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A Review on In Limbal Stem Cell Deficiency Syndrome

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Abstract

Limbal Stem Cell Deficiency Syndrome (LSCD) is a serious ocular surface disorder caused by the loss or dysfunction of limbal stem cells located at the corneoscleral junction of the eye. These stem cells are responsible for maintaining corneal epithelial integrity, transparency, and regeneration. Damage to the limbal stem cell niche leads to conjunctivalization, neovascularization, persistent epithelial defects, stromal scarring, chronic inflammation, and progressive visual impairment. LSCD may occur due to chemical burns, infections, autoimmune diseases, genetic disorders, chronic inflammation, trauma, or prolonged contact lens use. Diagnosis is mainly based on clinical examination, impression cytology, slit-lamp biomicroscopy, in vivo confocal microscopy, and imaging techniques such as AS-OCT. Management depends on disease severity and includes medical therapy, amniotic membrane transplantation, and advanced limbal stem cell transplantation techniques such as CLAU, CLAL, KLAL, CLET, and SLET. Recent advances in regenerative medicine and tissue engineering have significantly improved the prognosis of LSCD. Early diagnosis and timely intervention are essential to restore ocular surface stability and prevent irreversible blindness.

I. INTRODUCTION

Stem cells are undifferentiated cells capable of self-renewal and differentiation into specialized cell types. They play a major role in tissue repair, regeneration, and maintenance of normal physiological functions. Limbal stem cells (LSCs) are adult stem cells located in the limbus, the transition zone between the cornea and sclera. These cells continuously regenerate the corneal epithelium and preserve corneal transparency.

The limbal region contains specialized fibrovascular ridges known as the Palisades of Vogt, which serve as the stem cell niche and provide nutritional and structural support for LSC survival and proliferation. When these cells or their microenvironment become damaged, the cornea loses its regenerative capacity, resulting in Limbal Stem Cell Deficiency Syndrome (LSCD).

LSCD is clinically important because it leads to persistent epithelial defects, conjunctivalization, corneal neovascularization, chronic inflammation, pain, photophobia, and severe visual impairment. Advances in stem cell biology, ocular surface reconstruction, and regenerative medicine have transformed the management of LSCD and improved patient outcomes.

CONTENTS

1. Stem Cells

Stem cells possess unique properties including self-renewal, clonogenicity, differentiation, plasticity, and regenerative capacity. Depending on their potency, stem cells are classified into totipotent, pluripotent, multipotent, oligopotent, and unipotent cells.

Based on their source, stem cells are classified as:

1. Embryonic stem cells
2. Induced pluripotent stem cells

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3. Somatic stem cells
4. Mesenchymal stem cells

Stem cells play essential roles in tissue regeneration, wound healing, immune regulation, and regenerative medicine.

2. Limbal Stem Cells

Limbal stem cells are undifferentiated adult stem cells found at the cornea–sclera junction. They maintain the corneal epithelium through continuous renewal and differentiation into corneal epithelial cells.

The limbal stem cell niche provides:

Nutritional support

Protection from environmental injury

Regulation of stem cell proliferation and differentiation

3. Anatomy of the Limbus

The limbus is a 1–2 mm transitional zone between the cornea and sclera. It contains:

Thick epithelial layers

Stromal connective tissue

Blood vessels

Palisades of Vogt

The Palisades of Vogt are radial fibrovascular ridges rich in melanocytes and blood vessels that support limbal stem cells and protect them from ultraviolet damage.

4. Limbal Stem Cell Deficiency

LSCD occurs when limbal stem cells or their niche are destroyed or become dysfunctional. This leads to failure of corneal epithelial regeneration and invasion of conjunctival epithelial cells onto the corneal surface.

Types of LSCD

1. **Partial LSCD**
- Some functional limbal stem cells remain
- Sectoral corneal involvement
- Mild to moderate visual impairment

2. **Complete LSCD**
- Total loss of limbal stem cells
- Entire cornea covered by conjunctival epithelium
- Severe vision loss and corneal opacity

5. Etiology and Pathophysiology

Etiology

1. **Genetic Causes**
- Aniridia
- PAX6 mutations
- Xeroderma pigmentosum
- Turner syndrome

2. **Acquired Causes**
- Chemical and thermal burns
- Infections
- Stevens–Johnson syndrome
- Ocular cicatricial pemphigoid
- Chronic inflammation
- Contact lens trauma

Pathophysiology

Loss of limbal stem cells results in:
- Failure of corneal epithelial renewal
- Breakdown of limbal barrier
- Conjunctivalization
- Neovascularization
- Stromal scarring
- Chronic inflammation

These changes ultimately lead to pain, photophobia, recurrent epithelial defects, and progressive vision loss.

6. Clinical Features

Symptoms
- Ocular pain
- Redness
- Photophobia
- Decreased vision

Signs
- Conjunctivalization of cornea
- Persistent epithelial defects
- Corneal opacity
- Neovascularization
- Chronic inflammation

7. Diagnosis and Classification

Diagnostic Methods
- Impression cytology
- Slit-lamp biomicroscopy
- Fluorescein staining
- In vivo confocal microscopy
- AS-OCT and AS-OCTA imaging

Clinical Staging

Stage I (Mild)
- Peripheral corneal involvement
- Minimal epithelial irregularity

Stage II (Moderate)
- Central extension
- Increased vascularization

Stage III (Severe)
- Total corneal involvement
- Dense scarring and severe visual impairment

8. Treatment and Management

Medical Management

Goals include reducing inflammation, promoting epithelial healing, and preventing further damage.

Treatment options:
- Artificial tears
- Topical antibiotics

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- Corticosteroids
- Autologous serum drops
- Therapeutic contact lenses

Surgical Management

Conservative Procedures

- Corneal scraping
- Amniotic membrane transplantation (AMT)

Limbal Stem Cell Transplantation

- Conjunctival limbal autograft (CLAU)
- Conjunctival limbal allograft (CLAL)
- Keratolimbal allograft (KLAL)
- Cultivated limbal epithelial transplantation
- Simple limbal epithelial transplantation (SLET)

9. Recent Advances in LSCD Therapy

SLET

Simple Limbal Epithelial Transplantation is a single-stage and cost-effective technique where small limbal grafts are distributed over an amniotic membrane to regenerate corneal epithelium.

CLET

Cultivated Limbal Epithelial Transplantation involves laboratory expansion of limbal stem cells before transplantation onto the ocular surface.

Biological Therapies

- Amniotic membrane transplantation
- Stem cell-based therapies
- Interferon α-2b therapy
- Retinoic acid therapy

Prosthetic Innovations

- Boston Keratoprosthesis
- Lux Keratoprosthesis
- CorNeat Keratoprosthesis

10. Complications and Prognosis

Untreated LSCD may lead to:

- Persistent epithelial defects
- Infectious keratitis
- Corneal ulceration
- Corneal perforation
- Permanent blindness

Modern surgical procedures such as SLET and CLET have significantly improved prognosis and visual rehabilitation. However, long-term follow-up is essential to prevent graft rejection and recurrence.

II. CONCLUSION

Limbal Stem Cell Deficiency Syndrome is a severe vision-threatening disorder caused by disruption of the limbal stem cell niche responsible for maintaining corneal epithelial integrity and transparency. Damage to limbal stem cells results in conjunctivalization, neovascularization, chronic inflammation, stromal scarring, and progressive visual impairment. Early diagnosis using advanced imaging and cytological techniques plays a vital role in preventing irreversible corneal damage. Recent advances in regenerative medicine, stem cell transplantation, and tissue engineering have revolutionized LSCD management and significantly improved patient outcomes. Techniques such as SLET, CLET, and COMET offer promising results in restoring ocular surface stability and preserving vision. Continued research in stem cell biology and regenerative therapies may further improve long-term prognosis and accessibility of treatment for LSCD patients.

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