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Trismus: Interprofessional Assessment, Functional Implications, and Rehabilitation Strategies in Clinical Practice

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Abstract

Background: Trismus is a clinical condition characterized by restricted mandibular mobility, often resulting from trauma, infection, temporomandibular disorders, or oncologic treatments. It significantly impacts mastication, speech, oral hygiene, and airway management.

Aim: To review the etiologies, functional implications, and management strategies for trismus in clinical practice.

Methods: A comprehensive literature-based analysis was conducted, synthesizing evidence on pathophysiology, epidemiology, diagnostic approaches, and therapeutic interventions.

Results: Trismus arises from diverse causes including trauma, dental procedures, infections, and radiotherapy-induced fibrosis. Acute cases typically resolve with conservative measures such as heat therapy, NSAIDs, and muscle relaxants. Chronic or refractory cases require physiotherapy, jaw motion devices, and occasionally surgical intervention. Early recognition and multidisciplinary care improve outcomes.

Conclusion: Timely diagnosis and tailored management are essential to prevent long-term disability and optimize functional recovery.

Keywords: Trismus, mandibular mobility, temporomandibular disorders, rehabilitation, physiotherapy..

Introduction

Trismus is a clinical condition characterized by a limitation in mandibular mobility, resulting in a reduced ability to open the mouth fully. Although the term was originally used to describe the jaw rigidity associated with tetanus, its contemporary usage has broadened to encompass any pathological or functional process that restricts jaw movement, regardless of underlying cause. This limitation in oral opening reflects impairment of the masticatory muscles, temporomandibular joint structures, or associated neuromuscular control mechanisms. As a result, trismus is best understood as a symptom rather than a disease entity, with diverse etiologies that may be local or systemic in origin.[1][2] In most clinical contexts, trismus is a transient condition. Acute cases frequently arise following dental procedures, maxillofacial trauma, local inflammation, or infection and tend to resolve spontaneously within a relatively short period, often less than two weeks. During this phase, the restriction in jaw motion may be mild to moderate and responsive to conservative management. However, in a subset of patients, trismus may persist or progress, leading to chronic or even permanent limitation of mouth opening. Persistent trismus is of particular clinical concern, as

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it can significantly compromise essential daily functions and overall quality of life. The functional consequences of sustained jaw restriction are substantial. Limited oral aperture interferes with basic activities such as mastication, speech articulation, and swallowing, thereby affecting nutritional intake and communication. Oral hygiene becomes increasingly difficult, predisposing patients to dental caries, periodontal disease, and oral infections. From a clinical perspective, trismus also complicates diagnostic evaluation and therapeutic interventions, particularly in dental, surgical, and airway management settings, where adequate mouth opening is critical for safe and effective care delivery.[1][2] Beyond its functional impact, trismus may serve as an important clinical indicator of underlying pathology, including infection, malignancy, fibrosis, or neuromuscular disorders. Early recognition and appropriate assessment are therefore essential to identify reversible causes and prevent long-term morbidity. Understanding trismus as a multifactorial condition with variable duration and severity underscores the need for timely evaluation and interdisciplinary management. When addressed promptly, most cases resolve without lasting impairment, whereas delayed or inadequate intervention increases the risk of permanent dysfunction with significant personal and healthcare system implications.[1][2]

Etiology

Trismus is a multifactorial clinical condition with a broad etiological spectrum that encompasses traumatic, inflammatory, infectious, neoplastic, iatrogenic, neurogenic, metabolic, congenital, and psychogenic processes. Its development reflects disruption of normal mandibular mechanics, whether through direct injury to the temporomandibular joint, impairment of the muscles of mastication, or secondary effects arising from pain, fibrosis, or neuromuscular dysfunction. The temporal course of trismus often correlates with its cause, as acute forms are commonly associated with transient insults, whereas chronic or permanent trismus is more frequently linked to structural damage, fibrosis, or progressive disease.[3] Acute trismus most often arises following trauma to the face or mandible or as a consequence of medical or dental interventions. Facial or mandibular trauma can provoke muscle spasm, joint effusion, or localized inflammation that restricts mouth opening. Iatrogenic trauma represents a particularly common cause in dental practice and includes procedures such as surgical extraction of impacted teeth or intramuscular administration of local anesthetic agents.[3] In these settings, trismus is typically self-limiting, resolving as tissue inflammation subsides and muscle function normalizes. In contrast, high-energy trauma or repeated injury may produce more extensive tissue damage, increasing the likelihood of persistent or chronic limitation of mandibular mobility.[3] One well-recognized iatrogenic cause of trismus is the administration of local anesthesia, particularly inferior alveolar nerve blocks. During this procedure, inadvertent penetration of the medial pterygoid or adjacent muscles of mastication may occur, leading to localized muscle injury and pain.[4] Pain-induced reflex muscle contraction limits mandibular opening as a protective response. In some cases, vascular injury to branches of the inferior alveolar artery or vein results in the formation of an intramuscular or pterygomandibular space hematoma, further exacerbating muscle stiffness and jaw restriction.[4] When trismus develops several days after injection, a needle-track infection should be suspected, as localized infection can provoke inflammation and muscle spasm.[4] Although anesthesia-related trismus is generally temporary, delayed recognition or inadequate management may prolong symptoms.

Dental extractions, particularly surgical removal of impacted mandibular third molars, represent another frequent cause of acute trismus. Postoperative pain, swelling, and restricted mouth opening are well-documented sequelae of such procedures.[5] These manifestations arise from the inflammatory response to surgical trauma, including edema of the surrounding soft tissues and irritation of the muscles of mastication.[5][6][5] In most patients, postoperative trismus resolves spontaneously within a short period; however, severe inflammation, secondary infection, or repeated surgical manipulation may extend the duration of symptoms. Trauma to the facial skeleton and mandible constitutes an important etiological category, particularly in cases of moderate to severe injury. Mandibular fractures commonly result in pain-related restriction of jaw movement, mechanical obstruction, or joint derangement.[1] Although less frequent, fractures of the zygomatic arch or zygomaticomaxillary complex can also lead to trismus by mechanically impeding the excursion of the coronoid process during mouth opening.[1] Such injuries may produce persistent trismus if not promptly identified and managed, especially when accompanied by malunion, fibrosis, or intra-articular damage. Temporomandibular disorders represent one of the most prevalent nontraumatic causes of trismus. These disorders encompass a group of conditions characterized by dysfunction and pain involving the temporomandibular joint and associated masticatory musculature.[7] Patients with temporomandibular disorders commonly report facial or preauricular pain, joint sounds such as clicking or crepitus, and limitations in mandibular range of motion.[7] Consequently, a substantial proportion of individuals presenting with restricted mouth opening are ultimately diagnosed with a temporomandibular disorder. Among these, myofascial pain and disc displacement without reduction are particularly associated with trismus, as muscle hyperactivity or

mechanical obstruction within the joint restricts mandibular translation.[8]

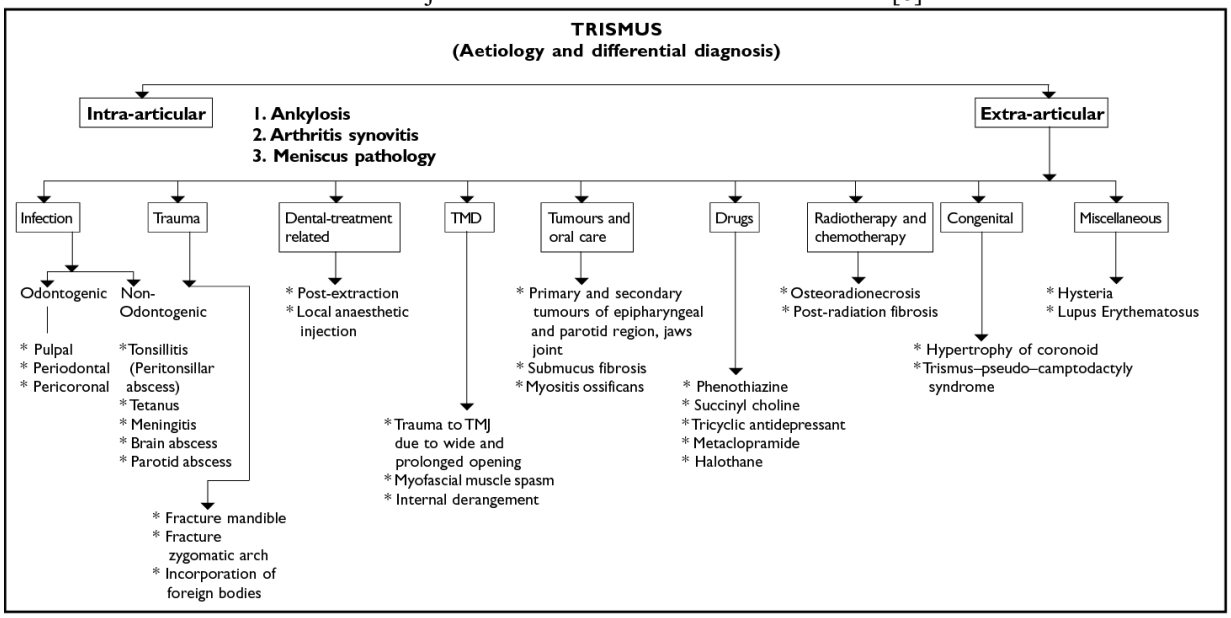

Fig. 1: Etiology of Trismus.

Infectious processes are critical considerations in the evaluation of acute trismus, as severe infections involving the masticatory spaces frequently produce marked jaw restriction.[9] Odontogenic infections, in particular, can extend from the dentoalveolar structures into adjacent fascial spaces of the head and neck, involving muscles of mastication and provoking intense inflammatory responses.[1] Pericoronitis, an infection associated with partially erupted teeth, is especially linked to trismus due to its proximity to the masticatory musculature.[1] Beyond dental origins, non-odontogenic infections such as tonsillitis, parotid abscess, tetanus, meningitis, and intracranial abscesses may also precipitate trismus through local inflammation, muscle spasm, or neurogenic mechanisms.[1] The potential for these infections to progress into life-threatening conditions, including cervical cellulitis or mediastinitis, underscores the importance of prompt recognition and intervention. Although less common, trismus may serve as a clinical manifestation of malignancy. Patients presenting with jaw restriction and preauricular pain are often initially presumed to have temporomandibular disorders.[10] However, neoplastic processes involving the head and neck region, including tumors of the pharynx, parotid gland, or adjacent osseous structures, may infiltrate or compress the muscles of mastication or temporomandibular joint, resulting in progressive trismus. Trismus rarely constitutes the primary presenting feature of malignancy, yet documented cases highlight the risk of misdiagnosis and delayed cancer detection.[10] For instance, reports describe patients treated for presumed temporomandibular disorders who were later found to have malignant tumors following prolonged symptom persistence.[10] To mitigate this risk, specialized clinics have developed criteria to identify atypical presentations warranting further investigation, including imaging and referral to senior clinicians.[8]

Trismus is also a frequent and clinically significant complication of head and neck cancer treatment. Radiotherapy, a cornerstone of oncologic management in this region, induces fibrosis within irradiated tissues, including the temporomandibular joint and muscles of mastication.[11] Fibrotic changes reduce tissue elasticity and impair muscle excursion, leading to restricted mandibular opening. The risk of developing trismus increases substantially when the medial pterygoid muscle lies within the radiation field.[14] Surgical interventions for head and neck malignancies may further contribute to trismus through scarring, muscle resection, or disruption of normal joint mechanics.[15] Epidemiological data indicate that trismus develops in approximately 38% to 42% of patients treated for head and neck cancer, reflecting its substantial impact on long-term functional outcomes.[12][13] From a classificatory perspective, trismus has been organized according to several conceptual frameworks. Some authors distinguish intra-articular causes, involving direct pathology of the temporomandibular joint, from extra-articular causes affecting surrounding muscles or soft tissues.[2] Others categorize etiologies based on their underlying nature, such as traumatic, infectious, inflammatory, or neoplastic origins.[1] Trismus may also be classified as iatrogenic when it results from therapeutic interventions, including surgery, anesthesia, or radiotherapy. These classification systems emphasize the heterogeneity of trismus and the necessity of a comprehensive diagnostic approach. Traumatic causes of trismus encompass a range of intra- and

extra-articular injuries, including hemarthrosis, hematoma formation, fractures or dislocations of the mandible or zygomatic arch, temporomandibular joint contusions, displaced articular discs, and direct muscle injury. Inflammatory etiologies include degenerative joint disease, soft tissue fibrosis, ankylosis of the temporomandibular joint, and systemic inflammatory disorders such as rheumatoid arthritis, scleroderma, and temporal arteritis. Infectious causes span pyogenic arthritis, osteomyelitis of the mandible, tonsillar and pharyngeal abscesses, tetanus, odontogenic abscesses, mumps, and parotid infections.

Congenital and developmental conditions, though rare, may also present with trismus. Syndromes such as Pierre Robin sequence or trismus-pseudocamptodactyly syndrome involve craniofacial or musculoskeletal abnormalities that restrict mandibular motion from early life. Neoplastic causes include primary and secondary tumors of the pharynx, parotid gland, and odontogenic structures, which may progressively impair jaw mobility through infiltration or mass effect. Neurogenic mechanisms contribute to trismus in disorders characterized by abnormal muscle activation or spasm, including tetanus, status epilepticus, Parkinsonian syndromes, and toxin-mediated conditions such as strychnine or phenothiazine poisoning. Adverse effects of certain medications, including phenothiazines, metoclopramide, and tricyclic antidepressants, may also provoke jaw rigidity through extrapyramidal reactions. Metabolic disturbances, such as hypocalcemia, hypomagnesemia, or respiratory alkalosis, can alter neuromuscular excitability and precipitate trismus. Finally, psychogenic causes, including conversion disorder, highlight the complex interplay between psychological factors and somatic manifestations of restricted jaw movement. Collectively, the diverse etiologies of trismus underscore its role as a clinical sign rather than a singular diagnosis. Accurate identification of the underlying cause requires careful history-taking, thorough physical examination, and judicious use of imaging and laboratory investigations. Recognition of both common and rare etiologies is essential to prevent misdiagnosis, guide appropriate management, and minimize the risk of long-term functional impairment.[1][2][3]

Epidemiology

The epidemiology of trismus is characterized by considerable variability, largely due to the absence of universally accepted diagnostic thresholds defining abnormal limitation of mandibular opening. As a result, reported prevalence rates differ substantially across studies and clinical settings, with estimates strongly influenced by the underlying cause, population characteristics, and method of assessment. Trismus should therefore be understood not as a single disease entity with a fixed prevalence, but as a clinical manifestation whose frequency reflects the distribution of its diverse etiologies. In the general population, transient trismus is relatively uncommon and often underreported, as mild and self-limited cases frequently resolve without medical attention. However, in specific clinical subgroups, trismus represents a frequent and clinically significant finding. Patients with congenital craniofacial anomalies, particularly syndromes associated with micrognathia or abnormal mandibular development, demonstrate a higher baseline prevalence of restricted mouth opening due to structural limitations and altered temporomandibular joint mechanics.[16] In these individuals, trismus may present early in life and persist chronically, contributing to feeding difficulties, speech impairment, and challenges in airway management. Trismus is especially prevalent among patients undergoing treatment for head and neck malignancies. Radiotherapy, with or without surgical intervention, is a major contributor to jaw motion restriction in this population. Fibrosis of the muscles of mastication and periarticular tissues following radiation exposure leads to a progressive reduction in mandibular mobility. Epidemiological studies indicate that a substantial proportion of patients treated with radiotherapy for head and neck cancers develop clinically meaningful trismus, with reported rates varying according to tumor location, radiation dose, and involvement of the medial pterygoid muscle within the radiation field.[17] As cancer survival improves, the long-term burden of treatment-related trismus has become increasingly relevant in survivorship care.

In contrast, trismus may occur infrequently as a complication of otherwise common and typically benign conditions. Acute upper respiratory tract infections, such as pharyngitis or tonsillitis, rarely result in trismus; when present, it usually reflects local inflammation, peritonsillar involvement, or early deep neck space infection. Although uncommon, recognition of trismus in these settings is clinically important, as it may signal progression to more serious pathology requiring urgent intervention. Dental and iatrogenic causes contribute variably to the epidemiological profile of trismus. Procedures such as mandibular third molar extraction or inferior alveolar nerve block injections are common worldwide, yet only a small proportion of patients develop clinically significant trismus. When it does occur, it is generally temporary and resolves within days to weeks, limiting its impact on prevalence estimates in long-term studies. Overall, the epidemiology of trismus reflects its heterogeneity and context-dependent nature. Higher prevalence is consistently observed in populations with structural craniofacial abnormalities and in patients receiving head and neck cancer therapy, whereas sporadic cases arise across a wide range of medical, dental, and infectious conditions. Improved standardization of diagnostic criteria and routine measurement of

maximal interincisal opening may enhance future epidemiological understanding and allow more accurate estimation of the true burden of trismus across healthcare settings.[16][17]

History and Physical

Assessment of trismus begins with careful history taking and detailed physical examination, as limitation of mandibular movement is a clinical sign rather than a diagnosis. Normal maximal mouth opening typically falls within the range of 40 to 60 mm and is often estimated clinically as two to three finger breadths. Many authors define trismus as a maximum interincisal opening of less than 35 mm.[1] This threshold is not absolute. Normal jaw mobility varies between individuals and is influenced by anatomical and demographic factors, including sex, with males generally demonstrating greater maximal mouth opening than females.[1] For this reason, comparison with the patient's baseline function, when known, is clinically valuable. Patients who present with trismus usually report difficulty opening the mouth and may experience pain during attempted mandibular movement. In many cases, however, the patient's primary complaint relates to the underlying cause rather than the mechanical restriction itself. Individuals with odontogenic etiologies frequently describe localized tooth pain, gingival swelling, or discomfort exacerbated by chewing. Those with traumatic causes often report facial or mandibular pain, a history of direct impact, or recent dental or surgical procedures involving the jaw. Acute onset following an identifiable event suggests inflammatory or traumatic origins, whereas gradual progression raises concern for chronic disorders such as temporomandibular joint pathology or neoplastic disease. A comprehensive medical history is essential to identify systemic contributors. The presence of fever may indicate an infectious etiology, particularly in association with facial swelling or odynophagia. Unintentional weight loss, night sweats, or persistent pain unresponsive to conservative measures should heighten suspicion for malignancy. Neuromuscular or metabolic causes may manifest with associated symptoms such as carpopedal spasms, muscle cramps, or paresthesias, which can accompany electrolyte disturbances or neurogenic conditions. A history of tobacco use, alcohol consumption, or previously diagnosed cancer significantly increases the likelihood of a neoplastic process involving the head and neck.[1]

Medication history also provides important diagnostic clues. Exposure to drugs known to affect neuromuscular function or dopamine pathways may contribute to jaw rigidity or dystonic reactions. Prior radiation therapy or surgical intervention in the head and neck region should be specifically explored, as these treatments are well established risk factors for fibrosis-related trismus. Duration and progression of symptoms help differentiate transient inflammatory causes from chronic structural or malignant conditions. Physical examination may be challenging, as restricted mouth opening can limit visualization of intraoral and pharyngeal structures. Nonetheless, the examination should be as focused and systematic as possible. Inspection and palpation of the teeth and gingiva are critical for identifying dental infections, abscesses, or pericoronal inflammation. Evaluation of the facial bones and mandible may reveal tenderness, deformity, or asymmetry suggestive of fracture or dislocation. Palpation of the temporomandibular joint during attempted opening and closing can identify joint tenderness, crepitus, or deviation of the mandible, which may indicate internal derangement or inflammatory joint disease. Examination of the oropharynx remains essential despite technical difficulty. The pharyngeal pillars, tonsils, uvula, and posterior pharyngeal wall should be assessed for erythema, asymmetry, fluctuance, or exudate. Certain infections associated with trismus can alter phonation. Assessment of speech may therefore provide indirect diagnostic information. A muffled or "hot potato" voice is classically associated with tonsillitis or peritonsillar abscess and should prompt urgent evaluation.[2] Cervical examination should include palpation for lymphadenopathy, masses, or tenderness, as these findings may support infectious or malignant etiologies.

A directed neurologic examination is necessary when neurogenic causes are suspected. Evaluation of cranial nerve function, muscle tone, and involuntary movements may reveal underlying central or peripheral nervous system pathology.[2] Signs of generalized neuromuscular irritability further support metabolic or toxic causes. Overall, the history and physical examination provide the foundation for identifying the cause of trismus and determining the need for further diagnostic testing. Careful attention to symptom pattern, associated systemic features, and focused examination findings allows clinicians to distinguish benign, self-limited conditions from serious infectious, neurologic, or malignant disorders that require urgent intervention.[1][2]

Evaluation

The evaluation of trismus relies primarily on clinical assessment, as the diagnosis is established through history and physical examination rather than laboratory testing. Measurement of maximal interincisal distance provides an objective estimate of severity and allows for monitoring of progression or response to treatment over time. Clinical evaluation should focus on identifying whether the limitation of mandibular movement is acute or chronic and whether it is associated with pain, inflammation, neurologic signs, or systemic symptoms, as these features often indicate the underlying cause. Imaging studies serve as important adjuncts when the etiology cannot be determined clinically or when serious

pathology is suspected. Computed tomography is particularly valuable in the assessment of traumatic causes of trismus. It enables accurate visualization of facial and mandibular fractures, joint dislocations, and the presence of hematomas within the masticatory spaces or temporomandibular joint region. CT imaging also assists in evaluating bony abnormalities that may mechanically restrict mandibular movement. Magnetic resonance imaging plays a complementary role, especially when soft tissue pathology is suspected. MRI provides superior contrast resolution for identifying space-occupying lesions, inflammatory changes, and neoplastic processes affecting the masticatory muscles, temporomandibular joint, or adjacent pharyngeal and oral structures. It is also useful for assessing internal derangements of the temporomandibular joint, including disc displacement and joint effusion. The selective use of imaging, guided by clinical findings, supports accurate etiological diagnosis and informs appropriate management strategies [3].

Treatment / Management

The management of trismus is primarily directed toward identifying and addressing the underlying cause while providing symptomatic relief to restore mandibular mobility and reduce patient discomfort. In most cases, trismus is transient and responds well to conservative measures, particularly during the acute phase. Early intervention focuses on reducing muscle spasm, inflammation, and pain, which are the principal contributors to restricted jaw movement in uncomplicated presentations. Symptomatic treatment remains the cornerstone of care, especially when trismus follows dental procedures, minor trauma, or local inflammation. During the acute phase, local heat therapy is widely recommended to promote muscle relaxation and improve regional blood flow. The application of moist heat, such as warm towels applied for 15 to 20 minutes each hour, has been shown to reduce muscle stiffness and pain, facilitating gradual improvement in mouth opening [1]. Analgesic therapy is equally important, with non-steroidal anti-inflammatory drugs serving as first-line agents. Aspirin is often sufficient for pain control and inflammation reduction in mild to moderate cases [1]. In patients who exhibit significant muscle spasm, the short-term use of muscle relaxants may be indicated. Benzodiazepines, particularly diazepam at doses ranging from 2.5 to 5 mg administered three times daily, are commonly prescribed to reduce involuntary muscle contraction and improve mandibular mobility [1]. These agents should be used judiciously and for limited durations to avoid adverse effects.

Dietary modification represents an essential supportive measure during the acute stage. Patients are advised to maintain a soft or semi-liquid diet to minimize strain on the masticatory muscles and temporomandibular joint while healing occurs [1]. Activities that may exacerbate muscle tension, including prolonged mouth opening or further dental interventions, should be deferred until the trismus resolves. In uncomplicated cases, noticeable improvement is typically observed within 48 hours after initiating conservative treatment [18]. When trismus persists beyond the acute inflammatory phase or extends longer than one week, rehabilitative strategies become increasingly important. Stretching and jaw mobility exercises are indicated in post-traumatic, post-operative, or prolonged cases to prevent fibrosis and restore functional range of motion. These exercises generally involve repeated attempts to open the mouth against gentle resistance, performed several times daily [11]. Structured physiotherapy programs emphasize controlled opening and closing movements as well as lateral mandibular excursions for approximately five minutes every three to four hours [19]. The use of sugar-free chewing gum may further encourage lateral movements and stimulate coordinated muscle activity, supporting gradual recovery [1].

Physical therapy plays a central role in managing persistent trismus by addressing both functional and structural limitations. Regular therapeutic exercises help reduce edema, limit fibrotic changes, enhance local circulation, and improve muscle strength and flexibility. Maintaining continuous muscle activity is essential to prevent long-term restriction and functional impairment [3]. Patient adherence to prescribed exercise regimens significantly influences outcomes, particularly in cases where trismus follows surgery or trauma. In certain clinical contexts, trismus may progress to a chronic condition, especially when associated with head and neck radiotherapy, extensive fibrosis, or malignancy-related interventions. These cases often require more intensive and prolonged physiotherapeutic approaches. Commercially available jaw motion rehabilitation devices may be utilized to provide controlled, progressive stretching and to standardize exercise intensity. Microcurrent therapy has also been explored as an adjunctive modality in selected patients, particularly those who do not respond adequately to conventional physiotherapy. Pharmacologic agents such as xanthine derivatives, including pentoxifylline, have been described in the literature for their potential role in reducing radiation-induced fibrosis and improving tissue perfusion, thereby contributing to improved jaw mobility [1][20].

Fig. 2: Treatment of Trismus.

Referral to an oral and maxillofacial surgeon should be considered when trismus fails to improve after two to three days of appropriate conservative management or when the degree of mouth opening is severely limited [1]. Specialist evaluation is particularly important in cases where infection, malignancy, or structural abnormalities are suspected. Severe or refractory trismus that persists despite exhaustive non-surgical measures may necessitate operative intervention. Surgical options are reserved for carefully selected patients and aim to mechanically increase mandibular opening or release restrictive tissues. Surgical management strategies include procedures such as coronoidectomy, which removes mechanical obstruction caused by an elongated or fibrotic coronoid process, and mandibular height reduction to facilitate increased mouth opening. In cases where extensive soft tissue fibrosis is present, tissue release combined with free flap reconstruction may be required to restore functional anatomy and mobility [11]. These interventions carry inherent risks and require comprehensive postoperative rehabilitation to maintain surgical gains. Overall, the management of trismus requires a stepwise and etiology-focused approach, beginning with conservative symptomatic care and progressing to rehabilitative and surgical interventions when necessary. Early recognition, timely initiation of therapy, and patient adherence to treatment recommendations are critical determinants of successful outcomes. By addressing both the symptoms and the underlying cause, clinicians can minimize functional impairment, prevent chronic disability, and improve quality of life for affected individuals.

Differential Diagnosis

Trismus is defined by restricted mandibular opening regardless of cause, which narrows its differential diagnosis compared with symptom-based clinical entities. The diagnostic challenge lies not in distinguishing trismus from other conditions, but in identifying the anatomical and physiological mechanism responsible for the limitation. Some authors restrict the term to neuromuscular restriction mediated by the trigeminal nerve and its motor branches, describing this entity as true trismus. Under this interpretation, conditions that limit mouth opening through intraarticular mechanisms, such as temporomandibular joint ankylosis, intraarticular fibrosis, or structural joint derangement, are considered separate disorders rather than causes of trismus [1]. Other classifications adopt a broader framework and include intraarticular pathology as a subtype within the spectrum of trismus, recognizing the overlap in clinical presentation and functional impairment [20]. Clinical differentiation requires careful assessment of pain pattern, onset, progression, and associated neurological or mechanical findings. Neuromuscular restriction often presents with acute painful spasm and preserved joint architecture, whereas intraarticular disease tends to produce progressive limitation with reduced joint translation. Imaging studies and response to conservative therapy further assist in distinguishing these mechanisms. Accurate differentiation guides management strategy and prognosis, particularly when invasive intervention is under consideration.

Prognosis

The prognosis of trismus is favorable in most clinical contexts. The condition is commonly transient and self-limited, with spontaneous or treatment-assisted resolution occurring within two weeks in uncomplicated cases. This pattern is typical following dental procedures, minor trauma, or acute inflammatory conditions affecting the muscles of mastication. Early symptom-directed therapy and avoidance of aggravating factors support rapid recovery. Prognosis becomes less favorable when trismus arises from chronic structural or fibrotic processes. Patients exposed to head and neck radiotherapy represent a distinct group with increased risk of persistent limitation due to progressive fibrosis of masticatory muscles and periarticular tissues [17]. In these cases, trismus may show limited response to conservative therapy and may require prolonged rehabilitation or surgical intervention. Delayed recognition and inadequate early management further worsen outcomes by allowing irreversible tissue changes. Overall prognosis depends on etiology, duration before intervention, and patient adherence to rehabilitative strategies. When the underlying cause is reversible and addressed promptly, functional recovery is expected.

Complications

Trismus produces significant functional consequences when persistent or severe. Restricted mouth opening interferes with speech articulation, mastication, and effective swallowing, leading to nutritional compromise and reduced oral intake. Impaired swallowing mechanics increase the risk of

aspiration, particularly in patients with concurrent neurological or infectious disease. Oral hygiene becomes difficult to maintain, increasing susceptibility to dental decay and periodontal disease. From an airway management perspective, significant trismus presents a critical challenge. Conventional oropharyngeal intubation may be impossible, necessitating alternative approaches such as nasopharyngeal intubation or surgical airway access. Prolonged trismus leads to reduced joint mobility and adaptive shortening of muscles and connective tissue, which promotes temporomandibular joint fibrosis and long-term dysfunction [20]. These complications reinforce the importance of early intervention and active rehabilitation to prevent permanent limitation and secondary morbidity.

Consultations

Consultative care is guided by the suspected underlying cause and severity of functional impairment. Dental practitioners or oral and maxillofacial surgeons play a central role in cases related to odontogenic infection, dental trauma, or postoperative complications. Otolaryngology consultation is often required when trismus is associated with deep neck space infection or peritonsillar abscess requiring drainage. Neurological consultation may be appropriate when neurogenic causes or central nervous system pathology are suspected. Physiatrists and physical therapists contribute to the management of persistent or refractory trismus by designing structured rehabilitation programs aimed at restoring mandibular mobility and preventing fibrosis [1]. Multidisciplinary coordination improves diagnostic accuracy and ensures timely escalation of care when conservative measures fail.

Patient Education

Patient education is essential in preventing worsening and recurrence of trismus. Individuals should be instructed to rest the jaw during the acute phase and avoid behaviors that increase muscle tension, including teeth clenching, excessive chewing, and prolonged mouth opening. Awareness of early symptoms encourages prompt medical evaluation before functional limitation becomes established. In selected cases, collaboration with a physical therapist supports safe initiation of jaw mobility exercises and reinforces adherence to therapy. Education also addresses realistic expectations regarding recovery time and the importance of continued exercise after symptom improvement to prevent relapse.

Other Issues

Trismus frequently develops after common procedures such as third molar extraction, yet it may also complicate rare conditions including radiation-induced fibrosis. Effective management depends on identifying the precipitating cause rather than treating jaw restriction in isolation. Most cases resolve with conservative measures such as heat therapy and anti-inflammatory medication. Chronic or refractory cases benefit from structured physiotherapy. Atypical presentations warrant heightened suspicion for malignancy, and structured assessment tools may support early detection. Mandibular opening devices should be prescribed and supervised by experienced clinicians to avoid injury and ensure benefit.

Enhancing Healthcare Team Outcomes

Trismus reflects a diverse range of etiologies that span dental, infectious, neurological, oncological, and rehabilitative domains. Optimal outcomes depend on coordinated interprofessional management. Timely referral based on suspected cause reduces diagnostic delay and prevents progression to chronic disability. When the primary pathology is treated effectively, recovery is usually complete. In contrast, patients with severe trauma, extensive scarring, or prior radiotherapy often experience persistent limitation with reduced quality of life despite intervention [21][22][23][24]. Continuous communication among healthcare professionals and early rehabilitation planning remain central to improving functional outcomes.

Conclusion:

Trismus represents a multifactorial condition with significant functional and clinical implications. While most cases are transient and respond well to conservative therapy, delayed recognition or inadequate management can lead to chronic restriction, fibrosis, and compromised quality of life. The condition not only interferes with essential functions such as mastication, speech, and oral hygiene but also complicates airway management and surgical procedures. Effective care requires a stepwise approach—beginning with symptomatic relief and progressing to rehabilitative exercises and surgical intervention when necessary. Multidisciplinary collaboration among dental, surgical, and rehabilitative teams is critical for accurate diagnosis and comprehensive treatment. Patient education and adherence to physiotherapy regimens further enhance recovery and prevent recurrence. Ultimately, early intervention and etiology-focused management remain the cornerstone of favorable outcomes, reducing morbidity and improving functional independence for affected individuals.

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