



# Insights into Corneal Neuropathic Pain: Unraveling the Neurophysiological Mechanisms

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## Abstract

Corneal neuropathic pain is a debilitating condition characterized by persistent ocular discomfort, burning, and stinging sensations, often resulting from damage or dysfunction of corneal nerves. This condition poses a significant challenge in ophthalmology and pain management. Patients suffering from corneal neuropathic pain often describe symptoms such as persistent burning, stinging, and foreign body sensation, severely impacting their quality of life. Understanding the neurophysiological mechanisms underlying this condition is crucial for developing effective treatments and improving patient outcomes. Corneal neuropathic pain can be central, peripheral or have both central and peripheral components. Corneal neuropathic pain is a complex process involving different cell types and molecules; nerves, dendritic cells, neurokinins, neuropeptides, and axon-guidance molecules which causes a high level of sensory rearrangement. These processes emanating in corneal neuropathic pain is not well understood warranting further studies to ascertain appropriate pharmacotherapeutics required in specific clinical scenarios [1]. This paper reviews the current understanding of the neurophysiological pathways of corneal neuropathic pain and current state of its neuropharmacological management.

## Introduction

Corneal neuropathic pain, a debilitating condition resulting from damage or dysfunction of corneal nerves, presents a significant challenge in ophthalmology and pain management. Patients suffering from corneal neuropathic pain often describe symptoms such as persistent burning, stinging, and foreign body sensation, severely impacting their quality of life. Understanding the neurophysiological mechanisms underlying this condition is crucial for developing effective treatments and improving patient outcomes. Corneal neuropathic pain can be central, peripheral or have both central and peripheral components. Corneal neuropathic pain is a complex process involving different cell types and molecules; nerves, dendritic cells, neurokinins, neuropeptides, and axon-guidance molecules which causes a high level of sensory rearrangement. These processes emanating in corneal neuropathic pain is not well understood warranting further studies to ascertain appropriate pharmacotherapeutics required in specific clinical scenarios [1]. This paper reviews the current understanding of the neurophysiological pathways of corneal neuropathic pain and current state of its neuropharmacological management.

**Neuroanatomy of the cornea may contribute to the amplification of pain signals at the site of injury. Additionally, central sensitization, involving**

the trigeminal ganglion and the brainstem, may play a role in the pathogenesis of corneal neuropathic pain.

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