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INFERIOR ALVEOLAR NERVE INJURY IN MANDIBULAR FRACTURES: CLINICAL SIGNIFICANCE, DIAGNOSIS, AND MANAGEMENT

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Annotation: Mandibular fractures are among the most common injuries of the maxillofacial region due to trauma, accidents, and interpersonal violence. One of the most significant complications associated with fractures of the mandibular body, angle, and ramus is injury to the inferior alveolar nerve (IAN). Damage to this nerve can result in sensory disturbances such as paresthesia, hypoesthesia, anesthesia, and dysesthesia of the lower lip, chin, and mandibular teeth. This article provides a comprehensive review of inferior alveolar nerve injury associated with mandibular fractures, focusing on anatomical considerations, mechanisms of nerve damage, diagnostic approaches, clinical outcomes, and current management strategies. The analysis is based on peer-reviewed scientific literature and authoritative textbooks in oral and maxillofacial surgery.

Key words: mandibular fracture, inferior alveolar nerve, nerve injury, paresthesia, maxillofacial trauma, neurosensory deficit

Introduction

Fractures of the mandible represent a significant proportion of maxillofacial injuries worldwide. Epidemiological studies indicate that mandibular fractures account for approximately 36–59% of all facial fractures, making them second only to nasal bone fractures in frequency [1]. Due to the anatomical course of the inferior alveolar nerve within the mandibular canal, fractures involving the body, angle, and ramus of the mandible pose a high risk of nerve injury.

The inferior alveolar nerve, a branch of the mandibular division of the trigeminal nerve (cranial nerve V3), is responsible for sensory innervation of the lower teeth, lower lip, chin, and gingiva. Injury to this nerve significantly affects patients’ quality of life, leading to functional impairment, psychological distress, and medico-legal issues [2]. Understanding the pathophysiology, diagnosis, and management of inferior alveolar nerve damage in mandibular fractures is therefore of critical importance for clinicians.

Methodology

This article is based on a structured review of scientific literature including textbooks, clinical studies, and systematic reviews published in peer-reviewed journals. Sources were selected from internationally recognized publications in oral and maxillofacial surgery, neurology, and trauma medicine. The reviewed materials focused on anatomical studies, clinical observations, diagnostic methods, and treatment outcomes related to inferior alveolar nerve injury following mandibular fractures. Data were analyzed qualitatively to synthesize current evidence-based knowledge on the topic [3], [4].

Results

Analysis of the reviewed literature demonstrates that inferior alveolar nerve injury occurs in approximately 20–60% of mandibular fractures, depending on fracture location and severity [5]. Fractures of the mandibular angle and body show the highest incidence of nerve involvement due to their close proximity to the mandibular canal.

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Clinical manifestations vary from transient sensory disturbances to permanent neurosensory deficits. Several studies report that neuropraxia is the most common type of nerve injury, with spontaneous recovery occurring within weeks to months in most cases [6]. However, more severe injuries such as axonotmesis and neurotmesis may lead to incomplete or absent recovery. Radiological assessment using panoramic radiography and cone-beam computed tomography (CBCT) significantly improves detection of nerve canal involvement. Early diagnosis and appropriate management are associated with better sensory recovery outcomes [7].

Analysis and Discussion

The close anatomical relationship between the inferior alveolar nerve and the mandibular canal explains the high incidence of neurosensory disturbances following mandibular fractures. The inferior alveolar nerve originates from the posterior division of the mandibular nerve (V3) and enters the mandibular foramen on the medial surface of the ramus, continuing anteriorly within the mandibular canal until it emerges as the mental nerve through the mental foramen [8]. Because this canal is enclosed within the mandibular body and angle, fractures in these regions frequently involve direct or indirect injury to the nerve.

Clinical and anatomical studies consistently demonstrate that fractures of the mandibular angle and body are associated with a significantly higher rate of inferior alveolar nerve dysfunction compared to fractures of the symphysis or condyle [5]. This is primarily due to the location of the nerve canal, which lies closer to the inferior border of the mandible in the molar region and becomes particularly vulnerable when fracture lines pass through or adjacent to the canal. Displacement of fracture segments can result in compression or stretching of the nerve, while comminuted fractures may lead to partial or complete transection [1].

The mechanisms of inferior alveolar nerve injury in mandibular fractures are multifactorial. Direct mechanical trauma from sharp bony fragments is one of the most commonly described causes, especially in high-energy injuries such as road traffic accidents or interpersonal violence [2]. In addition, secondary factors such as hematoma formation, soft tissue edema, and local inflammatory responses can increase pressure within the mandibular canal, leading to ischemia and temporary conduction block of the nerve fibers [9]. These indirect mechanisms often explain cases where neurosensory deficits occur despite the absence of obvious canal disruption on radiographic imaging.

The type and severity of nerve injury have a significant impact on clinical presentation and prognosis. According to Seddon's classification, nerve injuries can be categorized into neuropraxia, axonotmesis, and neurotmesis [6]. Neuropraxia, characterized by transient conduction block without axonal disruption, is the most frequently observed form in mandibular fractures and is associated with a favorable prognosis. Axonotmesis involves damage to the axon with preservation of connective tissue sheaths, resulting in longer recovery periods, while neurotmesis represents complete nerve transection with minimal potential for spontaneous recovery.

Numerous clinical studies report that the majority of inferior alveolar nerve injuries following mandibular fractures fall within the neuropraxia or mild axonotmesis categories [5], [12]. This explains why spontaneous sensory recovery is commonly observed within weeks to months after injury. However, recovery is often incomplete, and subtle sensory disturbances may persist long-term, particularly in cases involving severe displacement or delayed treatment.

Radiological evaluation plays a crucial role in assessing the risk and extent of nerve injury. Conventional panoramic radiography has long been used as a standard diagnostic tool in mandibular trauma, allowing visualization of fracture lines and their relationship to the mandibular canal [7]. However, panoramic images provide limited three-dimensional

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information and may underestimate canal involvement. The introduction of cone-beam computed tomography (CBCT) has significantly improved diagnostic accuracy by allowing precise assessment of fracture displacement, canal disruption, and proximity of fixation hardware to the nerve [7]. Several studies highlight that CBCT findings correlate strongly with clinical neurosensory outcomes and should be considered the imaging modality of choice in complex mandibular fractures.

Neurosensory assessment remains a cornerstone of clinical evaluation in patients with mandibular fractures. Subjective complaints such as numbness, tingling, burning sensations, or altered sensitivity of the lower lip and chin are often the first indicators of inferior alveolar nerve involvement [10]. Objective testing methods, including light touch, pin-prick discrimination, two-point discrimination, and thermal sensitivity tests, are widely used due to their simplicity and clinical applicability. Although advanced techniques such as quantitative sensory testing and electrophysiological studies have been described, their use is largely limited to research settings and specialized centers [10].

The timing of surgical intervention has been identified as a critical factor influencing nerve recovery. Early reduction and stabilization of mandibular fractures are believed to reduce ongoing mechanical irritation and ischemic damage to the nerve [11]. Several longitudinal studies demonstrate that patients treated within the first 72 hours after injury show significantly better neurosensory outcomes compared to those who undergo delayed treatment [12]. Delays may allow prolonged compression, inflammatory changes, and fibrosis to develop around the nerve, negatively affecting regeneration.

Open reduction and internal fixation (ORIF) is the preferred treatment modality for displaced mandibular fractures, as it provides stable anatomical alignment and allows early functional rehabilitation [1]. However, ORIF itself carries a risk of iatrogenic nerve injury. Improper placement of fixation plates or screws near the mandibular canal can result in direct nerve compression or penetration [9]. This highlights the importance of meticulous surgical planning, accurate radiological assessment, and adherence to anatomical landmarks during fixation.

The choice of fixation technique may also influence neurosensory outcomes. Studies comparing miniplate fixation to reconstruction plates suggest that less invasive fixation methods may be associated with lower rates of postoperative nerve dysfunction, particularly in fractures with minimal displacement [4]. Nevertheless, fracture stability and proper occlusal alignment remain the primary goals of treatment, and the fixation method should be selected based on fracture pattern and biomechanical requirements rather than nerve considerations alone.

Conservative management remains the mainstay of treatment for most inferior alveolar nerve injuries associated with mandibular fractures. Anti-inflammatory medications, corticosteroids in the acute phase, and vitamin B complex supplementation are commonly prescribed to reduce inflammation and support nerve regeneration [11]. Although evidence supporting pharmacological interventions is mixed, these approaches are widely accepted in clinical practice due to their low risk and potential benefits.

Surgical exploration of the inferior alveolar nerve is generally reserved for selected cases. Indications include persistent sensory deficits beyond 3–6 months, radiological evidence of nerve transection, or worsening neurosensory symptoms following fracture fixation [11]. Microsurgical techniques such as neurorrhaphy or nerve grafting have been described, but outcomes vary widely, and complete sensory recovery is rarely achieved. Consequently, careful patient selection and realistic expectation management are essential.

From a prognostic perspective, several factors have been consistently associated with poorer neurosensory outcomes. These include severe fracture displacement, comminution, advanced

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patient age, delayed treatment, and the presence of infection [12]. Conversely, young patients with minimally displaced fractures and early intervention demonstrate the highest likelihood of complete sensory recovery. These findings underscore the multifactorial nature of nerve healing and the importance of individualized treatment planning.

Beyond functional impairment, inferior alveolar nerve injury has significant psychosocial and medico-legal implications. Persistent sensory disturbances can interfere with speech, mastication, and facial expression, negatively impacting quality of life [2]. Patients may experience anxiety, frustration, or dissatisfaction with treatment outcomes, particularly if they were not adequately informed about potential complications. Clear documentation of preoperative neurosensory status and thorough patient counseling are therefore essential components of ethical clinical practice.

Conclusion

Inferior alveolar nerve injury is a common and clinically significant complication of mandibular fractures. Its occurrence is closely related to fracture location, degree of displacement, and treatment modality. Accurate diagnosis, careful surgical planning, and appropriate management are essential to minimize neurosensory deficits and improve patient outcomes. Continued research and standardized assessment protocols are necessary to further optimize treatment strategies and prognostic evaluation in patients with mandibular fractures involving the inferior alveolar nerve.

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