but recipient-site scalp necrosis is a rare and potentially severe complication. A 49-year-old heavy smoker developed a 3-cm vertex ulceration two days after hair transplantation and presented one month later with a fibrinous, exudative lesion and only a few visible surviving grafts. Treatment consisted of topical fusidic acid, a hydrocolloid dressing promoting autolytic debridement, a topical copper–zinc cream, and adjunctive light-emitting diode photobiomodulation three times weekly. Complete wound closure was achieved within two weeks, with preservation of several transplanted hairs confirmed by dermoscopy. Early recognition, identification of vascular risk factors, and prompt wound care are essential to prevent scarring and graft loss. Photobiomodulation may represent a useful adjunct, although its specific benefit requires further investigation.

Keywords: Hair Transplantation; Scalp Necrosis; Photobiomodulation; Light-Emitting Diode; Wound Healing; Case Report**.**

1. Introduction

Hair transplantation is an increasingly performed surgical procedure for the management of androgenetic alopecia. Modern techniques involve harvesting follicular units from androgen-independent donor areas and implanting them into recipient sites affected by progressive hair loss. Both follicular unit transplantation and follicular unit excision are generally considered safe when performed with appropriate patient selection, aseptic precautions, and meticulous surgical technique [1,2].

Most adverse events are mild and transient, including pain, edema, pruritus, folliculitis, crusting, postoperative effluvium, and temporary sensory disturbances [1–3]. Recipient-site scalp necrosis is considerably less common but represents one of the most serious complications because it may result in permanent scarring, graft failure, and substantial aesthetic and psychological consequences [4,5].

Its pathogenesis is likely multifactorial. Proposed mechanisms include cumulative microvascular injury during recipient-site creation, excessive incision density, deep or closely spaced implantation, marked tissue tumescence, high concentrations of epinephrine, and pre-existing impairment of tissue perfusion [4–7]. Patient-related factors, including smoking, diabetes mellitus, hypertension, vascular disease, and previous scalp surgery or scarring, may further compromise local circulation [4,5].

We report a case of recipient-site scalp necrosis following hair transplantation in a heavy smoker, with rapid wound closure and partial graft preservation after conservative wound care combined with adjunctive light-emitting diode photobiomodulation.

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2. Case Report

A 49-year-old man presented to our dermatology department with a persistent scalp ulceration following hair transplantation. He had undergone the procedure one month earlier at an outside plastic surgery facility for the treatment of androgenetic alopecia.

The patient reported that the lesion had initially appeared as a tissue defect over the recipient area approximately two days after transplantation. Details regarding the transplantation technique, the number and density of implanted follicular units, the composition of the tumescent anesthesia, the concentration of epinephrine, and the duration of the procedure were unavailable.

His medical history was notable for chronic cigarette smoking for approximately 20 years at a rate of one pack per day. He had no known history of diabetes mellitus, hypertension, peripheral vascular disease, or previous surgery or radiotherapy involving the scalp.

Clinical examination revealed a well-demarcated ulceration of the vertex measuring approximately 3 cm in its greatest diameter. The lesion had an exudative surface and a predominantly fibrinous base. The surrounding skin was erythematous and hyperpigmented. Only a limited number of apparently surviving follicular grafts were visible within and around the ulcerated area (Figure 1). The clinical presentation and postoperative chronology were consistent with recipient-site scalp necrosis following hair transplantation.

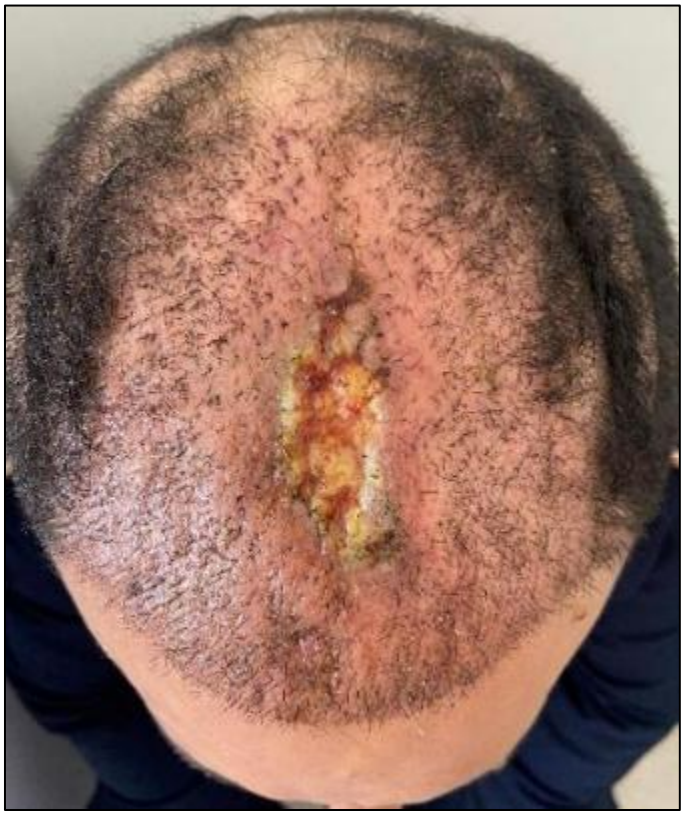

Figure 1 Clinical presentation one month after hair transplantation, showing a well-demarcated ulceration of the vertex with a purulent surface, fibrinous base and surrounding erythematous-hyperpigmented skin.

Management aimed to control local bacterial colonization, remove devitalized tissue, promote wound healing, and preserve as many remaining follicular grafts as possible. Topical fusidic acid was applied twice daily for 10 days. A hydrocolloid dressing was used to maintain a moist wound environment and promote autolytic debridement, while a copper–zinc cream was applied to the perilesional skin. In addition, the patient underwent light-emitting diode photobiomodulation three times weekly.

A favorable response was observed. Complete epithelialization and wound closure were achieved within two weeks of treatment initiation. Several follicular grafts remained visible within the healed area (Figure 2). Dermoscopic

examination showed a healing pink-white background, residual yellowish crusting, and multiple transplanted hair shafts emerging from preserved follicular openings, supporting partial graft survival (Figure 3).

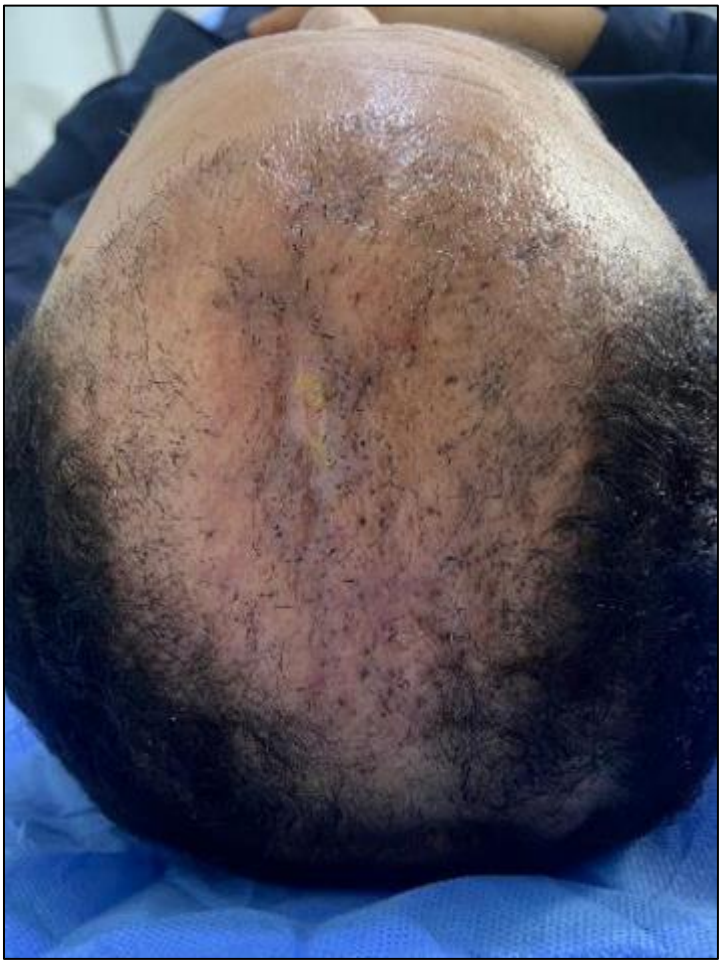

Figure 2 Clinical appearance two weeks after initiation of treatment, showing complete wound closure with preservation of several transplanted hair grafts.

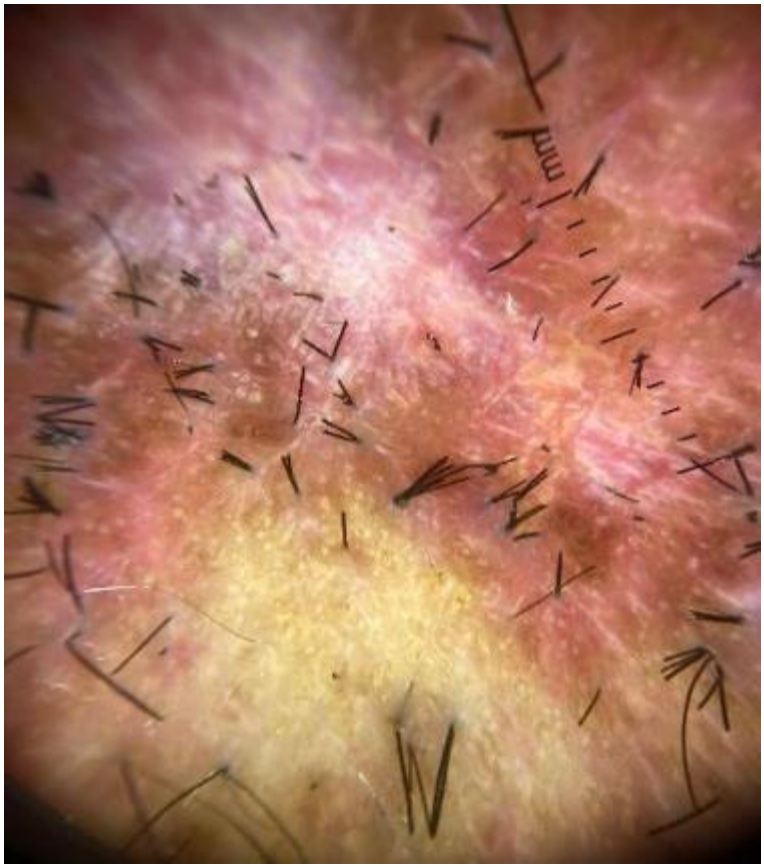

Figure 3 Dermoscopic examination two weeks after initiation of treatment, showing a healing pink-white background, residual yellowish crusting and several transplanted hair shafts emerging from preserved follicular openings.

3. Discussion

Recipient-site scalp necrosis is an uncommon but potentially disfiguring complication of hair transplantation. Its true incidence remains uncertain because the available evidence consists mainly of isolated case reports and small retrospective series. In a series of more than 10,000 hair transplantation procedures, Chen et al. identified three cases occurring at their center and described an additional externally referred patient [4]. A more recent retrospective study of 18 affected patients confirmed that graft failure and residual scarring may be frequent consequences of established necrosis [5].

The central scalp and vertex may be particularly vulnerable when local perfusion is compromised. During transplantation, numerous small incisions are created within the recipient bed. Excessive incision density, overly deep recipient-site creation, or extensive dense packing may collectively damage the microvascular network and reduce tissue perfusion [2,4–6]. Experimental vascular mapping has also suggested that the risk of ischemic damage may be influenced by the number of recipient sites per unit area and by instrument-related tissue trauma [7].

Vasoconstrictors represent another potential contributing factor. Epinephrine is commonly used during hair transplantation to reduce bleeding and prolong the effect of local anesthesia. However, excessive concentrations or repeated infiltration may cause sustained vasoconstriction and exacerbate tissue ischemia. Yildiz et al. reported recipient-site necrosis following tumescent infiltration containing adrenaline [6]. The risk may be amplified when pharmacological vasoconstriction is combined with high graft density or underlying vascular impairment.

Chronic smoking was the main identifiable risk factor in our patient. Nicotine-induced vasoconstriction, endothelial dysfunction, reduced tissue oxygenation, and impaired inflammatory and reparative responses may adversely affect surgical wound healing. In the context of hair transplantation, smoking has repeatedly been cited as a potential risk factor for recipient-site necrosis, together with diabetes, hypertension, vascular disease, scarred recipient skin, and previous surgery [1,4,5]. The onset of the lesion two days after surgery and its localization within the grafted area

support an ischemic mechanism. Nevertheless, the respective contributions of the surgical technique, recipient-site density, and epinephrine exposure could not be established because procedural details were unavailable.

Early warning signs may include disproportionate pain, dusky or violaceous discoloration, persistent pallor, crusting, and progressive eschar formation. Necrosis may initially appear limited but can extend in surface area and depth when diagnosis or appropriate wound care is delayed [4]. In the series by Chen et al., patients treated promptly experienced confinement of the necrotic area, healing within approximately two to three weeks, and partial survival of follicular grafts. In contrast, delayed or inappropriate initial management was associated with deeper tissue damage, delayed healing, and complete graft loss [4].

No standardized treatment guidelines are currently available. Management should be adapted to the stage and depth of the lesion and to the presence or absence of secondary infection. Proposed measures include close clinical surveillance, avoidance of additional pressure or vasoconstriction, appropriate cleansing, selective removal of devitalized tissue, moist wound-healing dressings, and antimicrobial treatment when infection is clinically suspected or microbiologically documented [1,4]. Topical nitroglycerin has been reported for impending ischemia before irreversible necrosis develops, but the supporting evidence remains limited, and its use requires caution because of potential systemic adverse effects [8].

Our patient was evaluated one month after the procedure, when ulceration and tissue necrosis were already established. Treatment therefore focused on local wound care, autolytic debridement, control of bacterial colonization, and preservation of the remaining follicular grafts rather than reversal of acute ischemia. Complete wound closure was obtained within two weeks, with preservation of several grafts. This favorable outcome may have been related to the relatively limited size and depth of the lesion and to the persistence of viable tissue within the recipient area.

Photobiomodulation was used as an adjunctive intervention. It involves the delivery of nonthermal light, generally within the red or near-infrared spectrum, to modulate cellular activity. Proposed effects include modulation of mitochondrial cytochrome c oxidase activity, increased adenosine triphosphate production, transient regulation of reactive oxygen species and nitric oxide, and downstream effects on inflammation, cellular proliferation, migration, and tissue repair [9,10].

Photobiomodulation has been investigated in wound healing and hair disorders and may enhance tissue repair and support follicular activity [9–11]. However, clinical studies specifically evaluating photobiomodulation for scalp necrosis following hair transplantation are lacking. Moreover, our patient simultaneously received topical antimicrobial treatment, hydrocolloid dressings, and local wound care. Therefore, a direct causal relationship between photobiomodulation and accelerated wound closure or graft preservation cannot be established. Its role in this case should be considered potentially supportive rather than curative.

4. Conclusion

Recipient-site scalp necrosis is a rare but potentially serious complication that may result in permanent scarring and complete loss of transplanted follicular units. Preoperative identification of risk factors, particularly heavy smoking and metabolic or vascular comorbidities, is essential. Careful control of recipient-site density, tissue trauma, tumescence, and epinephrine exposure may reduce the risk of ischemic injury.

Postoperative pain, persistent discoloration, crusting, or tissue breakdown should prompt immediate evaluation. In limited lesions, appropriate wound care, autolytic debridement, and control of secondary bacterial colonization may lead to rapid closure and partial graft preservation. Photobiomodulation may represent a useful adjunctive treatment, although controlled studies are required to determine its specific benefit.

Compliance with ethical standards

Acknowledgments

The authors have no acknowledgements to declare.

Disclosure of conflict of interest

The authors declare that they have no conflicts of interest regarding the publication of this paper.

Statement of informed consent

Written informed consent was obtained from the patient for publication of this case report and the accompanying clinical and dermoscopic images. All procedures performed were in accordance with the ethical standards of the institutional and national research committee and with the principles of the Declaration of Helsinki.

References

[1] Kerure AS, Patwardhan N. Complications in hair transplantation. J Cutan Aesthet Surg. 2018;11(4):182-189. doi:10.4103/JCAS.JCAS_125_18.

[2] Garg AK, Garg S. Complications of hair transplant procedures: causes and management. Indian J Plast Surg. 2021;54(4):477-482. doi:10.1055/s-0041-1739255.

[3] Avram MR, Rogers NE, Watkins S. Side-effects from follicular unit extraction in hair transplantation. J Cutan Aesthet Surg. 2014;7(3):177-180. doi:10.4103/0974-2077.146681.

[4] Chen J, Qu Q, Guo Y, Hu Z, Li X, Fan Z, et al. Recipient site scalp necrosis: a rare postoperative complication of hair transplantation. J Cosmet Dermatol. 2024;23(2):622-629. doi:10.1111/jocd.16017.

[5] Ceran F. Recipient site necrosis after follicular unit excision technique for hair transplantation: evaluation of 18 patients. Aesthet Plast Surg. 2024;48(21):3735-3740. doi:10.1007/s00266-024-04305-6.

[6] Yildiz H, Ercan E, Alhan D, Sezgin M. Recipient site necrosis after tumescent infiltration with adrenaline in hair transplantation. Acta Dermatovenerol Croat. 2015;23(3):233-234.

[7] Balik O. Determination of scalp vascular topography by laser Doppler flowmetry and comparison of vascular damage by different blades in hair transplantation. J Craniofac Surg. 2023;34(8):e803-e806. doi:10.1097/SCS.0000000000009765.

[8] Abbas Z, Kufi H. Prevention of impending scalp necrosis by topical nitroglycerine in hair transplant surgery. J Cosmet Dermatol. 2021;20(3):906-910. doi:10.1111/jocd.13659.

[9] Mosca RC, Ong AA, Albasha O, Bass K, Arany P. Photobiomodulation therapy for wound care: a potent, noninvasive, photoceutical approach. Adv Skin Wound Care. 2019;32(4):157-167. doi:10.1097/01.ASW.0000553600.97572.d2.

[10] Dompe C, Moncrieff L, Matys J, Grzech-Leśniak K, Kocherova I, Bryja A, et al. Photobiomodulation: underlying mechanism and clinical applications. J Clin Med. 2020;9(6):1724. doi:10.3390/jcm9061724.

[11] Hamblin MR. Photobiomodulation for the management of alopecia: mechanisms of action, patient selection and perspectives. Clin Cosmet Investig Dermatol. 2019;12:669-678. doi:10.2147/CCID.S184979.