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Review Article

Dystrophic epidermolysis bullosa: a comprehensive review of therapeutic strategies and the role of skin grafting in wound management and tissue regeneration

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ABSTRACT

Dystrophic epidermolysis bullosa (DEB) is a severe inherited disorder caused by COL7A1 mutations, leading to deficient type VII collagen (COL7), chronic wounds, and fibrosis. Skin grafting-autologous, allogeneic, or bioengineered-offers potential for wound stabilization. This review evaluates grafting efficacy, limitations, and future directions. A systematic literature analysis (PubMed, Embase, Cochrane) highlighted clinical trials and mechanistic studies. Autologous grafts reduce wound burden but face donor-site fragility; allogeneic grafts provide temporary coverage but risk rejection. Bioengineered substitutes (e.g., gene-corrected autografts) show promise for COL7A1 restoration. While grafting remains palliative, gene-edited (e.g., CRISPR-Cas9) and tissue-engineered approaches may transform DEB care, necessitating multicenter trials for protocol standardization.

Keywords: Dystrophic epidermolysis bullosa, COL7A1 mutations, Skin grafting, Autologous grafts, Allogeneic grafts, Bioengineered skin substitutes

INTRODUCTION

Dystrophic epidermolysis bullosa (DEB), a subset of inherited epidermolysis bullosa (EB), is a debilitating dermatological condition characterized by extreme mucocutaneous fragility due to pathogenic variants in the COL7A1 gene. This gene encodes type COL7, the primary component of anchoring fibrils critical for dermal-epidermal adhesion. Its deficiency results in subepidermal blistering following minor trauma, progressing to chronic wounds, mutilating scarring, and a high risk of metastatic squamous cell carcinoma.[1,2]

Current DEB management is predominantly supportive, emphasizing wound care, infection prevention, and nutritional support. However, the recalcitrant nature of DEB-associated ulcers necessitates advanced interventions, among which skin grafting has emerged as a viable strategy to facilitate wound closure and mitigate complications.[2,3]

Autologous skin grafts, though limited by donor-site morbidity in DEB patients, offer permanent coverage for localized defects. Allogeneic grafts (e.g., cadaveric skin) provide temporary biological dressings but lack long-term

integration. Recent breakthroughs in bioengineered skin-such as composite cultured autografts (CCS) and *ex vivo* gene-corrected epidermal sheets-hold transformative potential by addressing the underlying COL7 deficiency.[4]

This review synthesizes contemporary evidence on skin grafting modalities in DEB, analyzing their clinical outcomes, immunological challenges, and integration with novel therapies like gene editing and protein replacement. By evaluating the state of the art, we aim to delineate a roadmap for optimizing graft-based therapies in this devastating disorder.[4]

DEB

DEB is a severe, genetically inherited dermatological disorder characterized by profound mucocutaneous fragility, recurrent blister formation, and chronic wound development secondary to mutations in the COL7A1 gene, which encodes type COL7. This structural protein is indispensable for the formation of anchoring fibrils at the dermal-epidermal junction, and its deficiency or dysfunction results in mechanical instability, leading to sublamina densa tissue separation upon minimal trauma. The clinical spectrum of DEB ranges from milder, localized presentations to severe, generalized recessive DEB (RDEB), which is associated with life-threatening complications, including progressive fibrosis, pseudo-syndactyly, malnutrition due to esophageal strictures, and an elevated risk of aggressive cutaneous squamous cell carcinomas (cSCCs).[4,5]

The management of DEB remains predominantly palliative, focusing on meticulous wound care, infection prevention, analgesia, and nutritional support. However, the chronic, non-healing wounds that typify this condition often prove refractory to conventional therapies, necessitating more advanced interventions to promote epithelialization, reduce infection risk, and improve quality of life. Among these interventions, skin grafting has emerged as a critical therapeutic modality, offering both temporary and, in some cases, long-term stabilization of cutaneous defects.[4,5]

Historically, autologous split-thickness skin grafts (STSGs) have been utilized to address persistent ulcers in DEB patients, leveraging the patient's own keratinocytes to achieve permanent wound coverage. However, the inherent fragility of donor sites in DEB often limits graft availability and increases the risk of iatrogenic lesion formation. Allogeneic skin grafts, derived from cadaveric or living donors, serve as temporary biological dressings, providing wound protection and facilitating a favorable healing microenvironment, albeit without long-term engraftment due to immune-mediated rejection. Recent advancements in regenerative medicine have introduced bioengineered skin substitutes, including composite cultured skin (CCS) and tissue-engineered autografts, which aim to circumvent donor-site limitations while promoting sustained wound closure.[5,6]

Perhaps the most promising developments in this field involve the integration of molecular therapies with surgical approaches. Gene-corrected epidermal grafts, generated through *ex vivo* transduction of autologous keratinocytes with functional COL7A1, have demonstrated potential in preclinical and early-phase clinical trials, offering not just symptomatic relief but also addressing the underlying molecular defect. Additionally, CRISPR-Cas9-based gene editing and protein replacement strategies are being explored to enhance graft viability and COL7 deposition. Despite these innovations, challenges persist, including immune compatibility, long-term graft stability, and scalability for widespread clinical application.[6,7]

Given the progressive and debilitating nature of DEB, the exploration of skin grafting techniques-both traditional and novel-represents a crucial avenue for improving patient outcomes. This review seeks to comprehensively analyze the evolution, current applications, and future directions of skin grafting in DEB, emphasizing its role in wound management, tissue regeneration, and the broader context of emerging molecular therapies.[7,8]

DIAGNOSTIC METHODS FOR DEB: A MULTIMODAL APPROACH

The accurate diagnosis of DEB necessitates a comprehensive, multimodal approach integrating clinical, histopathological, immunohistochemical, ultrastructural, and molecular genetic analyses. Given the phenotypic overlap between DEB and other subtypes of EB, as well as within the spectrum of DEB itself-ranging from mild dominant DEB (DDEB) to severe RDEB-a systematic diagnostic workup is essential for precise classification, prognostication, and therapeutic planning.[8]

CLINICAL EVALUATION

The initial diagnostic assessment relies heavily on meticulous clinical examination, with particular attention to the distribution and severity of cutaneous and mucosal lesions. DEB is characterized by trauma-induced blistering that heals with atrophic scarring, milia formation, and, in severe cases, progressive contractures and pseudosyndactyly. Extracutaneous manifestations, including esophageal strictures, corneal erosions, and genitourinary involvement, further support the diagnosis. A detailed family history is crucial to distinguish between autosomal dominant and recessive inheritance patterns, though sporadic mutations may also occur.[8]

HISTOPATHOLOGICAL AS WELL AS THE IMMUNOHISTOCHEMICAL ANALYSIS

Light microscopy of a freshly induced blister can reveal dermal-epidermal separation below the lamina densa, a hallmark of DEB. However, this finding alone is nonspecific, necessitating further characterization through immunohistochemistry (IHC). Staining for type COL7 and

other basement membrane zone (BMZ) proteins, such as laminin-332 and type IV collagen, helps differentiate DEB from other EB subtypes. In DEB, COL7 immunostaining is typically absent or markedly reduced in severe RDEB, while it may be present but attenuated or abnormally distributed in DDEB.[9]

TRANSMISSION ELECTRON MICROSCOPY

TEM remains the gold standard for confirming the level of blister formation and assessing anchoring fibril morphology. In DEB, TEM demonstrates cleavage beneath the lamina densa, with variable degrees of anchoring fibril reduction, fragmentation, or complete absence depending on disease severity. Quantitative assessment of anchoring fibril density can further aid in subclassifying DEB, though this technique requires specialized expertise and is less accessible in routine clinical practice.[9]

GENETIC TESTING AND MOLECULAR ANALYSIS

Definitive diagnosis of DEB relies on the identification of pathogenic variants in the COL7A1 gene, which encodes type COL7. Next-generation sequencing (NGS), including whole-exome sequencing (WES) and targeted gene panels, has become the preferred method for detecting mutations, offering high sensitivity and the ability to identify novel variants. Sanger sequencing may be employed for familial variant confirmation. Genetic testing not only confirms the diagnosis but also facilitates genetic counseling, prenatal testing, and, increasingly, personalized therapeutic strategies such as gene therapy or COL7A1-targeted interventions.[9,10]

ADVANCED AND EMERGING DIAGNOSTIC MODALITIES

Recent advances in proteomic and transcriptomic profiling have enabled deeper mechanistic insights into DEB pathophysiology, potentially identifying biomarkers for disease progression and treatment response. Additionally, non-invasive imaging techniques, such as reflectance confocal microscopy (RCM) and optical coherence tomography (OCT), are being explored for real-time assessment of skin integrity and monitoring therapeutic efficacy in clinical trials.[11]

PRE-AND POST-THERAPEUTIC ASSESSMENT IN SKIN GRAFTING

In cases where skin grafting is considered for wound management, pre-interventional diagnostic rigor is paramount. Biopsies from both affected and potential donor sites may be evaluated for COL7 expression and structural integrity to predict graft viability. Post-grafting, immunohistochemical and molecular analyses can assess COL7 restoration in gene-corrected autografts, while non-invasive imaging may track wound re-epithelialization and long-term graft stability.[11]

The diagnosis of DEB requires a layered, interdisciplinary approach, combining traditional histopathology with cutting-edge molecular genetics. As therapeutic strategies-particularly skin grafting and regenerative medicine-continue to evolve, precise diagnostic frameworks will be indispensable for patient selection, intervention tailoring, and outcome optimization.[11]

THERAPEUTIC MODALITIES FOR DEB: CURRENT PARADIGMS AND EVOLVING STRATEGIES IN CUTANEOUS RECONSTRUCTION

The management of DEB constitutes a formidable clinical challenge, necessitating a multidisciplinary therapeutic approach aimed at mitigating blister formation, promoting wound healing, preventing complications, and improving quality of life. Given the molecular pathogenesis rooted in COL7A1 mutations and the consequent deficiency of functional type COL7, therapeutic interventions span palliative wound care, surgical reconstruction, and emerging molecular therapies, with skin grafting occupying a pivotal role in severe cases.[11]

SUPPORTIVE WOUND MANAGEMENT AND LOCAL THERAPIES

The cornerstone of DEB treatment remains meticulous wound care to prevent infection, reduce pain, and facilitate re-epithelialization. Non-adherent, silicone-based dressings are employed to minimize trauma during dressing changes, while antimicrobial topical agents, such as silver sulfadiazine and polyhexanide, help control bacterial colonization in chronic wounds. Advanced dressings incorporating collagen matrices or hyaluronic acid scaffolds may enhance granulation tissue formation in recalcitrant ulcers. Additionally, regular debridement of necrotic tissue, often performed under analgesia or sedation due to patient fragility, is crucial to optimize the wound bed for potential grafting.[12]

SYSTEMIC PHARMACOLOGICAL INTER-VENTIONS

Although no targeted systemic therapy currently reverses the underlying COL7 deficiency, several adjunctive treatments aim to modulate disease progression. Oral anti-inflammatory agents, including corticosteroids and tetracycline-class antibiotics, are sometimes used to reduce blistering and inflammation, albeit with limited evidence. More recently, fibroblast growth factor (FGF) and epidermal growth factor (EGF) analogs have been explored to stimulate keratinocyte proliferation and wound closure. Pain management, often requiring opioids and neuropathic agents, is integral to patient care, alongside nutritional support to counteract the catabolic state induced by chronic wounds and mucosal involvement.[12]

SURGICAL INTERVENTIONS: SKIN GRAFTING IN DEB

For extensive, non-healing wounds, surgical intervention with skin grafts represents a critical therapeutic option. Autologous STSGs are frequently utilized, harvesting keratinocytes and partial dermis from donor sites, typically less affected areas. However, donor-site morbidity in DEB patients poses significant limitations, as these sites often exhibit delayed healing and secondary blistering. Meshed grafts may expand coverage but can lead to hypertrophic scarring in this population.[12]

Allogeneic skin grafts, derived from cadaveric donors, serve as temporary biological dressings, providing wound protection and reducing pain while facilitating autologous epithelial migration. While immunologically rejected over time, they are valuable in acute settings to stabilize severe erosions. Cryopreserved allografts, amniotic membranes, and acellular dermal matrices have also been employed with varying success, offering varying degrees of biocompatibility and wound bed preparation.[12]

BIOENGINEERED SKIN SUBSTITUTES AND TISSUE-ENGINEERED CONSTRUCTS

The advent of bioengineered skin substitutes has revolutionized DEB wound management. Bilayered living cellular constructs (BLCCs), composed of allogeneic fibroblasts and keratinocytes in a collagen matrix, provide temporary wound coverage and stimulate host cell recruitment. More advanced autologous CCS grafts, generated by expanding patient-derived keratinocytes *ex vivo*, offer permanent wound closure with reduced immunogenic risk. However, these constructs often lack functional COL7, limiting long-term stability in DEB. [13]

GENE THERAPY AND GENETICALLY CORRECTED GRAFTS

The most transformative advancements in DEB treatment involve gene-editing strategies to restore COL7 expression. *Ex vivo* gene therapy, wherein autologous keratinocytes are transduced with viral vectors carrying functional COL7A1, has demonstrated promising results in clinical trials, with some patients achieving sustained wound healing. CRISPR-Cas9-based approaches are being investigated for precise genomic correction, while intradermal injection of COL7-expressing fibroblasts may serve as a less invasive alternative. [13]

Intravenous or intradermal recombinant COL7 replacement is under exploration, aiming to systemically restore anchoring fibrils. Additionally, read-through compounds for nonsense mutations and chaperone-mediated therapies for misfolded COL7 are being studied as potential disease-modifying agents.[13]

The therapeutic landscape for DEB is rapidly evolving, with skin grafting remaining a cornerstone for severe wound management, while gene and protein-based therapies hold promise for addressing the root molecular defect. A tailored, multimodal approach-integrating surgical, bioengineered, and molecular strategies-is essential to optimize outcomes for this devastating disorder.[14]

SURGICAL TECHNIQUE FOR SKIN GRAFTING IN DEB: A DETAILED PROCEDURAL APPROACH

The application of skin grafting techniques in patients with DEB requires meticulous surgical planning and execution due to the inherent fragility of the integumentary system in these individuals. The procedure demands an interdisciplinary approach involving dermatologists, plastic surgeons, anesthesiologists, and wound care specialists to optimize outcomes while minimizing iatrogenic trauma. The technical aspects can be divided into preoperative preparation, intraoperative execution, and postoperative management, each requiring specialized considerations unique to this patient population.[14]

Preoperative evaluation begins with comprehensive patient assessment, focusing on nutritional status, coagulation profile, and cardiopulmonary function, as many DEB patients present with multisystem involvement. The selection of donor sites represents a critical decision point, as traditional harvest locations may be compromised by disease activity. Typically, less traumatized areas such as the upper back or lateral thighs are preferred, though even these sites may demonstrate subclinical fragility. Preoperative imaging with reflectance confocal microscopy can help identify areas with relatively preserved dermal-epidermal junction integrity. Recipient site preparation involves gentle debridement of necrotic tissue using atraumatic techniques, often employing hydrotherapy with saline-soaked gauze rather than sharp dissection to prevent wound extension.[15]

Intraoperative management presents unique challenges, beginning with careful patient positioning on pressure-relieving surfaces to prevent new blister formation. Anesthetic considerations are paramount, as endotracheal intubation carries significant risk of mucosal injury. The use of total intravenous anesthesia with careful airway management is preferred, often utilizing smaller-than-standard endotracheal tubes and abundant lubrication. For graft harvest, the dermatome setting must be adjusted to account for the reduced dermal thickness in DEB patients, typically employing an ultra-thin (0.1-0.2 mm) split-thickness graft to balance adequate donor site healing with graft viability. The harvesting technique itself requires modified handling, using non-toothed forceps and skin hooks with minimal tension to prevent iatrogenic injury to both the graft and donor site.[15]

Graft preparation and application involve specialized techniques to account for the abnormal wound bed in DEB. The graft may be meshed at a low expansion ratio (1:1.5) to allow for drainage while minimizing interstices that

could lead to healing complications. Some centers advocate for the use of non-meshed grafts to provide complete coverage, particularly when dealing with areas prone to contracture. The graft is carefully applied to the recipient site using fibrin sealants rather than sutures or staples to minimize trauma. A multilayer dressing approach is then employed, beginning with non-adherent silicone contact layers, followed by absorbent secondary dressings, and finally a stabilizing outer layer secured with gentle compression wraps rather than the adhesive tapes.[15,16]

Postoperative care represents perhaps the most critical phase in the grafting process. Dressings are typically left in place for an extended period (5-7 days) to minimize disturbance to the fragile graft-bed interface. When dressing changes are necessary, they are performed under analgesia and often with the aid of soaking solutions to prevent mechanical disruption. Monitoring for complications requires vigilance for both graft loss and donor site morbidity, with particular attention to signs of infection or hematoma formation. Pain management protocols must be aggressive yet carefully titrated to avoid respiratory depression in these vulnerable patients. Long-term follow-up focuses on assessing graft integration, monitoring for contracture development, and evaluating functional outcomes through standardized measures such as the Birmingham EB severity score.[17]

The technical nuances of skin grafting in DEB continue to evolve with advancements in surgical technology and wound care science. The emergence of laser-assisted graft harvesting, robotic surgical platforms, and smart dressings with embedded sensors for continuous wound monitoring may further refine the procedural approach. However, the fundamental principles of atraumatic technique, individualized patient assessment, and multidisciplinary care remain the cornerstone of successful outcomes in this challenging patient population.[18]

CONCLUSION

DEB represents one of the most formidable challenges in dermatological and surgical practice, characterized by profound mucocutaneous fragility and relentless wound progression secondary to type COL7 deficiency. This comprehensive review has elucidated the critical role of skin grafting techniques as both a palliative measure and potential disease-modifying intervention in the multidisciplinary management of this devastating genodermatosis.

The current evidence underscores that while conventional autologous and allogeneic skin grafts provide immediate wound coverage and temporary symptomatic relief, their long-term efficacy remains constrained by the inherent biological limitations of DEB-affected skin, including donor site morbidity, graft fragility, as well as the persistent underlying molecular defect.

The advent of bioengineered skin substitutes and genetically modified autografts has ushered in a new era of therapeutic possibilities, bridging the gap between surgical wound management and molecular correction. These advanced grafting modalities demonstrate particular promise in addressing the dual challenges of wound closure and partial restoration of the dermal-epidermal junction's structural integrity. However, significant barriers persist in terms of scalability, cost-effectiveness, and long-term graft stability, particularly in the context of the patient's systemic disease burden and propensity for malignant transformation in chronic wounds.

The technical execution of skin grafting in DEB demands an unparalleled level of surgical precision and perioperative care, necessitating specialized centers of excellence with multidisciplinary teams. From micro-thin graft harvesting techniques to fibrin-secured application and sophisticated postoperative monitoring, each procedural step requires meticulous adaptation to the patient's unique cutaneous vulnerability. Emerging technologies in robotic surgery, laser-assisted grafting, and smart wound dressings may further refine these approaches, though their accessibility remains limited in many clinical settings.

Looking forward, the integration of skin grafting with emerging molecular therapies-including CRISPR-based gene editing, protein replacement strategies, and stem cell therapies-presents the most promising avenue for comprehensive DEB management. The ideal therapeutic paradigm will likely combine immediate wound stabilization through optimized grafting techniques with long-term molecular correction, creating a synergistic effect that addresses both symptoms and disease pathogenesis. This dual approach may ultimately transform DEB care from purely supportive to potentially curative, particularly if achieved during early disease stages before irreversible complications develop.

In conclusion, while skin grafting remains an indispensable tool in the current therapeutic arsenal for DEB, its future role will increasingly depend on successful integration with groundbreaking molecular therapies. Continued research investment, standardized outcome measures, and international collaborative efforts are essential to optimize existing techniques and develop next-generation solutions. The ultimate goal remains not merely wound closure, but the restoration of functional skin architecture and quality of life for patients burdened by this devastating condition. As we stand at the crossroads of surgical innovation and molecular medicine, the management of DEB serves as a compelling example of how traditional surgical techniques can evolve to meet the challenges of genetic skin disorders in the precision medicine era.

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