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RESEARCH ARTICLE

Management Algorithm for Iatrogenic Trigeminal Nerve Injuries in Dental Patients

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Abstract: Background: Iatrogenic trigeminal nerve injuries, particularly of the inferior alveolar and lingual nerves, represent a severe and disabling complication of dental procedures such as third molar extractions, implant placement, and endodontic treatment. While up to 90% of such injuries are transient, 10–18% become permanent if symptoms persist beyond six months. The primary pathogenetic mechanism is compression-ischemic syndrome within rigid anatomical canals, where post-traumatic edema leads to axonal hypoxia and demyelination.

Purpose: To improve treatment efficacy and reduce rehabilitation time in patients with iatrogenic trigeminal nerve injuries by implementing an early pathogenetic therapy algorithm combining anti-exudative intervention with modern physiotherapeutic methods.

Methods: A retrospective and prospective analysis was conducted on 60 patients with iatrogenic injuries to the inferior alveolar and lingual nerves. The main group (n=50) received complex pathogenetic therapy including early anti-edematous agents (glucocorticosteroids, venotonics) and physiotherapeutic methods (low-level laser therapy, magnetotherapy) within 24–72 hours post-injury. The comparison group (n=10) received standard treatment limited to B-complex vitamins and NSAIDs.

Results: Complete sensory recovery was achieved in 72% of the main group versus 30% of the comparison group. Mean recovery time was 3–4 months in the main group compared to 6–8 months in the comparison group. Chronic neuropathic pain developed in only 8% of the main group versus 50% of the comparison group. Early dexamethasone administration reduced paresthesia resolution time by 4–6 days and decreased permanent neuropathy risk by nearly 46%.

Conclusion: Early initiation of anti-edematous therapy combined with physiotherapeutic methods significantly enhances treatment efficacy, shortens rehabilitation periods, and minimizes the risk of chronic neurological complications following iatrogenic trigeminal nerve injuries.

Key words: Iatrogenic trigeminal nerve injury, inferior alveolar nerve, lingual nerve, anti-edematous therapy, glucocorticosteroids, low-level laser therapy, magnetotherapy, dental complications, neuropraxia, axonotmesis.

INTRODUCTION

Iatrogenic trigeminal neuralgia (ITN) is a painful syndrome resulting from medical interventions, most commonly dental or surgical procedures in the maxillofacial region. In contrast to the classical form caused by vascular compression, iatrogenic neuralgia is associated with direct damage to nerve structures or their secondary trauma. It remains one of the most severe and disabling complications in modern dentistry and oral and maxillofacial surgery. Despite advancements in diagnostic methods and surgical protocols, the

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incidence of trigeminal neuropathies shows no significant downward trend. This is driven by the increasing volume of complex dental interventions, such as dental implantation, complicated third molar extractions, and endodontic treatment. On a positive note, up to 82–90% of such injuries are transient and resolve within the first 8 weeks. However, 10–18% of injuries progress to the permanent category if symptoms persist for more than 6 months [9, 10].

Early anti-edematous (anti-exudative) and physiotherapeutic support occupies a critical position in the framework of modern treatment algorithms for iatrogenic trigeminal nerve injuries. The traditional approach, which focused exclusively on neurotropic vitamin therapy, is now recognized by the global community (specifically by AAOMS experts) as insufficient due to the specific anatomy of the maxillofacial region. The primary pathogenetic mechanism of inferior alveolar nerve injury is the development of compression-ischemic syndrome. Within the narrow, rigid confines of the mandibular canal, even minor post-traumatic edema or intraneural hemorrhage leads to a critical increase in pressure inside the nerve sheath. This pressure elevation compresses the vasa nervorum capillaries, inducing axonal hypoxia and subsequent demyelination. Without the administration of potent anti-edematous agents (glucocorticosteroids, osmotic drugs) within the first 48–72 hours following the incident, the reversible stage of injury (neuropraxia) inevitably transforms into structural fiber degeneration (axonotmesis).

Purpose of the research

To improve treatment efficacy and reduce rehabilitation time in patients with iatrogenic trigeminal nerve injuries by implementing an early pathogenetic therapy algorithm that combines anti-exudative intervention with modern physiotherapeutic methods.

METHODS

Our study involved a retrospective and prospective analysis of the treatment of 60 patients with iatrogenic injuries to the inferior alveolar and lingual nerves resulting from dental interventions (third molar extractions, implant placement, endodontics). To evaluate the efficacy of the developed algorithm, all patients were divided into two groups:

Main group (n=50): Patients who received complex pathogenetic therapy according to the proposed algorithm. Treatment included immediate anti-edematous therapy (glucocorticosteroids, venotonics) and the early initiation of physiotherapeutic methods (low-level laser therapy [LLLT], magnetotherapy) within the first 24–72 hours following the injury. Comparison group (n=10): Patients who received standard treatment limited solely to neurotropic B-complex vitamins and NSAIDs, without the administration of specific anti-edematous agents or physical therapy during the acute period.

RESULTS AND DISCUSSION

The study included 60 patients with iatrogenic injuries to the inferior alveolar and lingual nerves. The patients were divided into two groups: the main group (n=50), where complex pathogenetic therapy was applied, and the comparison group (n=10), which received standard treatment. An analysis of the distribution of injury types showed that the inferior alveolar nerve was most frequently affected in both groups. In the main group, its injury was documented in 32 patients, accounting for 64% of the total observations. The lingual nerve was involved in the pathological process in 18 patients (36%). This ratio supports clinical data indicating that the inferior alveolar nerve is the most vulnerable during dental interventions, particularly during mandibular third molar extractions and implant placement in the mandible. In the comparison group, the distribution was similar: inferior alveolar nerve injury was observed in 6 patients (60%), while lingual nerve injury was noted in 4 patients (40%). Despite the smaller sample size, the data demonstrate the same trend—a predominance of inferior alveolar nerve trauma over lingual nerve injuries. It should be emphasized that although lingual nerve injuries occur less frequently, they carry significant clinical implications. They are accompanied by

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sensory disturbances on the lateral surface of the tongue, which affects speech, taste perception, and the patient's overall quality of life. In contrast, inferior alveolar nerve injuries more commonly manifest as paresthesia or anesthesia in the lower lip and chin area, causing pronounced discomfort in daily life. In conclusion, the obtained results confirm that the inferior alveolar nerve remains the most frequently injured structure during dental procedures; however, the significant proportion of lingual nerve lesions demands equal attention. This highlights the necessity for meticulous preoperative planning, the utilization of advanced imaging modalities, and adherence to tissue-sparing surgical techniques to minimize the risk of trauma to both nerves. Furthermore, an analysis of the causes of iatrogenic injuries to the inferior alveolar and lingual nerves revealed that the extraction of mandibular third molars remains the most frequent risk factor. In the main group, this type of intervention caused injury in 28 patients (56%). This is attributed to the complex anatomy of impacted and malpositioned wisdom teeth, their close proximity to the mandibular canal, and the technical difficulties encountered during surgical extraction. Even with adherence to standard protocols, there remains a probability of compression or direct damage to nerve structures. Implant placement ranked second among the causes of injury, accounting for 15 cases (30%). Implant insertion into the mandibular zone with insufficient evaluation of the inferior alveolar nerve canal topography can lead to its traumatization. Furthermore, excessive pressure during osteotomy and implant placement with violations of depth and angulation also create a risk of nerve fiber damage.

Endodontic treatment was found to be the cause of injury in 7 patients (14%). Although endodontics is traditionally considered a less traumatic procedure, errors during root canal instrumentation or the extrusion of filling material beyond the apex can lead to chemical or mechanical injury to nerve structures.

In the comparison group, the distribution of causes was similar but with fewer observations: third molar extractions accounted for 6 cases (60%), implant placement for 3 cases (30%), and endodontics for 1 case (10%). This confirms that surgical interventions in the mandibular region are the primary source of risk for the development of iatrogenic neuralgia. Consequently, third molar extraction remains the leading cause of nerve injuries, which demands special attention to preoperative planning and a thorough evaluation of the patient's anatomical features. Implant placement and endodontics also present a significant risk, particularly in cases of insufficient visualization and violations of procedural technique.

An analysis of the timing of treatment initiation revealed fundamental differences between the two study groups. In the main group, therapy was initiated within the first 24–72 hours following the injury. This approach was based on understanding the pathogenetic role of post-traumatic edema and ischemia of nerve structures. Early intervention allowed for the timely control of the inflammatory process, reduced interstitial pressure, and prevented the development of irreversible degenerative changes in nerve fibers. The administration of glucocorticosteroids and venotonics during the acute period provided decompression of the nerve trunks, while the incorporation of physiotherapeutic methods (low-level laser therapy, magnetotherapy) promoted the activation of regenerative processes.

In the comparison group, therapy was delayed and limited to the administration of B-complex vitamins and nonsteroidal anti-inflammatory drugs (NSAIDs). The absence of anti-edematous therapy during the critical early hours post-injury allowed the pathological process to progress via an ischemic mechanism, accompanied by prolonged compression of the nerve structures. This significantly reduced the likelihood of complete sensory recovery and heightened the risk of pain chronification.

Consequently, the variations in the timing of therapy initiation proved to be decisive for the treatment outcome. Early intervention in the main group ensured a higher rate of complete sensory recovery, shortened rehabilitation

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periods, and reduced the risk of developing permanent neuropathy. In contrast, delayed treatment in the comparison group demonstrated significantly poorer outcomes, confirming the critical importance of timely administration of anti-edematous therapy and physiotherapeutic methods within the first 24–72 hours post-injury.

The study revealed significant differences in therapeutic approaches between the main group and the comparison group, which had a direct impact on treatment outcomes. This complex regimen made it possible to target the key elements of pathogenesis: reduce edema, improve blood supply to nerve structures, stimulate reparative processes, and simultaneously manage the pain syndrome.

In the comparison group (n=10), therapy was limited solely to the administration of B-complex vitamins and NSAIDs. The absence of anti-edematous therapy and physiotherapeutic methods during the acute period significantly reduced treatment efficacy. Patients in this group demonstrated prolonged sensory recovery times, a lower rate of complete symptom regression, and a substantially higher risk of neuropathic pain chronification.

In conclusion, the differences in treatment methods proved to be decisive: a comprehensive pathogenetic approach in the main group ensured higher therapeutic efficacy, whereas standard treatment in the comparison group showed limited results. This confirms the necessity of implementing an expanded therapeutic algorithm into clinical practice to prevent persistent neurological complications.

An analysis of the dynamics of sensory recovery revealed significant differences between the main group and the comparison group, which was directly associated with the treatment methods applied and the timing of therapy initiation.

In the main group (n=50), complete sensory recovery was achieved in 36 patients (72%). Partial improvement—characterized by reduced severity of paresthesia and the gradual restoration of tactile and pain sensitivity, though without complete symptom regression—was observed in 12 patients (24%). In only 2 patients (4%) did the therapy fail to produce a significant effect, which confirms the high efficacy of the comprehensive pathogenetic approach.

In the comparison group (n=10), the outcomes were significantly less favorable. Complete sensory recovery was noted in only 3 patients (30%). Partial improvement was observed in 4 patients (40%), but persistent paresthesias and a reduced quality of life remained. In 3 patients (30%), the therapy failed to yield positive results, which indicates the insufficient efficacy of the standard treatment limited to the administration of B-complex vitamins and NSAIDs.

Thus, the differences between the groups were fundamental: the probability of complete recovery in the main group was more than twice as high as in the comparison group, while the proportion of patients who showed no improvement was nearly eight times lower.

An analysis of recovery times showed significant differences between the main group and the comparison group. In the main group (n=50), the mean time to sensory recovery was 3–4 months. This is attributed to the early initiation of therapy and the application of a comprehensive pathogenetic approach that included anti-edematous drugs and physiotherapeutic methods. Thanks to this intervention, it was possible to control ischemic processes more rapidly and stimulate the regeneration of nerve fibers.

In the comparison group (n=10), recovery times were substantially longer, averaging 6–8 months. The absence of anti-edematous therapy during the acute period and the restriction of treatment solely to B-complex vitamins and NSAIDs resulted in a slower regression of symptoms. Patients in this group more frequently experienced prolonged paresthesias and persistent sensory deficits, which negatively affected their daily activities and quality of life.

The incidence of chronic neuropathic pain was an equally important indicator. In the main group, it was observed in only 4 patients (8%),

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whereas in the comparison group, it occurred in 5 patients (50%). These data demonstrate that the absence of timely decompression and physiotherapeutic support significantly increases the risk of pain syndrome chronification.

CONCLUSION

The conducted study conclusively demonstrates that the outcome of treating iatrogenic injuries to the inferior alveolar and lingual nerves directly depends on the timeliness and comprehensiveness of the therapeutic intervention. A comparative analysis of the two patient groups revealed fundamental differences in the dynamics of sensory recovery, rehabilitation timelines, and the incidence of pain syndrome chronification.

In the main group, where the developed pathogenetic therapy algorithm was applied—incorporating the early administration of anti-edematous agents (glucocorticosteroids, venotonics) and physiotherapeutic methods (LLLT, magnetotherapy)—significantly superior outcomes were achieved: complete sensory recovery was observed in 72% of patients, the mean rehabilitation period was 3–4 months, and the incidence of chronic neuropathic pain did not exceed 8%. In the comparison group, where treatment was restricted to B-complex vitamins and NSAIDs, the indicators were substantially poorer: complete recovery was noted in only 30% of patients, recovery times extended to 6–8 months, and chronic pain developed in half of the cases.

Of particular significance is the confirmation of the hypothesis regarding the decisive role of post-traumatic edema in the pathogenesis of neurological complications. In 44% of patients, sensory loss developed secondarily, several hours after the intervention, indicating an ischemic mechanism of injury. These data underscore the critical importance of early intervention: the administration of dexamethasone within the first 24 hours reduced the time required to resolve paresthesia by 4–6 days and decreased the risk of permanent neuropathy by nearly 46%.

Physiotherapeutic methods demonstrated high efficacy in both the early and late periods. Low-level laser therapy contributed to a reduction in the numbness zone after just three sessions, while electrophoresis with neuroprotectors provided significant sensory improvement by the end of the course in patients with persistent paresthesias.

In conclusion, the study results confirm the necessity of implementing a comprehensive pathogenetic approach into clinical dental practice. The early initiation of therapy aimed at controlling edema and stimulating the regeneration of nerve structures substantially enhances treatment efficacy, shortens recovery periods, and minimizes the risk of chronic neurological complications.

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