



Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology

CLINICOPATHOLOGIC CONFERENCE

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Clinicopathologic conference: Trismus following dental treatment

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Myositis ossificans traumatica is a rare clinical entity in the maxillofacial region. We present a case of myositis ossificans traumatica of the temporalis muscle following dental treatment in the form of a clinicopathologic conference. A review of the literature supporting our findings is provided. (*Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2004;98:261-6)

CLINICAL PRESENTATION

A 68-year-old white man presented complaining of a limited mouth opening. He had progressive decrease in maximal mouth opening for the previous 2 weeks. Five weeks prior to presentation, the patient had undergone endodontic and restorative treatment of the left mandibular quadrant. The patient further reported that 4 injections were needed to achieve anesthesia during his last dental visit and that his mouth had been widely open for an extended period of time. The patient denied a history of joint sounds, pain, or any previous limitation of mandibular movement. His past medical history was significant for hiatal hernia repair in 1984, right hemicolectomy for treatment of carcinoma of the colon in 1988, hypertension, and an unspecified dermatitis. The latter conditions were controlled with nifedipine (60 mg qd) and prednisone (5 mg qd) with alternating 2-week cycles. Prior to consultation, the patient reported that his restorative dentist had prescribed nonsteroidal anti-inflammatory drugs and intensive mouth-opening exercises. After 2 weeks and no improvement, the patient was referred to an oral and maxillofacial surgeon.

On clinical examination, a maximal interincisal opening of 5 mm without deviation was recorded. Lateral excursions were limited to approximately 2 mm bilaterally. The protrusive movement was also limited to an estimated 2 mm. Despite the limited range of motion, the patient reported no associated pain or change in his occlusion. There was no evidence of extraoral swelling or asymmetry. On palpation, the neck was soft with full range of motion and there was no evidence of lymphadenopathy or tenderness. A panoramic radiograph revealed partial maxillary and mandibular edentulism with multiple dental restorations; however, no significant pathologic findings were noted (Fig 1).

DIFFERENTIAL DIAGNOSIS

In cases of trismus, important diagnostic information can be obtained through careful history taking and evaluation of any deviation on opening and protrusion or limitation in lateral excursive movements of the mandible. The findings in this case suggested that both translation and complete rotation of the condylar head in the glenoid fossa were being prevented bilaterally. The next diagnostic step was to determine whether the restriction was of an intra- or an extra-articular etiology.

Intra-articular causes of trismus

In the present case, exacerbation of a chronic temporomandibular joint disorder was excluded on the basis of patient history. With the acute onset and clinical findings, possible considerations included bilateral fibrous ankylosis, the anchored disk phenomenon, and bilateral anterior disc displacement without reduction (acute closed lock). The latter condition was not felt to

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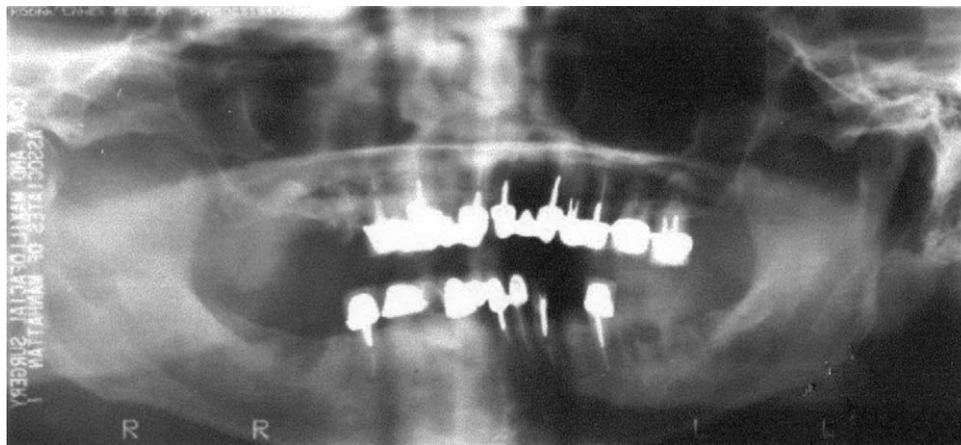


Fig 1. Panoramic radiograph at presentation.

be likely in our patient, because it is typically preceded by a history of clicking and intermittent locking. Also, anterior disc displacement without reduction usually results in immediate trismus, followed by gradual improvement of jaw mobility as the posterior discal tissue is stretched. Such a pattern would essentially be the reverse of the progressive trismus described by our patient.

The anchored disc phenomenon is a proposed etiologic factor of severe and persistent closed lock of the temporomandibular joint. Initially described by Nitzan and Marmary in 1997,¹ its existence as a distinct clinical entity is still debated. The main purported functional disturbance in the anchored disk phenomenon is inability of the disk to translate secondary to the formation of adhesions or a “suction cup effect.” Because the condylar head is still able to translate, the maximum mouth opening is usually between 15 and 20 mm.

Fibrous ankylosis, the development of strong adhesions within the temporomandibular joint, was considered a plausible explanation for the clinical presentation of our patient. The patient reported keeping his mouth widely open for a prolonged period of time at his last dental appointment. This could have produced intracapsular injury leading to hematoma formation and/or severe inflammation with scarring. Fibrous ankylosis may lead to painless severe trismus similar to that of our patient. Magnetic resonance imaging and temporomandibular joint arthroscopy can be used to confirm fibrous ankylosis.

Extra-articular causes of trismus

Extra-articular etiologies can include infection, foreign body reaction, musculoskeletal injury or disorder, and neoplasm. Trismus is often associated with infections involving the masticator space. Needle tract infection or foreign body reaction involving the

pterygomandibular space following administration of mandibular nerve block is an uncommon but recognized complication of local anesthesia. In this case, the complete lack of other symptoms and absence of an identifiable source of infection or foreign body made these considerations seem unlikely. Similarly, a neoplastic process was not favored owing to the acute onset of symptoms.

Following injury to the muscles of mastication, hematoma formation with eventual fibrosis and even ossification may result. The anterior attachments of the temporalis muscle as well as the medial pterygoid muscle are at risk of injury during administration of a mandibular nerve block. Our patient may have been particularly prone to hematoma formation due to the capillary fragility induced by chronic steroid therapy. Furthermore, the patient reported that he had multiple left inferior alveolar nerve blocks at his last dental appointment, increasing the likelihood of intramuscular injury. In cases of myositis ossificans traumatica, fibrosis and ossification may occur within 2-3 weeks following injury. Unfortunately, evidence of calcification within the muscles of mastication is often difficult to detect initially on a panoramic radiograph owing to the superimposition of the mandible.

In summary, the 3 most likely diagnoses consistent with the given history and clinical findings were fibrous ankylosis, muscle fibrosis secondary to hemorrhage, and myositis ossificans traumatica.

SUBSEQUENT COURSE

Considering the history, the oral and maxillofacial surgeon attempted to perform manipulation and forced mouth opening under IV conscious sedation, but without success. Computerized tomography (CT) imaging was then obtained. It revealed inflammation and calcification



Fig 2. Axial CT scan just superior to the level of the zygomatic arch showing mass with peripheral calcification within the left temporalis muscle.

within the temporalis and medial pterygoid muscle on the left side (Fig 2). This finding was felt to be consistent with a radiological diagnosis of myositis ossificans.

The patient was then scheduled for surgery to release the trismus and improve his range of motion. Following fiberoptic nasotracheal intubation, a transoral approach was used to make a full thickness incision from the left anterior mandibular ramus extending on the external oblique ridge to the level of the first molar. Subperiosteal dissection was performed to expose both the medial and lateral aspect of the mandibular ramus as well as the coronoid process. Using a reciprocating saw, a left coronoidectomy was performed (Fig 3). Calcified and fibrotic fibers of the anterior attachment of the temporalis muscle were isolated with blunt dissection and excised.

Next, areas of fibrosis and calcification were located within the left medial pterygoid muscle and left masseter muscle. Excision of these areas was accomplished using sharp and blunt dissection. In addition, releasing incisions were performed on the lateral surface of the medial pterygoid and medial aspect of the masseter muscle. Following this procedure, an interincisal distance of 41 mm was recorded upon manual opening. A

Penrose drain was placed medial to the mandibular ramus and the wound was closed following copious irrigation.

The patient tolerated the procedure well and was discharged in stable condition that same evening. He was provided with a Therabite™ appliance for intensive home physical therapy and appointed for follow-up evaluation.

DIAGNOSIS

Microscopic examination was performed on the excised lesional tissue consisting of a portion of the left temporalis muscle and adjoining tissue. Microscopic evaluation revealed an arc-like arrangement of woven and lamellar bone within a dense fibrous connective tissue (Fig 4). Higher magnification revealed fibrovascular connective tissue and skeletal muscle fibers adjacent to the bony trabeculae (Fig 5).

Final diagnosis was myositis ossificans traumatica.

FOLLOW-UP

After 16 weeks of close follow-up and home therapy, the patient displayed a 38-mm interincisal opening. Left

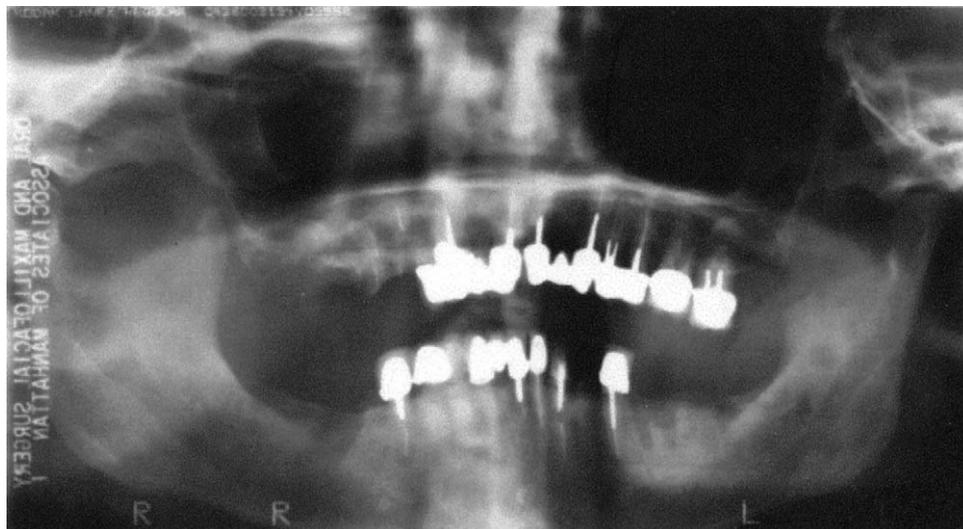


Fig 3. Panoramic radiograph following left coronoidectomy.

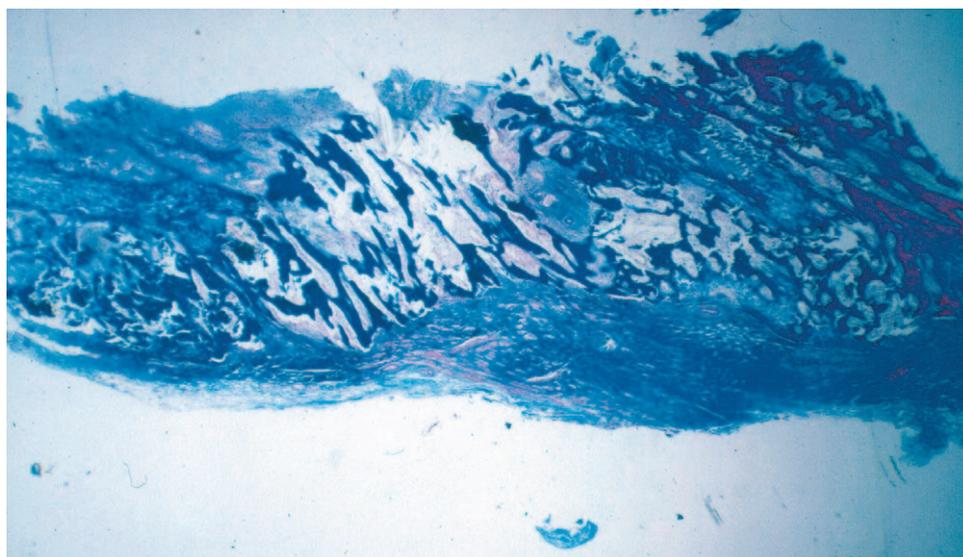


Fig 4. Low-power view demonstrating bony trabeculae (dark blue) within a dense fibrous connective tissue stroma (lighter blue areas). Skeletal muscle bundles (red) are seen at the periphery of the specimen. (Masson's trichrome, original magnification $\times 25$.)

lateral excursive and protrusive movements measured 5-7 mm while the right lateral excursion was approximately 3-5 mm. Three and a half years after surgery the patient demonstrated a maximal interincisal opening of 40 mm.

DISCUSSION

Limited information is available regarding myositis ossificans traumatica (MOT) affecting the oral and maxillofacial area. Spinazze et al² presented a single case and reviewed the literature for a total of 29 cases involving the head and neck region. A thorough literature search revealed 21 additional cases.³⁻¹⁰ With the addition

of our patient, a total of 51 cases of MOT in the head and neck have now been reported. There is a male predominance of nearly 3:1 (37 males, 13 females, 1 gender unreported), with the masseter muscle being most commonly affected. The relative vulnerability of the masseter to trauma may explain this localization.

Following significant physical injury, skeletal muscle has a limited capacity for regeneration. Initially, the muscle fibers surrounding the site of injury degenerate in a zonal fashion. Central necrosis may be seen, followed by engulfment and digestion of the necrotic material by macrophages and other phagocytic cells. Simultaneously, proliferating myoblasts fuse to form

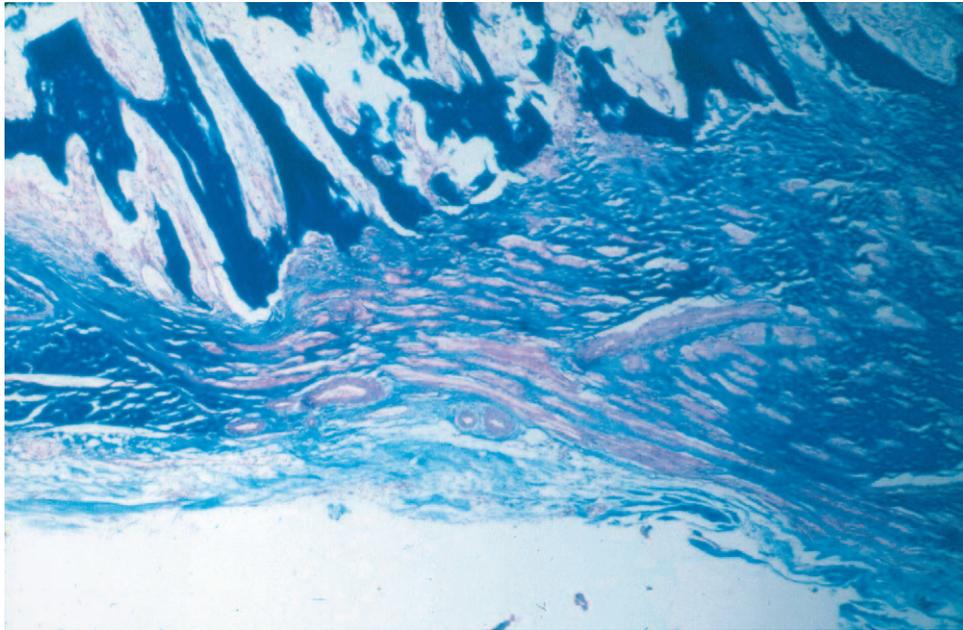


Fig 5. Higher-power view revealing striated muscle fibers (red) and fibrovascular connective tissue (blue) adjacent to the bony trabeculae. (Masson's trichrome, original magnification $\times 100$.)

myotubules and ultimately new muscle in smaller areas of injury. In cases of larger insults, the repair process predominates and usually leads to formation of scar tissue at the site of injury rather than muscle.¹¹

While the pathogenesis of MOT is unclear, most authors consider it as an aberrant form of physiologic healing. Carey¹² proposed 4 main theories of its development: 1) displacement of bony fragments into the soft tissue with subsequent proliferation, 2) detachment of periosteal fragments into the surrounding tissue with proliferation of osteoprogenitor cells, 3) migration of subperiosteal osteoprogenitor cells into surrounding soft tissue through periosteal perforations induced by trauma, and 4) metaplasia of extraosseous cells exposed to bone morphogenic protein derived from lysis of bone fragment(s) displaced within the soft tissue during traumatic injury. Among these alternatives, the latter theory seems to be the most widely accepted. More recently, it has been proposed that MOT results from initial intramuscular hemorrhage with exuberant proliferation of vascular granulation tissue that undergoes subsequent metaplasia to cartilage or bone.³

Four different phases of MOT have been described that correlate well to the histologic and radiologic findings.¹³ The initial pre-bone-forming phase features capillary and mesenchymal cell proliferation at the periphery of the wound. Lacking calcifications, this phase is inconspicuous on radiographic examination. The succeeding phases exhibit the classical radiographic changes and include the incipient bone-producing phase

(1-2 weeks), the intermediate phase (4 weeks), and the late phase (6 weeks). Radiographically, the mature lesion appears as a central lucency surrounded by a rim of bone.¹³

The histopathology of the mature MOT lesion presents a zonal architecture that recapitulates the radiographic findings. The central zone is cellular with variable mitotic activity. The intermediate zone shows immature osteoid formation with a fibromuscular background. Finally, the outer zone is characterized by mature peripheral bone in a less cellular fibrous stroma.¹⁴ This histology allows MOT to be distinguished from other forms of dystrophic calcification. Careful histopathologic evaluation of these lesions is warranted, however, because rare examples of osteogenic sarcoma have been reported to arise from MOT or to mimic its clinical features.^{15,16}

Patients with MOT involving the muscles of mastication typically present with limitation of mandibular movement. While severe trismus is common, Tong et al⁴ reported a case of MOT involving bilateral medial pterygoid muscles that presented as limited excursive jaw movement without trismus. A painless, indurated mass is often palpable within the involved musculature. While the patient may not initially report recent injury to the area, a potential traumatic event or origin can occasionally be elicited through careful history taking. What remains unclear is the extent of injury necessary to develop MOT. Takahashi and Sato⁵ reported that 40% to 60% of MOT patients present with a history of

significant blunt trauma and that the remainder of cases were probably secondary to chronic microtrauma. The time interval between trauma and first detection of a calcified mass has ranged from 3 weeks to 20 years.¹⁷ In the current case, it seems likely that the combination of multiple injections and prolonged mouth opening represented the initiating trauma that led to the development of MOT.

Effective management of MOT can be achieved through surgical excision of the entire calcified mass. To minimize the possibility of recurrence, some authors recommend that surgical removal be performed within 3-6 weeks following trauma.^{2,17} The secondary effect of postsurgical scarring should not be ignored and intensive physiotherapy should be part of postoperative care. Although multiple pharmacological agents such as bisphosphonates, warfarin, and corticosteroids have also been used in cases of MOT, they have demonstrated limited additional benefit.^{2,10}

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