

Review Article



Stem Cell-Based Liver Regeneration: From Cell Therapy to Vascularized, Immune-Compatible Engineered Tissue

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Summary

End-stage liver disease accounts for approximately two million deaths each year, and orthotopic transplantation remains constrained by donor scarcity and by the need for lifelong immunosuppression. Stem cell-based strategies aim to restore hepatic mass and function without whole-organ replacement. This review synthesizes the principal approaches and their translational status as of 2026. Mesenchymal stromal cells and their exosomes act mainly through immunomodulation, antifibrotic signaling, and paracrine support, and have reached randomized trials in chronic liver disease. Induced pluripotent stem cell-derived hepatocytes, direct lineage reprogramming, and transient in vivo reprogramming address the shortage of proliferation-competent hepatocytes. Liver organoids reproduce hepatic architecture and metabolism and have repaired injured bile ducts in perfused human liver. Bioengineering and three-dimensional bioprinting move these cells toward implantable tissue. Across every approach, two barriers determine clinical viability: perfusable vascularization sufficient to sustain a thick graft, and immune compatibility that avoids chronic immunosuppression. Recent work on self-organized vascularized hepatic organoids and on hypoimmunogenic, gene-edited constructs targets both barriers directly, and a 2026 program aims to bioprint a vascularized, immunocompetent liver from autologous cells. We frame these advances as augmentation of hepatic repair rather than as replacement of transplantation, and we outline the maturation, scalability, and safety questions that remain.

Keywords: Liver regeneration, Mesenchymal stromal cells, Liver organoids, Pluripotent stem cells, Cellular reprogramming, Regenerative medicine

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Introduction

Chronic liver disease causes approximately two million deaths worldwide each year, and its principal endpoints, cirrhosis and hepatocellular carcinoma, follow the progressive loss of functional hepatocytes.¹ Metabolic dysfunction-associated steatotic liver disease is now the most common chronic liver disease, and its 2023 renaming reflects the cardiometabolic drivers that also raise cardiovascular and renal risk.¹ Orthotopic liver transplantation remains the only definitive treatment for end-stage disease, yet the supply of donor organs meets a small fraction of need, and recipients accept lifelong immunosuppression with its infectious, metabolic, and oncologic costs. These constraints motivate strategies that restore hepatic mass and function without whole-organ replacement.

The liver has a distinctive capacity for regeneration. After acute injury or partial resection, mature hepatocytes re-enter the cell cycle and restore the original liver-to-body-weight ratio, a set-point often called the hepatostat.² When one epithelial compartment fails, hepatocytes and cholangiocytes can act as facultative stem cells and transdifferentiate to rebuild normal structure.² This endogenous program is efficient after a single insult but is

blunted in chronic disease, where fibrotic matrix, persistent inflammation, and replicative limits impair hepatocyte renewal.² The therapeutic problem is therefore twofold: to supply proliferation-competent hepatic cells, or to restore the endogenous capacity that chronic injury erodes.

This review maps the main stem cell-based approaches to liver regeneration and their translational status as of 2026. We consider cell and secretome therapy, pluripotent stem cell-derived hepatocytes, direct and in vivo reprogramming, liver organoids, and bioengineered constructs. We argue that two requirements now determine clinical viability across all of these approaches: perfusable vascularization sufficient to sustain a graft, and immune compatibility that avoids chronic immunosuppression (Figure 1). Throughout, we frame these technologies as augmentation of hepatic repair and of transplantation rather than as their replacement.

The endogenous regenerative response and its limits

Understanding where endogenous regeneration fails clarifies what cell therapy must provide. After two-thirds hepatectomy in rodents, quiescent hepatocytes synchronously re-enter the cell cycle under growth-factor and cytokine signaling, and the remnant restores mass



The Paracrine Power of MSCs in Liver Repair

Mesenchymal Stromal Cells (MSCs) and their exosomes act as "biological drug factories," releasing paracrine factors and extracellular vesicles to modify the diseased liver environment for endogenous repair.

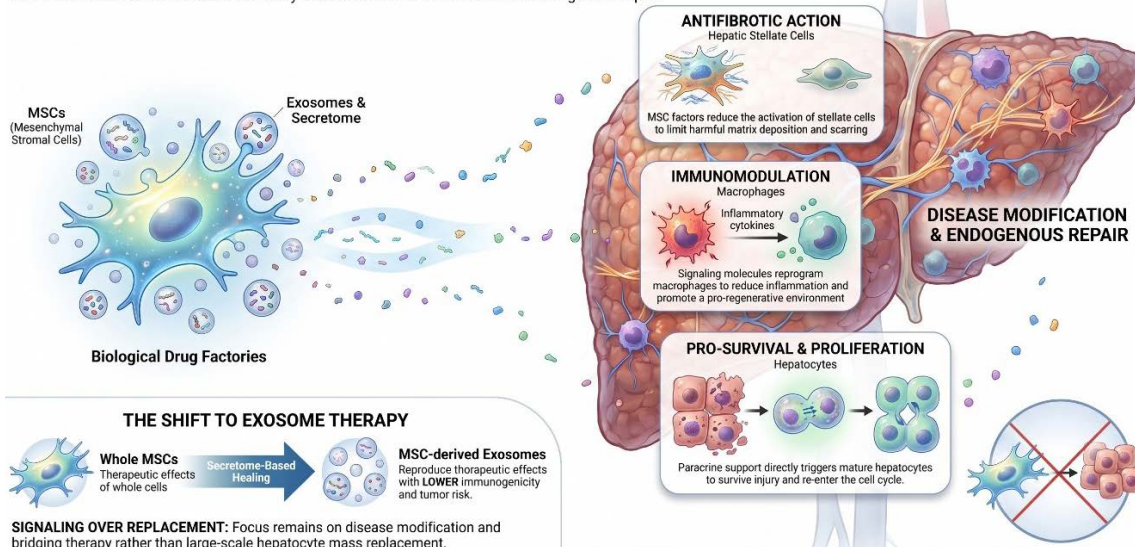


Figure 1. Stem cell-based strategies for liver regeneration and the two translational barriers to a functional graft. Cell sources feed multiple regenerative strategies, and every strategy must clear both vascularization and immune compatibility to yield transplantable liver tissue

within days.² Hepatocytes carry an essentially unlimited replicative reserve in serial transplantation models, and all zones of the lobule contribute to slow homeostatic turnover.² Damage also activates a ductular reaction, and Wnt-responsive Lgr5-positive cells emerge near bile ducts after injury and can generate both hepatocytes and cholangiocytes.³

In chronic disease this response is insufficient. Repeated hepatocyte death drives stellate-cell activation and matrix deposition, and the resulting fibrotic environment, together with senescence and inflammation, restricts effective hepatocyte proliferation.² Progenitor expansion in this setting often produces fibrosis rather than functional parenchyma. The clinical implication is that a durable therapy must either deliver cells that engraft and function within a diseased niche, or reactivate endogenous hepatocytes without amplifying fibrosis.

Cell therapy: mesenchymal stromal cells and their secretome

Mesenchymal stromal cells (MSCs) are the most widely tested cell product in liver disease. They are obtained from bone marrow, adipose tissue, and perinatal tissues, and umbilical-cord Wharton's jelly is an accessible source with low immunogenicity. The cellular composition of these source tissues is still being resolved; recent work in human umbilical cord identified telocyte-like cells adjacent to the classical stromal population, which illustrates the heterogeneity that affects product consistency.⁴

MSCs act less by engraftment and hepatocyte replacement than by paracrine signaling. Their released factors and extracellular vesicles modulate immune cells, reduce hepatic stellate-cell activation, and support hepatocyte survival and proliferation (Figure 2). Attention has shifted from whole cells to MSC-derived exosomes, which reproduce much of the therapeutic effect while carrying

lower immunogenicity and tumor risk; exosomes from adipose, bone-marrow, umbilical-cord, and iPSC sources have been compared directly, and engineering of the parent cell or the vesicle cargo can raise potency.^{5,6} These mechanisms position MSC products as disease-modifying and bridging therapies rather than as a means of large-scale hepatocyte replacement.

Clinical evidence is accumulating but remains mixed. A meta-analysis of randomized trials in acute-on-chronic liver failure and chronic liver disease found that MSC therapy improved the model for end-stage liver disease score, albumin, and coagulation relative to standard care, with fever as the main adverse event, but it did not show a clear survival benefit.⁷ Standardizing cell source, dose, and route, and defining potency for exosome products, are the principal barriers to wider use.^{5,6}

Pluripotent stem cell-derived hepatocytes and lineage reprogramming

Primary human hepatocytes proliferate poorly in culture, which limits their supply for transplantation. Induced pluripotent stem cells (iPSCs) offer an unlimited starting material and can be differentiated into hepatocyte-like cells, but these cells typically retain a fetal-like phenotype with incomplete cytochrome P450 activity and urea-cycle function, and their maturation and scalable manufacture remain unsolved.^{8,9}

Direct, or lineage, reprogramming converts somatic cells such as fibroblasts into induced hepatocytes without passing through a pluripotent state, using defined transcription factors, small molecules, messenger RNA, or CRISPR-based activation.⁸ This route offers autologous hepatocytes and avoids some risks of pluripotency, although conversion efficiency and the safety of integrating factors require attention. Hepatocyte transplantation itself has served as a clinical antecedent for these approaches, and its limits,

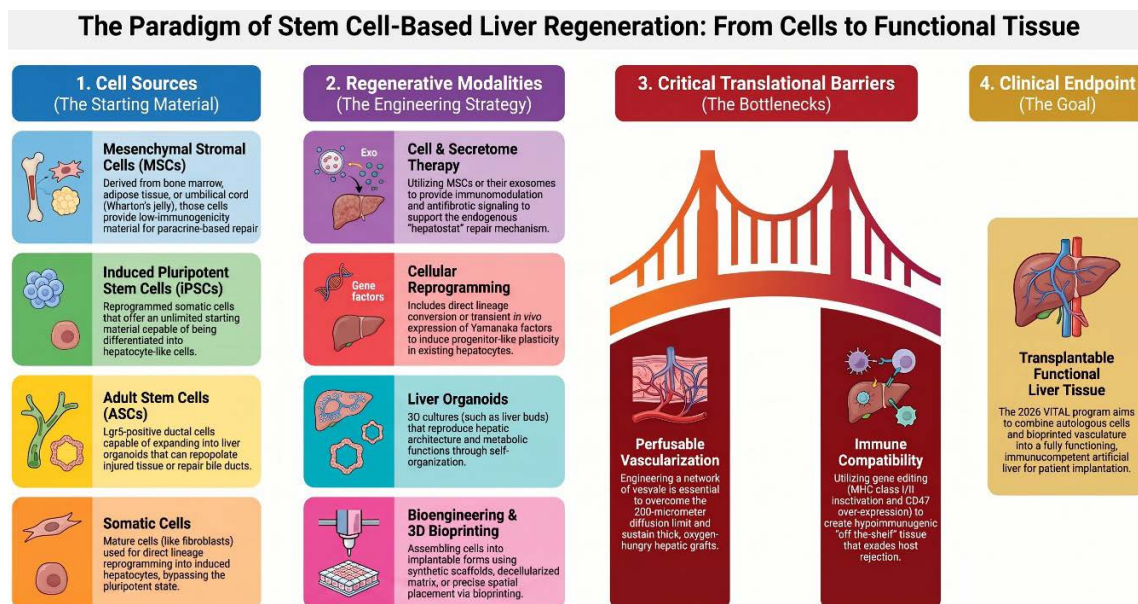


Figure 2. Mechanisms of mesenchymal stromal cell and exosome action in the injured liver. Rather than replacing hepatocytes, these products act by paracrine signaling on hepatic stellate cells (antifibrotic), macrophages (immunomodulation), and hepatocytes (pro-survival and proliferation)

transient engraftment and cell availability, are precisely the problems that reprogramming and organoid technologies aim to solve.⁸

Liver organoids

Liver organoids are three-dimensional cultures that reproduce hepatic architecture, gene expression, and metabolic functions such as drug metabolism and protein synthesis.^{9,10} Two main sources are used. Adult stem cell-derived organoids grow from Lgr5-positive ductal cells and can differentiate into functional hepatocytes that repopulate injured mouse liver.³ iPSC-derived organoids follow a developmental route, and co-culture of hepatic endoderm with endothelial and mesenchymal cells yields self-organizing liver buds.¹¹

Organoids have moved from disease modeling toward regeneration. The clearest human demonstration to date used cholangiocyte organoids to repair intrahepatic bile ducts in human livers undergoing *ex vivo* normothermic perfusion, a proof of principle that laboratory-expanded epithelium can restore a damaged biliary tree.¹² For solid-organ repair, the remaining obstacles are functional maturation, reproducible scale-up, and the delivery of enough tissue with adequate perfusion, which returns the discussion to vascularization.^{9,10}

In situ reprogramming of endogenous liver cells

An alternative to transplantation is to reprogram cells inside the liver. Transient expression of the four Yamanaka factors specifically in mouse hepatocytes induces partial reprogramming to a progenitor-like, proliferative state and enhances regenerative capacity after injury through a topoisomerase-2-dependent mechanism, without permanent loss of hepatocyte identity.¹³ Related lineage-conversion strategies aim to generate hepatocytes *in situ* or to induce liver-like tissue at ectopic sites.⁸ The appeal of *in situ* reprogramming is that it avoids *ex vivo* manufacture

and the surgical delivery of a graft. Its central risk is the flip side of its mechanism, because dedifferentiation carries oncogenic potential, so the timing and dose of reprogramming factors must be tightly controlled. Demonstrating durable, tumor-free benefit in large-animal models is the next step.

Bioengineering: scaffolds, decellularization, and 3D bioprinting

Bioengineering assembles cells into implantable form. Synthetic and natural scaffolds, and decellularized liver matrix that preserves a native vascular template, provide the architecture that dispersed cells lack.¹⁰ Three-dimensional bioprinting places multiple cell types with spatial control, and organoid-on-chip and liver-on-chip systems add perfusion and mechanical cues that improve maturation.^{9,10} Comparable scaffold, decellularization, and bioprinting strategies are being pursued in other organ systems, and they meet the same constraints of vascular patency and immune compatibility.¹⁴ Whole-organ bioengineering, in which a decellularized scaffold is recellularized and reperfused, remains limited by incomplete recellularization and by loss of vascular patency after implantation. Table 1 summarizes how these strategies compare across cell source, mechanism, and principal limitation.

Vascularization and immune compatibility: the convergent frontier

Across cell therapy, organoids, and bioengineering, two barriers now govern whether a construct can become a functional graft (Figure 3, Table 2). The first is vascularization. Hepatocytes have high oxygen and metabolic demand, and tissue thicker than roughly 200 micrometers cannot be sustained by diffusion alone, so any sizeable graft needs a perfusable vascular network. The founding demonstration co-cultured iPSC-derived hepatic cells with endothelial and mesenchymal cells to form

Table 1. Comparison of stem cell-based strategies for liver regeneration

Strategy	Cell source or basis	Principal mechanism	Key limitation	Evidence
MSC and secretome therapy	Bone marrow, adipose, and umbilical-cord MSCs and their exosomes	Immunomodulation, antifibrotic signaling, paracrine support	Mostly disease-modifying; limited mass replacement; no clear survival benefit yet	5-7
iPSC-derived hepatocytes	Reprogrammed somatic cells differentiated to hepatocyte-like cells	Replacement of lost hepatocytes from an unlimited source	Fetal-like maturation; incomplete drug metabolism; scalability	8,9
Direct (lineage) reprogramming	Somatic cells converted to induced hepatocytes without pluripotency	Autologous hepatocytes via factors, small molecules, or mRNA	Conversion efficiency; safety of integrating factors	8
In vivo partial reprogramming	Transient Yamanaka-factor expression in endogenous hepatocytes	Progenitor-like plasticity and proliferation in situ	Oncogenic risk; requires tight temporal control	13
Liver organoids (adult stem cell)	Lgr5-positive ductal cells expanded in three dimensions	Repopulation of injured liver; biliary repair	Maturation; scale for solid grafts	3,12
Liver organoids (iPSC or liver bud)	iPSC hepatic endoderm with endothelial and mesenchymal cells	Self-organizing, vascularizable buds	Size and perfusion at graft scale	10,11
Bioengineered or 3D-bioprinted constructs	Cells with scaffolds, decellularized matrix, or bioprinting	Spatially defined, potentially perfusable tissue	Vascular patency; recellularization; GMP scale	10,14,15

Abbreviations: 3D, three-dimensional; GMP, good manufacturing practice; iPSC, induced pluripotent stem cell; mRNA, messenger RNA; MSC, mesenchymal stromal cell. Superscript numbers indicate representative references

Table 2. Principal translational barriers and current mitigation strategies

Barrier	Consequence	Current mitigation approaches	Evidence
Vascularization	Diffusion limit restricts graft thickness and hepatocyte survival	Endothelial and mesenchymal co-culture; self-organizing vascularized organoids; microfluidic and bioprinted vascular templates	11,15,16
Immune compatibility	Allogeneic rejection requires lifelong immunosuppression	HLA matching; encapsulation; hypoinmunogenic cells with MHC class I and II inactivation and CD47 over-expression	17
Functional maturation	iPSC-derived hepatocytes remain fetal-like with incomplete metabolism	Prolonged and three-dimensional culture; organoid-on-chip; co-culture and morphogen tuning	9,10
Scalability and GMP	Clinical doses need very large, reproducible cell or vesicle yields	Standardized expansion; bioreactors; exosome isolation and potency standardization	5,6
Tumorigenicity and safety	Residual pluripotent cells or insertional and off-target genetic changes	Purification, non-integrating methods, transient factor expression, genomic surveillance	8,13

Abbreviations: CD47, cluster of differentiation 47; GMP, good manufacturing practice; HLA, human leukocyte antigen; iPSC, induced pluripotent stem cell; MHC, major histocompatibility complex. Superscript numbers indicate representative references

The Convergent Frontier: Engineering the Future of Liver Regeneration

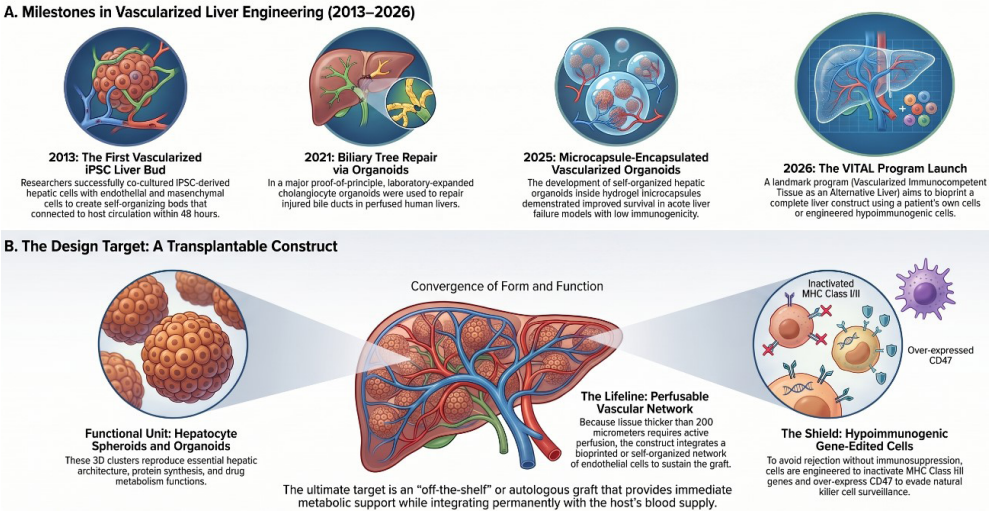


Figure 3. The convergent frontier. (A) Selected milestones from the first vascularized iPSC-derived liver bud to a 2026 program to bioprint a vascularized, immunocompetent liver. (B) Design target for a transplantable construct that combines hepatocyte spheroids or organoids, a perfusable vascular network, and hypoinmunogenic, gene-edited cells

vascularized liver buds whose human vessels connected to host circulation within 48 hours and matured the tissue after transplantation.¹¹ Recent work has advanced this principle, and self-organized vascularized hepatic organoids, generated by combining human endothelial cells with iPSC-derived hepatic organoids inside hydrogel microcapsules, improved survival in rats with acute liver

failure and showed low immunogenicity.¹⁵ Stepwise differentiation of induced pluripotent stem cells into endothelial progenitor and vascular smooth muscle cells, together with endothelial-mesenchymal co-culture, provides the enabling technology behind these constructs, and it places vascular biology at the center of liver tissue engineering.¹⁶

The second barrier is immune compatibility. Allogeneic cells and tissues face rejection, and the options are donor matching, physical encapsulation, or engineering cells that the immune system does not attack. Inactivation of major histocompatibility complex class I and class II genes together with over-expression of CD47 yields hypoimmunogenic iPSC derivatives, including endothelial cells, smooth-muscle cells, and cardiomyocytes, that evade rejection in fully mismatched immunocompetent recipients without immunosuppression.¹⁷ Applying the same engineering to the hepatic and vascular components of a liver construct would allow off-the-shelf, immune-compatible tissue.

These two lines are now converging. A 2026 program aims to bioprint a vascularized, immunocompetent liver from a patient's own cells, combining printed vasculature, autologous or engineered cells, and immune competence in a single construct.¹⁸ Organoid transplantation as a route to organ replacement is increasingly reviewed as a near-term goal rather than a distant one.¹⁹ The trajectory of the field is a shift from replacing lost hepatocytes toward building perfusable, immune-compatible tissue that can integrate with the host.

Discussion: challenges and translational outlook

Several problems separate current results from clinical use, and Table 2 pairs each barrier with the approaches now used to address it. Functional maturation is unresolved, because iPSC-derived hepatocytes remain fetal-like and lack full zonation and drug metabolism.⁹ Scalability and manufacture under good manufacturing practice are demanding, since clinical doses require very large, reproducible cell or vesicle yields, and exosome isolation and potency assays are not yet standardized.^{5,6} Vascularization has been achieved at the scale of microtissue but not yet at the scale of a graft that could support a failing liver.¹⁵ Hypoimmunogenic engineering raises its own questions, because loss of class I molecules can increase natural-killer-cell surveillance, a problem that CD47 over-expression mitigates but does not eliminate, and universal cell products need long-term safety monitoring.¹⁷ Tumorigenicity remains a shared concern across residual pluripotent cells, insertional events in reprogramming, and off-target genome edits.^{8,13}

The near-term outlook is nonetheless concrete. The convergence of vascularized organoids, hypoimmunogenic cell engineering, and bioprinting points toward first-in-human perfusion and engraftment studies for organoid and construct approaches, while MSC and exosome products continue to advance in fibrosis and acute-on-chronic liver failure.^{7,15} Computational tools can support this work through quality control, potency prediction, and donor-recipient matching, but they should augment rather than replace biological validation. The consistent theme is augmentation, because these technologies aim to extend and eventually reduce reliance on transplantation, not to promise its immediate replacement.

Conclusion

Stem cell-based liver regeneration has matured from

single-cell replacement toward engineered, perfusable, immune-compatible tissue. Mesenchymal stromal cells and their secretome offer near-term disease modification, iPSC-derived hepatocytes and reprogramming address the shortage of proliferation-competent hepatocytes, and bioengineering assembles these cells into implantable form. Vascularization and immune compatibility remain the decisive barriers, and advances reported in 2025 and 2026 target both directly. Whether these approaches reach the clinic will depend on functional maturation, scalable manufacture under good manufacturing practice, and long-term safety.

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Artificial Intelligence Use Disclosure

Tool: Claude (Anthropic), Opus model family, accessed June-July 2026.

Purpose of use: language editing and improvement of readability of the manuscript text, and assistance with formatting of the reference list in the journal's Vancouver style.

The tool was not used to generate original scientific content, to formulate the arguments or conclusions of this review, or to select or interpret the cited literature. The authors defined the scope of the review, selected all cited studies, and verified every reference against the primary source. All text produced with the assistance of the tool was reviewed and edited by the human authors, who take full responsibility for the accuracy, integrity, and originality of the work. Reporting of generative artificial intelligence use follows the GAMER (Generative Artificial Intelligence Tools in MEDical Research) reporting guideline.

Figures 1 to 3 are original schematic diagrams designed and finalized by the authors in vector graphics software. No generative artificial intelligence tool was used to create, generate, or alter any figure, image, or visual element of this manuscript.

Authors' Contribution

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Methodology: Mete Ucdal.

Project Administration: Mete Ucdal.

Supervision: Mohammadreza Dastouri.

Validation: Mohammadreza Dastouri.

Visualization: Mete Ucdal.

Writing-Original Draft: Mete Ucdal.

Writing-Review & Editing: Mete Ucdal, Mohammadreza Dastouri.

Competing Interests

The authors have relationships relevant to this submission that are declared in full on the title page and in the ICMJE disclosure forms submitted with this manuscript. The declaration is withheld here to preserve blinded peer review. No commercial or financial competing interest exists.

Ethical Approval

Not applicable. This narrative review is based exclusively on previously published literature and involved no new studies with human participants or animals performed by the authors. No individual patient data or identifiable information are included.

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