T-cell mediated tissue injury in chronic inflammatory diseases: emerging insights from immunopathology.

Ayesha Q. Malik*

Department of Pathology, Mayo Clinic, Rochester, MN, USA

Introduction

T cells, critical components of the adaptive immune system, are primarily responsible for recognizing and eliminating infected or malignant cells. However, in chronic inflammatory diseases, these same T cells can become dysregulated, leading to persistent inflammation and tissue damage. Immunopathological studies have increasingly highlighted the central role of T-cell mediated injury in diseases such as rheumatoid arthritis (RA), multiple sclerosis (MS), inflammatory bowel disease (IBD), and type 1 diabetes (T1D).

T-cell mediated tissue injury occurs when autoreactive T cells mistakenly recognize self-antigens as threats. In healthy individuals, central and peripheral tolerance mechanisms eliminate or suppress such autoreactive cells. However, genetic predisposition, environmental triggers, and epigenetic changes can disrupt these mechanisms, allowing pathogenic T cells to escape immune surveillance and initiate autoimmune responses.

In chronic inflammatory diseases, both CD4+ and CD8+ T cells contribute to tissue pathology. CD4+ T helper cells differentiate into various subsets, including Th1, Th17, and T regulatory (Treg) cells, each with distinct roles. Th1 cells produce interferon-gamma (IFN-γ), which activates macrophages and promotes inflammation. Th17 cells, known for secreting interleukin-17 (IL-17), recruit neutrophils and exacerbate tissue damage, particularly in autoimmune conditions like RA and psoriasis. On the other hand, a deficiency or dysfunction of Treg cells, which typically suppress immune responses and maintain tolerance, leads to unchecked inflammation and disease progression [1-5].

CD8+ cytotoxic T lymphocytes (CTLs) are particularly involved in direct tissue destruction. In diseases like T1D, CTLs infiltrate pancreatic islets and induce apoptosis in insulin-producing beta cells. Similarly, in MS, autoreactive CTLs breach the blood-brain barrier and attack myelin sheaths, causing neuronal damage and neurological deficits.

Immunopathological evidence from biopsy and autopsy studies has shed light on the spatial and cellular dynamics of T-cell mediated injury. In synovial tissue of RA patients, dense aggregates of T cells and macrophages are observed, accompanied by cytokine release and joint erosion. In MS, brain lesions exhibit perivascular cuffs of T cells and demyelination zones, reflecting targeted immune attacks. These findings are corroborated by the presence of pro-inflammatory cytokines and chemokines in affected tissues and circulation.

Emerging technologies such as single-cell RNA sequencing, spatial transcriptomics, and advanced imaging have further unraveled the complex roles of T cells in chronic inflammation. These tools allow researchers to map T-cell subsets, activation states, and interactions with other immune and stromal cells in affected tissues. This growing understanding is paving the way for more precise therapeutic interventions [6-10].

Current treatments targeting T-cell activity include biologics like abatacept, which interferes with T-cell costimulation, and monoclonal antibodies against IL-17 and IL-23, which modulate Th17 responses. New strategies aim to restore immune tolerance through Treg enhancement, antigen-specific immunotherapy, and personalized cellular therapies.

Conclusion

In conclusion, T-cell mediated tissue injury is a central driver of chronic inflammatory diseases. Immunopathological research continues to uncover how T-cell subsets orchestrate damage and dysfunction across various tissues. These insights are crucial for developing targeted, disease-specific interventions that can halt or reverse the course of chronic inflammation.

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***Correspondence to:** Ayesha Q. Malik, Department of Pathology, Mayo Clinic, Rochester, MN, USA. E-mail: aqmalik@mayo.edu

***Received:** 10-Feb-2025, Manuscript No. AACPLM- 25-164068; **Editor assigned:** 11-Feb-2025, Pre QC No. AACPLM- 25-164068 (PQ); **Reviewed:** 12-Feb-2025, QC No. AACPLM-25-164068; **Revised:** 19-Feb-2025, Manuscript No. AACPLM- 25-164068 (R); **Published:** 20-Feb-2025, DOI: 10.35841/ aacplm-7.1.248*

***Citation:** Malik AQ. T-cell mediated tissue injury in chronic inflammatory diseases: emerging insights from immunopathology. J Clin Path Lab Med.2025;7(1):248*

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Citation: *Malik AQ. T-cell mediated tissue injury in chronic inflammatory diseases: emerging insights from immunopathology. J Clin Path Lab Med.2025;7(1):248*