Dura Mater Healing and Regeneration: A Narrative Review of Physiological and Molecular Mechanisms

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Abstract. The dura mater is the outermost meningeal layer of the central nervous system and plays an essential role in neural protection and cerebrospinal fluid homeostasis. Impaired dural healing after trauma or neurosurgical intervention may result in cerebrospinal fluid leakage, pseudomeningocele, infection, pathological adhesion, and delayed wound recovery. This focused narrative review summarizes the physiological, cellular, and molecular mechanisms involved in dura mater healing, with emphasis on their relevance to dural substitute design. Dural repair occurs through overlapping phases of hemostasis, inflammation, proliferation, neodura formation, and remodeling. Early sealing is initiated by platelet activation, fibrin clot formation, and provisional matrix development. The subsequent inflammatory response may support constructive repair when appropriately regulated, but persistent macrophage activation, foreign-body reaction, and excessive fibrotic signaling may impair regeneration. Fibroblast migration, angiogenesis, extracellular matrix deposition, and collagen remodeling contribute to functional neodura formation. Key pathways, including nuclear factor-kappa B-mediated inflammation, transforming growth factor-beta/Smad-related fibrosis, vascular endothelial growth factor-associated angiogenesis, and matrix metalloproteinase/tissue inhibitor of metalloproteinase-mediated matrix turnover, may influence the quality of repair. Therefore, successful dural substitutes should be evaluated not only by immediate watertight closure, but also by immune modulation, fibrovascular integration, organized remodeling, anti-adhesion performance, and long-term restoration of meningeal function.

Keywords: Dura mater, dural healing, neodura, extracellular matrix, tissue remodeling.

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1 Introduction

The dura mater is the outermost meningeal layer and is essential for maintaining cerebrospinal fluid (CSF) homeostasis and protecting neural structures [1-3]. Failure of dural closure may cause CSF leakage, pseudomeningocele, meningitis, wound breakdown, and impaired neural protection. In a recent single-center spinal surgery cohort, intraoperative dural injury occurred in 176 of 4,014 patients (4.3%), and postoperative CSF leakage occurred in 22 of those 176 cases (12.5%) [4]. Danish et al. reported different complication profiles between acellular human dura and bovine collagen matrix in a paediatric Chiari decompression cohort. The DuraGen group had pseudomeningocele, wound infection and cerebrospinal fluid (CSF) leakage in 8.9%, 3.6% and 1.8% of cases, respectively. The AlloDerm group had the corresponding rates of 11.1%, 2.2% and 2.2% [5]. These figures highlight that the clinical consequences of dural failure depend on the surgical population, anatomic setting, and repair material.

Dural substitutes are used when primary watertight closure is not feasible. Autologous grafts remain biologically attractive but are limited by donor-site morbidity and availability. Allogeneic and xenogeneic materials may provide a collagen-rich matrix but raise concerns regarding immunogenicity, degradation behavior, and disease transmission. Synthetic substitutes offer mechanical strength and tunable architecture, yet may provoke poor integration, fibrosis, or foreign-body reactions if the host response is not controlled [1-3,6]. Therefore, modern dural reconstruction should be evaluated not only as mechanical defect coverage but also as a biological process involving immune regulation, fibrovascular ingrowth, extracellular matrix (ECM) remodeling, and prevention of pathological adhesion.

Emerging evidence further suggests that dural healing is not mediated exclusively by mature fibroblasts and inflammatory cells. Experimental models have shown that Dura-derived cells have the potential to act like mesenchymal progenitor cells. Prx1+ and Hic1+ mesenchymal progenitor cell populations have been found in the epidural fat and murine dura mater, where they help to repair dural injury [7,8]. Pericyte-associated and perivascular mesenchymal populations may also contribute to angiogenesis, immune modulation, stromal remodeling, and tissue homeostasis, although their precise role in adult human cranial dural healing remains incompletely defined [9,10]. This focused narrative review therefore synthesizes evidence on dura mater healing with particular emphasis on the molecular pathway and tissue integration processes that should guide next-generation dural substitute design.Subsequent paragraphs, however, are indented.

2 Methods

2.1 Review design and scope

The objective of this article was to synthesize mechanistic and translational evidence relevant to dura mater healing and dural substitute design. The review focused on the sequence of early sealing, biomaterial-associated host response, fibroblast-mediated

proliferation, neodura formation, matrix remodeling, and anti-adhesion performance. No patient records, human specimens, or identifiable clinical data were directly collected for this article. Therefore, institutional approval for patient-record research was not applicable. All data discussed in this review were derived from previously published literature.lease note that the first paragraph of a section or subsection is not indented.

2.2 Search strategy

A literature search was conducted in PubMed, Scopus, and Google Scholar up to 31 October 2025. Studies from January 2013 to October 2025 were prioritized to represent contemporary evidence. Seminal studies published before 2013 were included when they provided foundational information on dural fibroblast biology, dura-derived progenitor cells, sealing mechanics, scaffold architecture, biomaterial integration, or molecular mechanisms directly relevant to dura mater healing.

The core search string was adapted for each database using combinations of controlled vocabulary and free-text terms: ("dura mater" OR "dural defect" OR "dural injury" OR "dural repair" OR "duraplasty" OR "dural substitute") AND ("healing" OR "repair" OR "regeneration" OR "cerebrospinal fluid leak" OR "CSF leakage") AND ("fibroblast" OR "inflammation" OR "macrophage" OR "collagen" OR "extracellular matrix" OR "angiogenesis" OR "fibrosis" OR "biomaterial"). Additional focused terms included "platelet-rich plasma," "platelet-rich fibrin," "fibrin sealant," "dural fibroblast," "mesenchymal progenitor," "pericyte," "transforming growth factor-beta," "vascular endothelial growth factor," "nuclear factor-kappa B," "matrix metalloproteinase," "tissue inhibitor of metalloproteinase," "foreign-body reaction," and "anti-adhesion."

2.3 Study Selection and Evidence Appraisal

Studies were included when they were full-text English-language articles that provided original experimental, clinical, histological, cellular, biomechanical, or biomaterial evidence relevant to dural healing, dural substitutes, CSF leakage, scaffold integration, neodura formation, or adhesion prevention. Relevant reviews were used to identify background evidence and additional primary studies, but mechanistic conclusions prioritized primary data.

Articles were excluded if they were unrelated to dura mater, dural substitutes, or biological mechanisms of meningeal healing; focused solely on operative technique without relevant biological, mechanical, histological, biomaterial, or clinical outcomes; were editorials, commentaries, protocols, abstracts without full reports, or letters without original data; were duplicate or retracted publications; lacked accessible full text; or provided insufficient methodological detail for interpretation. Grey literature was considered only as supplementary or hypothesis-generating evidence and was not used as the sole basis for major mechanistic conclusions.

Evidence was mapped by study type and directness: human clinical or histopathological evidence, animal dural-defect models, human dural-cell and in vitro evidence, ex vivo biomechanical sealing evidence, biomaterial studies, and indirect molecular

evidence. The literature identification and evidence-mapping process used in this focused narrative review is shown in Fig. 1. Mechanistic interpretations based only on intervention type or histological appearance were separated from measured molecular findings.

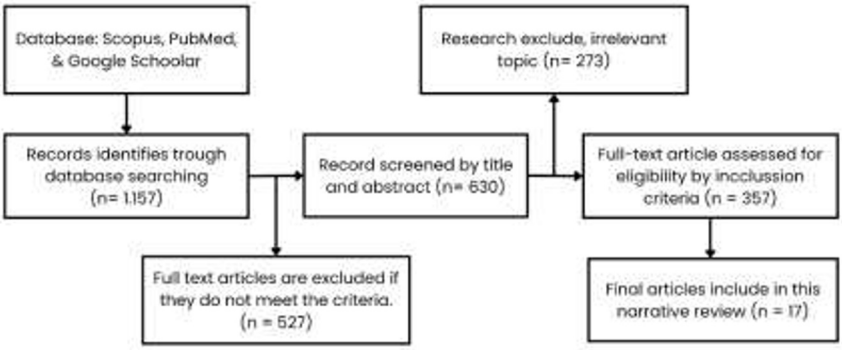


Fig. 1. Literature identification and evidence-mapping process for this focused narrative review.

3 Results and Discussion

3.1 Conceptual hierarchy: repair, healing, neodura formation, and functional regeneration

This study highlighted the need to distinguish healing from regeneration. In this review, dural repair refers to immediate closure of the defect and restoration of a watertight barrier [11-13]. Dural healing refers to the biological sequence that follows closure, including inflammation control, cellular infiltration, fibroblast activity, angiogenesis, and ECM deposition [14,15]. Neodura formation describes the development of a dural-like fibrocollagenous tissue that replaces or integrates with the implanted matrix. Functional regeneration represents the highest level of success: a stable CSF barrier with organized collagen architecture, appropriate immune resolution, low adhesion to neural tissue, and durable meningeal function. This hierarchy avoids overinterpreting neodura as a perfect histological replication of native dura mater [16,17]. The operational definitions used to distinguish dural repair, dural healing, neodura formation and functional regeneration are all summarised in Table 1.

Table 1. Conceptual hierarchy used to distinguish repair, healing, neodura formation, and functional regeneration.

Term	Core meaning	Typical evidence	Main limitation
Dural repair	Immediate mechanical closure of a dural defect and restoration of watertightness	Leak-pressure, burst-pressure, suture-retention, intraoperative watertight closure	Does not prove long-term biological regeneration
Dural healing	Cellular and tissue response after injury or implantation	Inflammation resolution, fibroblast infiltration, angiogenesis, collagen deposition	Can progress toward fibrosis or adhesion if dysregulated
Neodura formation	Formation of dural-like fibrocollagenous tissue replacing or integrating the scaffold	Histology showing fibrocollagenous tissue, endogenous collagen, scaffold resorption/integration	May be functional repair tissue rather than native-like dura
Functional regeneration	Durable barrier restoration with organized remodeling and minimal neural adhesion	Long-term CSF barrier, organized collagen, low adhesion, safe reoperation plane	Rarely proven by direct human molecular evidence

Clinical evidence should be interpreted separately from preclinical and ex vivo evidence because clinical outcomes are influenced by anatomy, underlying disease, surgical technique, and adjunctive closure strategy. In posterior fossa decompression for Chiari malformation, Danish et al. reported low CSF leak rates in both the DuraGen and AlloDerm groups, but different pseudomeningocele and infection rates [5]. Clinical studies of dural sealants suggest that fibrin-based adjuncts can improve intraoperative watertight closure; however, evidence for consistently reducing postoperative CSF leakage remains heterogeneous [18].

Leukocyte- and platelet-rich fibrin (L-PRF) membranes have been shown to be feasible during endoscopic endonasal skull base repair; however, conclusive efficacy findings are not possible due to the retrospective pilot design [19]. Platelet-rich plasma (PRP) was discovered to reduce CSF leakage, meningitis, partial skin necrosis, and wound dehiscence after meningomyelocele sac repair; however, the corrected interpretation indicates that wound dehiscence occurred in three PRP-treated patients and seven non-PRP patients, rather than wound healing occurring in those numbers [20]. These clinical data support the concept of early sealing and wound support, but they should not be generalized to all adult cranial or spinal dural defects.

3.2 Animal models and biomaterial-associated host response

Animal models provide the strongest direct evidence for the tissue response to dural substitute implantation. In a rabbit spinal dural defect model, A collagen matrix was

gradually absorbed in a rabbit spinal dural lesion model, causing no greater inflammatory response than primary closure and no capsule or adhesion formation [14]. A fully synthetic nanofabricated dural substitute increased neoduralization and reduced inflammation, fibrosis, and cortical adhesion compared with a bovine collagen substitute in rabbits [21,22]. Electrospun bacterial cellulose membranes lowered early inflammatory marker expression, supported more uniform collagen organization, and reduced adhesions and epidural scarring in a rabbit dural defect model [23-25].

Biomaterial immune response is central to these findings. After implantation, protein adsorption is followed by neutrophil and monocyte recruitment, macrophage adhesion and activation, and, in some materials, foreign-body giant-cell formation [26,27]. A sustained M1-like response is associated with proinflammatory mediators. It is also associated with tissue irritation, chronic inflammation, and fibrotic encapsulation. An M2-like reparative profile supports inflammatory resolution. It also supports angiogenesis, fibroblast coordination, matrix deposition, and constructive remodeling. The goal of regenerative dural biomaterial design is therefore not to eliminate immune activity, but to guide it toward controlled inflammation and host integration without chronic fibrosis or adhesion [25].

Postoperative adhesion should be considered a failure mode of dural repair from a biological perspective. Persistent inflammation or repeated material-related irritation may promote fibroblast overactivation, transforming growth factor-beta-associated profibrotic signalling, excessive collagen deposition and arachnoid or cortical scarring. This, in turn, can trigger profibrotic signalling associated with transforming growth factor-beta (TGF-beta). This signalling can result in excessive collagen being deposited, as well as scarring in the arachnoid or cortical regions. Anti-adhesion biomaterials attempt to reduce nonspecific protein adsorption and fibroblast attachment on the neural-facing surface while preserving cytocompatibility and integration on the dural-facing side. Kim et al. showed that poly (2-hydroxyethyl methacrylate) (pHEMA)-coated xenogeneic ECM reduced fibroblast adhesion in vitro and decreased severe intracranial adhesion in vivo; 90% of pHEMA-treated mice had no or minor adhesions [23].

3.3 Human dural-cell, in vitro, and ex vivo evidence

Cell-based studies clarify mechanisms that cannot be resolved from clinical outcome data alone. Goldschmidt et al. developed a human dural fibroblast culture model and showed fusiform fibroblast-like cells with pseudopod formation; dexamethasone reduced cell number and Ki-67 expression, whereas oxidized cellulose lowered culture pH and induced cellular necrosis and detachment [11]. This model supports the central role of viable dural fibroblast-like cells in tissue closure and demonstrates that local material chemistry can promote or impair the proliferative phase of dural healing.

Ex vivo biomechanical models primarily measure watertightness, not regeneration. Evaluation of 26 fresh bovine dura mater specimens showed that autologous PRF augmentation increased mean leak pressure from 10.5 ± 1.2 cmH_2O after running suture alone to 47.2 ± 2.6 cmH_2O after PRF augmentation [16]. A subsequent comparison of PRF and commercial sealants in a bovine dura model showed that onlay augmentation

increased median leak pressure from 11 cmH_2O to 38 cmH_2O [17]. These findings provide direct evidence for early mechanical sealing, but they should not be interpreted as proof of cellular regeneration or long-term neodura formation. The key clinical, animal, cellular, ex vivo and biomaterial studies supporting this evidence hierarchy are all summarised in Table 2.

Table 2. Reconstructed evidence matrix. Molecular mediators are listed only when directly measured in the corresponding study.

Author, year	Evidence category	Model / population	Material / intervention	Measured outcomes	Measured mediators / markers	Main interpretation
Danish et al., 2006	Clinical	Pediatric posterior fossa decompression for Chiari malformation	Acellular human dura vs bovine collagen matrix	Pseudomeningocele, wound infection, CSF leak	Not molecularly measured	Material-specific clinical complication rates; not general neurosurgery rates.
Arabaci et al., 2023	Clinical	40 infants undergoing meningomyelocele repair	PRP adjunct vs no PRP	CSF leak, meningitis, skin necrosis, wound dehiscence	Not molecularly measured	PRP reduced wound-related complications; corrected outcome is wound dehiscence, not wound healing.
Maeda et al., 2025	Human histopathology	Patients undergoing staged surgery after DuraGen duraplasty	Collagen matrix / DuraGen	Fibrin layer, fibroblasts, endogenous collagen, foreign-body reaction	Not molecularly measured	Fibrin/collagen matrix decreased over time while fibroblasts and endogenous collagen increased.
Calikoglu et al., 2019	Animal model	30 New Zealand White rabbits with spinal dural defect	Collagen matrix vs primary repair	Inflammation, fibroblast proliferation, foreign-body reaction, capsule, adhesion	Not molecularly measured	Controlled inflammatory response; minimal and resolving foreign-body reaction; no capsule or adhesion.
MacEwan et al., 2018	Animal model	New Zealand White rabbits with dural defect	Synthetic nanofabricated substitute vs bovine collagen substitute	Inflammation, fibrosis, neoduralization, resorption, adhesion	Not molecularly measured	Synthetic architecture improved neoduralization and reduced inflammation, fibrosis, and adhesion.

Author, year	Evidence category	Model / population	Material / intervention	Measured outcomes	Measured mediators / markers	Main interpretation
Yao Jing et al., 2020	Animal + in vitro	L929 fibroblasts and rabbit dural defect model	Electrospun bacterial cellulose membrane	Fibroblast response, histology, collagen distribution, adhesion, complications	TNF-alpha, IL-6, IL-1beta, iNOS, COX-2	EBC reduced early inflammatory signaling and adhesions while supporting collagen organization.
Goldschmidt et al., 2013	Human in vitro	Human dural fibroblast explant cultures	Spongostan, oxidized cellulose, dexamethasone, autologous tissues	Cell morphology, growth, viability	Ki-67, vimentin, desmin	Human dural fibroblast survival and proliferation are influenced by local material chemistry.
Vasilikos et al., 2020	Ex vivo biomechanics	26 fresh bovine dura specimens	PRF augmentation over running suture	Hydrostatic leak pressure	Not molecularly measured	Direct watertightness evidence; leak pressure increased more than fourfold after PRF augmentation.
Argiti et al., 2025	Ex vivo biomechanics	Fresh bovine dura testing model	PRF and commercial sealants as onlay augmentation	Hydrostatic leak pressure	Not molecularly measured	Onlay augmentation improved leak pressure, supporting early sealing but not proving regeneration.
Kim et al., 2025	In vitro + animal	Fibroblast adhesion assay and mouse intracranial adhesion model	pHEMA-coated xenogeneic ECM / Lyoplant	Fibroblast adhesion, adhesion score, cortical integrity	CD68-positive inflammatory cells reported	Hydrated anti-fouling surface reduced protein/cell adhesion and lowered severe cortical adhesion.
Cho et al., 2024	Animal + cytotoxicity	Male rat skull and dura mater defect model	Safe-Seal electrospun PLLA substitute	Cytotoxicity, mechanical testing, histology, giant cells, collagen, adhesion	Not molecularly measured	Giant cells decreased and fibroblast-associated repair increased by 12 weeks; adhesion was reduced.

3.4 Integrated molecular pathway and tissue integration across healing phases

The proposed pathway integrates four overlapping biological stages. First, hemostasis provides platelet activation, fibrin clot formation, and a provisional matrix that supports immediate CSF sealing. Second, controlled inflammation determines whether the material-host interface progresses toward constructive remodeling or foreign-body reaction. Nuclear factor-kappa B (NF-kB)-mediated cytokine activity, including interleukin-1 beta (IL-1beta), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-alpha), is relevant to this inflammatory checkpoint, although direct human dural molecular data remain limited [15]. Third, proliferation involves fibroblast migration, angiogenesis, and early ECM deposition. Platelet-derived growth factor, fibroblast growth factor, and vascular endothelial growth factor (VEGF) link the early clot and immune environment to fibrovascular ingrowth. Fourth, remodeling determines whether provisional tissue matures into organized neodura or fibrotic adhesion-prone scar [12,13].

3.5 Translational implications for dural substitute design

Mechanism-based dural substitute design should move beyond a single endpoint of watertight closure [28]. An ideal substitute should provide early mechanical sealing, controlled immune response, permissive fibroblast and vascular ingrowth, organized collagen remodeling, appropriate degradation or integration, and a neural-facing anti-adhesion surface [29]. Histological evaluation should include inflammatory cell infiltration, macrophage response, multinucleated giant cells, capsule formation, fibroblast ingrowth, collagen deposition, vascularization, scaffold integration, and adhesion grading. Molecular evaluation should report markers only when measured, and should distinguish direct dural evidence from general wound-healing inference [30]. Table 3 provides a summary of the proposed mechanism-based evaluation framework for future dural substitutes.

Table 3. Ideal biological healing cascade for dural substitute evaluation.

Stage	Dominant process	Desired material response	Key measurements	Failure mode
1. Hemostasis	Platelet activation, fibrin seal, provisional matrix	Immediate watertight closure and early scaffold support	Leak pressure, burst pressure, suture retention	Persistent CSF leak
2. Controlled inflammation	Macrophage response and debris clearance	Limit prolonged M1-like activation; support reparative remodeling	Inflammatory cells, giant cells, cytokines	Chronic inflammation or foreign-body reaction
3. Proliferation	Fibroblast migration and angiogenesis	Permit cellular infiltration and vascular support	Fibroblast density, Ki-67,	Weak repair or excessive fibroplasia

Stage	Dominant process	Desired material response	Key measurements	Failure mode
			CD31/vWF, collagen deposition	
4. Neodura formation	Collagen I/III deposition and tissue integration	Support endogenous ECM replacement or integration	Endogenous collagen, scaffold cellularization, tissue continuity	Incomplete neodura or fibrotic capsule
5. Functional remodeling	Organized collagen and anti-adhesion interface	Maintain stable CSF barrier and protect neural-facing surface	Adhesion score, collagen organization, long-term leak rate	Arachnoid/cortical adhesion or recurrent leak

4 Conclusion

Dura mater healing is a multistep biological process that includes early sealing, controlled inflammation, fibroblast migration, vascular ingrowth, collagen deposition, scaffold integration, remodeling, and adhesion prevention. The revised framework distinguishes immediate dural repair from dural healing, neodura formation, and functional regeneration. Available evidence supports the roles of platelet-fibrin sealing, macrophage-mediated host response, fibroblast survival, angiogenesis, and collagen remodeling, but direct human molecular evidence remains limited. Future dural substitute studies should therefore integrate mechanical, histological, molecular, and clinical endpoints while clearly separating clinical outcomes, animal-model findings, in vitro results, and inferred molecular pathways. A successful next-generation dural substitute should restore the CSF barrier immediately while guiding immune resolution, constructive fibrovascular integration, organized neodura formation, and long-term anti-adhesion performance.

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